HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Digitized by the Internet Archive
in 2015

https://archive.org/details/californiamedici1061cali

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

XGT-115

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
7 196

CIRCULATES AFTER
FEB

California
M ine

Serials Department
Health Sciences Library
111 South Greene St.
Baltimore, Md. 21201

VOLUME 106 • NUMBER 1
January 1967

1 *Circulatory Shock—A Symposium on Advances in the Understanding of Mechanisms and Treatment*

12 *The Role of External Irradiation in the Treatment of Transitional Cell Carcinoma of the Bladder—A Review of Fifty Cases* GEORGE JACK, *M.D.*, and FRANZ BUSCHKE, *M.D.*, San Francisco

17 *A Family Planning Clinic in a County Hospital* JOHN N. MILLER, JR., *M.D.*, Sacramento

20 *North American Blastomycosis in California—Diagnosis of a Case and a Survey with 28 California Observed Cases* DONALD E. CASAD, *M.D.*, and WILLIAM J. WALDMANN, *M.D.*, Fresno, NORMAN E. LEVAN, *M.D.*, Los Angeles, and ROYAL H. SORENSEN, *B.S.*, Fresno

28 *Systemic Agents in the Management of Acne* RONALD M. REISNER, *M.D.*, Torrance

35 *How Community Cardiovascular Disease Programs Can Aid the Private Physician* J. GORDON BARROW, *M.D.*, Atlanta

39 *An Exercise Program for Shoulder Disability* DAVID RUBIN, *M.D.*, Los Angeles

44 *Unsuspected Carcinoma Found Histologically After Minor Anorectal Operations* LEWIS GRODSKY, *M.D.*, San Francisco

MEDICAL PROGRESS ARTICLE

49 *The Problems of LSD*^{25} *and Emotional Disorder* J. THOMAS UNGERLEIDER, *M.D.*, and DUKE D. FISHER, *M.D.*, Los Angeles

CASE REPORTS

56 *Facial Paralysis Occurring with Hypothyroidism—A Report of Two Cases* JERRY M. EARLL, *M.D.*, and FELIX O. KOLB, *M.D.*, San Francisco

58 *Subcutaneous Fat Necrosis with Acute Hemorrhagic Pancreatitis—A Case in a Child with Steroid-Resistant Nephrosis Treated with 6-Mercaptopurine* EVA HEUSER, *M.D.*, ELLIN LIEBERMAN, *M.D.*, GEORGE N. DONNELL, *M.D.*, and BENJAMIN H. LANDING, *M.D.*, Los Angeles

MEDICAL STAFF CONFERENCE

64 *The Spectrum Produced by Malignant Carcinoid Tumor*

CALIFORNIA MEDICAL ASSOCIATION

73 *The Budgeting Process*

INFORMATION

80 *Statutory Consent for Chemical Test of Alcoholic Content of Blood, Breath or Urine in Drunk Driving Cases* HOWARD HASSARD, *LL.B.*, San Francisco

82 *Socialism in Saskatchewan: A Fiasco*

Editorial, 70 *Public Health Report*, 79 *News and Notes*, 83 *Book Reviews*, 85

1967 ANNUAL MEETING, LOS ANGELES, BILTMORE HOTEL, APRIL 15 TO 19, 1967
For Announcement and Hotel Reservations, see pages 77 and 78

OFFICIAL JOURNAL OF THE CALIFORNIA MEDICAL ASSOCIATION

when it counts...

Chloromycetin®

(chloramphenicol)

PARKE-DAVIS

PARKE, DAVIS & COMPANY, Detroit, Michigan 48232

Complete information for usage
available to physicians upon request.

01366

"Yes, Doctor, the pain is gone."

■ Despite introduction of synthetic substitutes, efficacy of 'Empirin' Compound with Codeine remains unchallenged.

'Empirin'® Compound with Codeine Phosphate gr. ½ No. 3
Each tablet contains: Codeine Phosphate gr. ½ (Warning—May be habit forming), Phenacetin gr. 2½, Aspirin gr. 3½, Caffeine gr. ½.

Keeps the Promise of Pain Relief

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N.Y.

Officers OF THE CALIFORNIA MEDICAL ASSOCIATION

General Officers

President: JAMES C. MACLAGGAN, 525 Hawthorn Street, San Diego 92101. 714 233-6519

President-Elect: JOHN G. MORRISON, 333 Estudillo, San Leandro 94577. 415 483-1332

Past President: RALPH C. TEALL, 2626 L Street, Sacramento 95816. 916 448-1851

Speaker of the House of Delegates: WILLIAM F. QUINN, 1401 S. Hope Street, Los Angeles 90015. 213 749-0181

Vice-Speaker of the House of Delegates: JOSEPH W. TELFORD, 3255 Fourth Avenue, San Diego 92103. 714 295-5151

Chairman of the Council: CARL E. ANDERSON, 1150 Montgomery Drive, Santa Rosa 95405. 707 546-1922

Secretary: HELEN B. WEYRAUCH, 450 Sutter Street, San Francisco 94108. 415 392-6909

Editor: DWIGHT L. WILBUR, 655 Sutter Street, San Francisco 94102. 415 673-4080

Executive Director: HOWARD HASSARD, 693 Sutter Street, San Francisco 94102. 415 776-9400

General Counsel: PEART, HASSARD, SMITH and BONNINGTON, 44 Montgomery Street, San Francisco 94104. 415 982-8585

* * *

District Councilors

FIRST DISTRICT—Roger C. Isenhour, (1967), 2881 Fourth Avenue, San Diego 92103. San Diego County. 714 295-2137

SECOND DISTRICT—Imperial, Inyo, Mono, Orange, Riverside and San Bernardino Counties—
- Office No. 1—Henry V. Eastman (1967), 661 W. First Street, Tustin 92680. 714 544-4560
- Office No. 2—Frank C. Melone (1968), 527 N. Palm Avenue, Ontario 91761. 714 986-1104

THIRD DISTRICT—Los Angeles County—
- Office No. 1—Malcolm C. Todd (1968), 2865 Atlantic Avenue, Long Beach 90806. 213 426-1355
- Office No. 2—Elmer F. Gooel (1969), 465 N. Roxbury Drive, Beverly Hills 90210. 213 271-5745
- Office No. 3—Richard L. Taw (1967), 3741 Stocker Street, Los Angeles 90008. 213 293-7157
- Office No. 4—Lewis T. Bullock (1968), 3875 Wilshire Boulevard, Los Angeles 90005. 213 382-2209
- Office No. 5—Joseph P. O'Connor (1969), 595 E. Colorado Boulevard, Pasadena 91101. 213 792-2154
- Office No. 6—Franklin F. Ham (1967), 14556 Hamlin Street, Van Nuys 91401. 213 786-5711
- Office No. 7—Wilbur G. Rogers (1968), 1122 N. Brand Boulevard, Glendale 91202. 213 241-3125
- Office No. 8—Jean F. Crum (1969), 8355 E. 3rd Street, Downey 90241. 213 869-1091
- Office No. 9—Joseph F. Boyle (1967), 1136 W. Sixth Street, Los Angeles 90017. 213 482-9891

FOURTH DISTRICT—Joseph F. Maguire (1967), 2755 Loma Vista Road, Ventura 93003. San Luis Obispo, Santa Barbara and Ventura Counties. 805 648-2567

FIFTH DISTRICT—Ralph W. Burnett (1969), 515 Truxton Avenue, Bakersfield 93301. Calaveras, Fresno, Kern, Kings, Madera, Mariposa, Merced, San Joaquin, Stanislaus, Tulare and Tuolumne Counties. 805 327-4411

SIXTH DISTRICT—Monterey, San Benito, San Mateo, Santa Clara and Santa Cruz Counties.
- Office No. 1—Richard S. Wilbur (1967), 300 Homer Avenue, Palo Alto 94301. 415 321-4121
- Office No. 2—Albert G. Miller (1968), 77 San Mateo Drive, San Mateo 94401. 415 343-9074

SEVENTH DISTRICT—San Francisco County—
- Office No. 1—Malcolm S. M. Watts (1969), 909 Hyde Street, San Francisco 94109. 415 776-3838
- Office No. 2—Roberta F. Fenlon (1967), 490 Post Street, San Francisco 94102. 415 986-1391

EIGHTH DISTRICT—Alameda and Contra Costa Counties—
- Office No. 1—Harold Kay (1969), 411 Thirtieth Street, Oakland 94609. 415 832-4822
- Office No. 2—William F. Kaiser (1967), 3000 Colby Street, Berkeley 94705. 415 843-6515

NINTH DISTRICT—Carl E. Anderson (1967), 1150 Montgomery Drive, Santa Rosa 95405. Del Norte, Humboldt, Lake, Marin, Mendocino, Napa, Solano and Sonoma Counties. 707 546-1922

TENTH DISTRICT—James H. Yant (1968), 2600 Capitol Avenue, Sacramento 95816. Alpine, Amador, Butte, Colusa, El Dorado, Glenn, Lassen, Modoc, Nevada, Placer, Plumas, Sacramento, Shasta, Sierra, Siskiyou, Sutter, Tehama, Trinity, Yolo and Yuba Counties. 916 446-5278

ELEVENTH DISTRICT—Forest J. Grunigen (1967), 3919 Beverly Boulevard, Los Angeles 90004. Forty First Medical Society. 213 663-1161

SCIENTIFIC BOARD REPRESENTATIVE—William P. Longmire, Jr. (1967), UCLA Department of Surgery, Los Angeles 90024. 213 272-8911

* * *

COMMISSION ON *Medical Services*

Ralph Burnett, Bakersfield
Elmer F. Gooel, Beverly Hills (Secty.)
Arthur F. Howard, Fresno (Chmn.)
Charles B. Hudson, Oakland
Homer C. Pheasant, Los Angeles
Milo A. Youel, San Diego.

Committee on Fees

H. Dean Hoskins, Oakland	1967
Wallace A. Gerrie, Jr., Newport Beach	1967
Robert L. Watson, Jr., Los Angeles	1967
Roger C. Isenhour, San Diego	1968
Alfred J. Murrieta, Jr., Los Angeles	1968
Maurice S. Salomon, San Francisco	1968
Homer C. Pheasant, Los Angeles (Chmn.)	1969
Glenn A. Pope, Sacramento	1969
Leo Bell, San Mateo	1969
Joseph F. Boyle, Los Angeles	1969

CONSULTANT: William H. Thompson, San Mateo.

Committee on Federal Medical Care Programs

Charles B. Canby, Van Nuys	1967
Merlin A. Hendrickson, Rialto	1967
Ralph L. Hoffman, San Diego	1967
John S. McKee, Jr., San Diego	1968
Arthur Howard, Fresno (Chmn.)	1968

Committee on Veterans Affairs

Charles B. Hudson, Oakland (Chmn.)
Ben D. A. Miano, San Bernardino
Robert L. Watson, Los Angeles
CONSULTANT: Lloyd E. Gould, Napa.

Committee on Mediation

Eli A. Layon, Brawley	1967
Milo A. Youel, San Diego (Chmn.)	1967
Richard L. Taw, Los Angeles	1968
Albert G. Clark, San Francisco	1969
George Babbin, Sacramento	1969

Committee on Insurance and Prepayment

Oscar W. Hills, San Mateo	1967
Dave F. Dozier, Sacramento	1967
Ralph W. Burnett, Bakersfield (Chmn.)	1968
Stanley J. Murasky, Reseda	1968
Ivan Neubauer, San Jose	1969
Thayer A. Smith, Norwalk	1969

* * *

COMMISSION ON *Public Agencies*

Lewis T. Bullock, Los Angeles
Joseph P. Cosentino, Sacramento (Secty.)
William F. Kaiser, Berkeley
Howard W. Lindsey, Jr., San Mateo
Albert Miller, San Mateo (Chmn.)
Stanley A. Skillicorn, San Jose
Howell E. Wiggins, San Diego
Joseph P. O'Connor, Pasadena
CONSULTANT: Malcolm C. Todd, Long Beach.

Committee on Public Health

Robert L. Garrett, Vallejo	1967
Karl L. Schaupp, Jr., San Francisco	1967
Allen F. Sterling, San Bernardino	1967
Austin W. Matthis, Sacramento	1967
Albert G. Miller, San Mateo	1968
Stanley A. Skillicorn, San Jose (Chmn.)	1968
Alfred F. Kandlbinder, Monterey	1969
Robert Watson, Jr., Los Angeles	1969
Samuel S. Woolington, Long Beach	1969

Committee on Mental Health

Emmanuel Silver, Palo Alto	1967
Ira H. Degenhardt, San Rafael	1967
Rudolph B. Toller, Stockton	1967
John A. Stroud, III, Sacramento	1967
Alfred Auerback, San Francisco	1968
David H. Payne, Los Angeles	1968
Jack B. Lomas, Beverly Hills	1968
Joseph J. Furlong, San Francisco	1969
Stuart C. Knox, Los Angeles (Chmn.)	1969

Committee on Welfare Medical Care Programs

Clyde L. Boice, Palo Alto	1967
Edgar Rosen, Oakland	1967
Thomas N. Elmendorf, Willows	1967
Vincent P. Carroll, Laguna Beach	1967
Orrin S. Cook, Jr., Sacramento	1968
Robert F. Schell, San Rafael	1968
William F. Quinn, Los Angeles	1969
James Dalton, Santa Barbara	1969
Arthur F. Howard, Fresno	1969
Howard W. Lindsey, Jr., San Mateo (Chmn.)	1969

SUBCOMMITTEE ON ADOPTIONS: Walter Beckh, San Francisco; Fred M. Kay, Fullerton; Robert J. McNeil, Los Angeles, (Chmn.); Donald Minkler, Berkeley; Dale W. Ritter, Chico; Ben J. Siebenthal, Sacramento.

Committee on Occupational Health and Rehabilitation

John H. Leimbach, Jr., San Francisco	1967
Herbert A. Holden, San Leandro	1967
Howell E. Wiggins, San Diego (Chmn.)	1967
Edward Zaik, Los Angeles	1968
Packard Thurber, Jr., Los Angeles	1968
Thomas Gucker, III, Los Angeles	1968
Herbert A. Lints, Oakland	1969
Joseph N. Vizzard, Los Gatos	1969
Patrick J. Maloney, Merced	1969

* * *

COMMISSION ON *Community Health Services*

Wayne P. Chesbro, Berkeley
Charles G. Hutter, Jr., Hollywood
Gerald G. Jampolsky, Tiburon (Secty.)
Elmer R. Jennings, Long Beach
Harold Kay, Oakland (Chmn.)
George A. Martin, Redding
Pierre Salmon, San Mateo
Leopold J. Snyder, Fresno
Beverly G. Stewart, Bakersfield

Committee on Rural Health

Mitry P. Ajalat, Calexico	1967
Richard W. Johnson, Arbuckle	1967
Leopold J. Snyder, Fresno (Chmn.)	1968
Erwin P. Brauner, Visalia	1968
Carrol W. Goss, Lamont	1968
Franklin L. Murphy, Oroville	1969
Thomas Rossitto, Auburn	1969
M. Marlin Clark, Delano	1969

Committee on School Health

B. Otis Cobb, Martinez	1967
Gerald G. Jampolsky, Tiburon (Chmn.)	1967
Byron H. Demorest, Sacramento	1967
William A. Leovy, La Mesa	1968
Tom W. Robinson, Newport Beach	1968
Glenn Austin, Los Altos	1968
Ruth A. Frary, Watsonville	1968
Leslie M. Holve, Los Angeles	1969
Henry B. Bruyn, Berkeley	1969

Committee on Health Care of the Aged

Franklin F. Ham, Van Nuys	1967
Pierre Salmon, San Mateo (Chmn.)	1967
Charles E. Schoff, Jr., Sacramento	1968
Raymond D. Manchester, Visalia	1968
Leo A. Wagner, Novato	1969
Lloyd E. Gould, Napa	1969
Walter Batchelder, San Mateo	1969

Committee on Disaster Medical Care

Charles C. Henderson, San Mateo	1967
Donald E. Barker, Van Nuys	1967
Wayne P. Chesbro, Berkeley (Chmn.)	1968
Michael J. Hitchko, Eureka	1968
J. T. McNally, Stockton	1969
John Heald, San Francisco	1969

CONSULTANTS: Charles N. Earl, Arcata; Frank F. Schade, Los Angeles; Robert S. Johnson, Chico; Stephen Bruny, Fresno.

Committee on Automotive and Traffic Safety

George C. Snively, Sacramento	1967
Beverly G. Stewart, Bakersfield (Chmn.)	1967
Arnold R. Friesen, Pasadena	1968
Joseph F. Maguire, Ventura	1969
Thomas W. Lyons, La Mesa	1969
Robert L. Tour, San Francisco	1969

CONSULTANTS: Julian A. Waller, Berkeley; Julian M. Sether, Los Angeles.

Committee on Medical Aspects Of Sports and Physical Fitness

John F. Blinn, Stockton	1967
Robert K. Kerlan, Los Angeles	1967
George S. Buehler, Whittier	1968
Philip H. McFarland, Fullerton	1968
Tom W. Robinson, Newport Beach	1968
Charles G. Hutter, Jr., Hollywood (Chmn.)	1969
Arnold A. Nutting, San Rafael	1969

Committee on Environmental Health

George A. Martin, Redding	1967
Robert L. Hippen, San Diego	1967
Grace M. Talbott, San Francisco	1968
Francis M. Pottenger, Jr., Monrovia	1968
Reginald H. Smart, Los Angeles	1968
Douglas Campbell, San Francisco	1969
John R. Lee, Mill Valley	1969

Committee on Blood Banks

Robert W. Purvis, Modesto	1967
Osman W. Hull, Monterey	1967
James H. Yant, Sacramento	1967
Elmer Jennings, Long Beach (Chmn.)	1968

CONSULTANT: Melvin L. Sommer, Beverly Hills.

COMMISSION ON Communications

Frederick Ackerman, Pleasant Hill
L. Morgan Boyers, Sacramento (Secty.)
John A. E. Bullis, Los Angeles
Roberta F. Fenlon, San Francisco
Elmer F. Gooel, Beverly Hills
Walter W. Hopps, Los Angeles
George Schwartz, Beverly Hills
Malcolm C. Todd, Long Beach (Chmn.)
Richard S. Wilbur, Palo Alto
EX-OFFICIO: James C. MacLaggan, San Diego; John G. Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Scientific Board

David A. Rytand, Palo Alto	1967
William W. Belford, San Diego	1967
Jerome W. Bettman, San Francisco	1967
John B. Dillon, Los Angeles	1967
Justin J. Stein, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
R. Kirklin Ashley, San Francisco	1967
Victor Richards, San Francisco	1967
David Rubin, Los Angeles	1967
Keith P. Russell, Los Angeles	1967
Edward J. Zaik, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Seymour J. Brockman, Los Angeles	1968
S. Austin Jones, Los Angeles	1968
Hobart M. Kelly, Riverside	1968
Saul J. Robinson, San Francisco	1968
Eugene Ziskind, Los Angeles	1968
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. deC. M. Saunders, San Francisco	1968
Herbert A. Holden, San Leandro	1968
David A. Wood, San Francisco	1968
Arthur A. Clinco, Los Angeles	1968
R. Theodore Bergman, Los Angeles	1968
Leland B. Blanchard, San Jose	1969
L. S. Goerke, Los Angeles	1969
Bert L. Halter, San Francisco	1969
Ellis Jones, Pasadena	1969
Carl McCandless, San Francisco	1969
J. Blair Pace, Oceanside	1969
Donald W. Petit, Los Angeles	1969
Glenn Pope, Sacramento	1969
Robert J. Prentiss, San Diego	1969
Edward B. Shaw, San Francisco	1969
Ralph E. Worden, Los Angeles	1969

COUNCIL REPRESENTATIVES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.
EX-OFFICIO: Dwight L. Wilbur, San Francisco.

Executive Committee

William W. Belford, San Diego	1967
Victor Richards, San Francisco	1967
Justin J. Stein, Los Angeles	1967
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. Dillon, Los Angeles	1969
Donald Petit, Los Angeles	1969

COUNCIL DESIGNEES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.

Committee on Nominations

Justin J Stein, Los Angeles (Chmn.)	1967
Edward B. Shaw, San Francisco	1969
Robert J. Prentiss, San Diego	1969

Committee on Scientific Assemblies

John B. Dillon, Los Angeles (Chmn.)	1967
Victor Richards, San Francisco	1967
David A. Rytand, Palo Alto	1967
John B. deC. M. Saunders, San Francisco	1968
Ellis Jones, Pasadena	1969
Donald Petit, Los Angeles	1969

CONSULTANT: Claron Oakley, Los Angeles

Committee on Continuing Medical Education

John B. Dillon, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
Saul J. Robinson, San Francisco	1968
Donald W. Petit, Los Angeles (Chmn.)	1969
Bert L. Halter, San Francisco	1969
J. Blair Pace, Oceanside	1969

Committee on CALIFORNIA MEDICINE

David Rubin, Los Angeles	1967
R. Kirklin Ashley, San Francisco	1967

Committee on Scientific Information

Justin J. Stein, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Arthur A. Clinco, Los Angeles	1968
Edward B. Shaw, San Francisco (Chmn.)	1969
L. S. Goerke, Los Angeles	1969

Committee on Maternal and Child Care

Russell W. Mapes, Beverly Hills	1967
Keith P. Russell, Los Angeles (Chmn.)	1967
Forrest H. Howard, Garden Grove	1967
Charles M. Blumenfeld, Sacramento	1968
Herbert A. Holden, San Leandro	1968
Hobart M. Kelley, Riverside	1968
Leland Blanchard, San Jose	1969

Committee on Cancer

John W. Cline, San Francisco	1967
Robert W. Jamplis, Palo Alto	1967
David A. Wood, San Francisco (Chmn.)	1967
John G. Walsh, Sacramento	1968
Lewis W. Guiss, Los Angeles	1969
Robert Prentiss, San Diego	1969

SUBCOMMITTEE ON CANCER EDUCATION: Sol R. Baker, Beverly Hills; Robert W. Jamplis, Palo Alto (Chmn.); Maurice Galante, San Francisco; Justin J. Stein, Los Angeles.

SUBCOMMITTEE ON TUMOR TISSUE REGISTRY: John W. Cline, San Francisco (Chmn.); Lewis W. Guiss, Los Angeles; James E. Kahler, Los Angeles; Carl M. McCandless, San Francisco; Weldon K. Bullock, Los Angeles.

SUBCOMMITTEE ON CONSULTATIVE TUMOR BOARDS: Bert L. Halter, San Francisco; Carl L. Moore, Bakersfield; Victor Richards, San Francisco; John G. Walsh, Sacramento (Chmn.).

Committee on Adverse Drug Reactions

Edward R. Bloomquist, Glendale	1967
Ralph O. Wallerstein, San Francisco	1967
John F. Murray, Fresno (Chmn.)	1968
Russell J. Tat, San Francisco	1969
William W. Thayer, Stockton	1969

CONSULTANT: John W. Brown, Berkeley; Ralph W. Weilerstein, Berkeley.

SUBCOMMITTEE ON DANGEROUS DRUGS: Edward Bloomquist, Glendale; Norman A. Gale, San Diego; Edward T. Jewett, Santa Rosa; William F. Quinn, Los Angeles (Chmn.); Helen B. Weyrauch, San Francisco.
CONSULTANT: Ralph W. Weilerstein, Berkeley.

Committee on Cardiovascular Diseases

Harold I. Griffeath, San Francisco (Chmn.); Lewis T. Bullock, Los Angeles; David J. New, Modesto; George C. Griffith, Los Angeles.
CONSULTANT: John E. Connolly, Los Angeles.

* * *

BUREAU OF Research and Planning

J. Brandon Bassett, Oakland
John Sheehy, Riverside
Samuel Sherman, San Francisco (Chmn.)
H. Russell Fisher, Glendale
G. Roger Ellison, Pomona
EX-OFFICIO: Malcolm S. M. Watts, San Francisco; Ralph C. Teall, Sacramento.
CONSULTANT: Gerald W. Shaw, Santa Monica.

* * *

COMMISSION ON Professional Welfare

Bert L. Halter, San Francisco
George K. Herzog, Jr., San Francisco (Chmn.)
Francis E. West, San Diego
James H. Yant, Sacramento (Secty.)

Committee on Physicians Group Insurance

Robert J. Wescott, El Centro	1967
Robert X. DeMangus, Glendale	1967
John B. Long, Sacramento	1967
George K. Herzog, Jr., San Francisco (Chmn.)	1968
John Hogan, San Diego	1969
Homer C. Pheasant, Los Angeles	1969

Liaison Committee to Medical Schools

Samuel R. Irvine, Beverly Hills	1967
Francis E. West, San Diego	1967
Frank J. Novak, Menlo Park	1968
Stanley Cowell, Los Angeles	1968
Bert L. Halter, San Francisco (Chmn.)	1969
Alonzo Neufeld, Los Angeles	1969
Herbert Jenkins, Sacramento	1969

EX-OFFICIO: James C. MacLaggan, San Diego; John G. Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Medical Review and Advisory Committee

Eugene M. Webb, San Francisco	1967
James H. Yant, Sacramento (Chmn.)	1967
John B. Dillon, Los Angeles	1968
Carl Goetsch, Berkeley	1968
Donald Donisthorpe, Los Angeles	1969

CONSULTANT: Don Harper Mills, Long Beach.

Liaison Committee to State Bar of California

David Rubsamen, Berkeley	1967
John V. Fiore, Westchester	1967
Francis E. West, San Diego (Chmn.)	1968
Carl Goetsch, Berkeley	1968
Robert J. McNeil, Los Angeles	1969
Henry A. Brown, San Mateo	1969

* * *

COMMISSION ON Allied Health Professions and Services

Roger W. Barnes, Los Angeles (Secty.)
Forest J. Grunigen, Los Angeles (Chmn.)
Frank C. Melone, Ontario
Thomas W. Ledwich, Napa
C. Gerald Scarborough, San Jose
Richard H. Schug, Long Beach

Committee on Paramedical Personnel

Frank C. Melone, Ontario (Chmn.)	1967
Gregory Bard, San Francisco	1968
Elmer R. Jennings, Long Beach	1968
Gerson R. Biskind, San Francisco	1969
Joseph Schaefer, Santa Rosa	1969
Leonard M. Asher, Los Angeles	1969
Richard H. Schug, Long Beach (Chmn.)	1969

Committee on Other Professions

George S. Buehler, Whittier	1967
Burt L. Davis, Palo Alto	1967
C. Gerald Scarborough, San Jose (Chmn.)	1968
William D. Evans, Los Angeles	1968
J. Gordon Epperson, Oakland	1969
Lewis T. Bullock, Los Angeles	1969

Liaison Committee to California Medical Assistants Association

Leon O. Burke, Sacramento	1967
John C. Brennan, Pico Rivera	1967
H. Russell Irwin, San Diego	1967
Peary Benn Berger, Inglewood	1968
Judith D. Smith, Vallejo	1969
Sanford E. Feldman, San Francisco	1969
Richard H. Schug, Long Beach (Chmn.)	1969

CONSULTANTS: Leland W. Bonar, Redding; Henry Bodner, Van Nuys.

Committee on Medicine and Religion

Albert E. Long, Daly City	1967
Roger W. Barnes, Los Angeles (Chmn.)	1967
Max Nelson, National City	1968
Frank J. Glassy, Sacramento	1969
Richard J. Lescoe, Torrance	1969

Committee on Voluntary Health Agencies

H. Mason Hohl, Beverly Hills	1967
Thomas W. Ledwich, Napa (Chmn.)	1968
Harvey May Cordua, Jr., San Diego	1969
J. Brandon Bassett, Oakland	1969

* * *

Judicial COMMISSION

Donald A. Charnock, Los Angeles	1967
Albert E. Long, Daly City	1967
Donald Cass, Hollywood	1967
Floyd O. Due, Oakland	1968
Sidney J. Shipman, San Francisco	1968
Francis E. West, San Diego (Chmn.)	1968
Douglas G. Campbell, San Francisco	1969
W. Philip Corr, Riverside (Secty.)	1969
L. E. Wilson, Anaheim	1969

* * *

COMMISSION ON Hospital Affairs

William L. Argo, Fresno (Secty.)
James C. Doyle, Beverly Hills
Bert L. Halter, San Francisco (Chmn.)
John C. Lungren, Long Beach
Glenn Pope, Sacramento
John T. Saidy, San Mateo
Joseph W. Telford, San Diego
Frederic N. Tyroler, Los Angeles

Medical Staff Survey Committee

Robert M. Adams, San Leandro
W. L. Argo, Fresno
Daniel W. Black, Castro Valley
Rush M. Blodget, Jr., Redding
Bert L. Halter, San Francisco (Chmn.)
J. Ralph Hughes, San Diego
Wm. Kenneth Jennings, Santa Barbara
Donald O. Lagerlof, Los Angeles
John C. Lungren, Long Beach
William A. Newsom, San Francisco
James L. Rhee, Oakland
John T. Saidy, San Mateo
Wilfred J. Snodgrass, Santa Monica
Alexander Hatoff, Oakland
Shepard F. Palitz, Bakersfield
William L. Sumption, Santa Monica
A. Jerome Hurter, Pasadena
Ernest R. Barnett, Alhambra
Richard L. Taw, Los Angeles
Richard H. McDonald, Garden Grove
Robert M. Zweig, Riverside
L. Morgan Boyers, Sacramento
James O. Farley, Sacramento
Joseph W. Telford, San Diego
Paul Deeb, Redlands
Maxwell Goldstein, Fontana
Ray Seavers, Jr., San Bernardino
William Soldmann, La Mesa
Lloyd Milburn, San Francisco
Frank Solomon, San Francisco
Sanford Feldman, San Francisco
John Bazzano, San Francisco
Kenneth Allen, San Mateo
Martin Kohn, San Bruno
William Stephenson, Santa Barbara
Merlin Hendrickson, Rialto
Andrew D. Lucine, Campbell
Fred Haven Draper, San Jose
Wayne O. Buck, San Jose
William A. Reeves, Mt. View
A. A. Thurlow, Jr., Santa Rosa
John A. Carollo, Sabastopol
William Rector, Sacramento
Gerald W. Shaw, Santa Monica
Charles L. Lear, Norwalk
John R. Heckman, Marysville
Robert Buckley, Santa Monica
Woodrow W. Burgess, Sacramento
Ira H. Degenhardt, San Rafael
Arthur E. Gladman, Oakland
Norman Graff, San Mateo
Frank V. Hoffman, Huntington Beach
Robert E. James, Belmont
Clement O. Juul, Oakland
Seymour Kolko, San Mateo
Melvin M. Lipsett, Berkeley
Jack B. Lomas, Los Angeles
Edward A. Macklin, San Francisco
D. Stuart MacRobbie, Berkeley
Felix H. Ocko, Berkeley
William R. Sheehy, Berkeley
John A. Stroud, Sacramento
R. J. Sutherland, Oakland
Rudolph B. Toller, Stockton
Albert T. Voris, Belmont
G. Cameron Cray, Palo Alto

(Continued on Page 24)

Officers of Scientific Sessions

Members who contemplate presentation of scientific papers should promptly address the secretary of the proper section at the address shown below.

Allergy

Leo N. Meleyco..........Chairman
2040 Forest Avenue, San Jose 95128
Ernest J. Saslow..........Secretary
2011-18th Street, Bakersfield 93301
Samuel Ross..........Assistant Secretary
2946 Fresno Street, Fresno 93706

Anesthesiology

Thomas W. McIntosh..........Chairman
686 East Union St., Pasadena 91101
Donald B. Dose..........Secretary
5625 Linfield Ave., San Diego 92120
Donald A. Walker..........Assistant Secretary
35 Spring Lane, Tiburon 94920

Dermatology

Robert G. Walton..........Chairman
1700 McHenry Village Way, Modesto 95350
Samuel Ayres, III..........Secretary
405 N. Bedford Rd., Beverly Hills 90210
George T. Wilson..........Assistant Secretary
Welch and Pasteur Rd., Stanford Medical Plaza
Palo Alto 94304

General Practice

J. Blair Pace..........Chairman
408 Cassidy St., Oceanside 92054
Alfred F. Kandlbinder..........Secretary
302 Via Paraiso St., Monterey 93940
Allen F. Sterling..........Assistant Secretary
1285 D St., San Bernardino 92045

General Surgery

Seibert Pearson..........Chairman
2865 Atlantic Ave., Long Beach 90807
Albert G. Clark..........Secretary
450 Sutter St., San Francisco 94108
Robert T. Hood, Jr...........Assistant Secretary
2010 Wilshire Blvd., Los Angeles 90057

Industrial Medicine and Surgery

Leon R. Rudnick..........Chairman
15921 E. 14th St., San Leandro 94578
Raymond O. Oja..........Secretary
525 Golden Gate Ave., San Francisco 94102
James G. Forgerson..........Assistant Secretary
Metropolitan Life Insurance Company
600 Stockton St., San Francisco 94108

Internal Medicine

Robert L. Paver..........Chairman
384 Post St., San Francisco 94108
John L. Benton, Jr...........Secretary
1216 Wilshire Blvd., Los Angeles 90017
Edgar Rosen..........Assistant Secretary
1340 K St., Sacramento 95814

Obstetrics and Gynecology

Lester T. Hibbard..........Chairman
1930 Wilshire Blvd., Los Angeles 90057
Warren E. Jones..........Secretary
1380-45th Street, Sacramento 95819
James W. Ravenscroft..........Assistant Secretary
2330 First Ave., San Diego 92101

Ophthalmology

Victor G. Fellows..........Chairman
595 Buckingham Way, San Francisco 94117
Wendell C. Irvine..........Secretary
3875 Wilshire Blvd., Los Angeles 90005
Robert E. Hopkins..........Assistant Secretary
4255 Pacific Ave., Stockton 95207

Orthopedics

David C. Monsen..........Chairman
2300 S. Hope St., Los Angeles 90007
Arthur R. Hartwig..........Secretary
490 Post St., San Francisco 94102
Vernon L. Smythe..........Assistant Secretary
3875 Wilshire Blvd., Los Angeles 90005

Otolaryngology

G. Howard Gottschalk..........Chairman
8618 S. Sepulveda Blvd., Los Angeles 90045
Charles P. Lebo..........Secretary
490 Post St., San Francisco 94102
Irving L. White..........Assistant Secretary
911 San Antonio Dr., Long Beach 90807

Pathology

Frank R. Dutra..........Chairman
20103 Lake Chabot Rd., Castro Valley 94546
William H. Kern..........Secretary
679 S. Westlake Ave., Los Angeles 90057
Donald L. Bittner..........Assistant Secretary
Bay Area Laboratories, 2245 Post St.,
San Francisco 94115

Pediatrics

Richard L. Anderson..........Chairman
636 Harris St., Eureka 95501
Sherrod C. Swift..........Secretary
14619 Whittier Blvd., Whittier 90605
Robert J. Harvey..........Assistant Secretary
909 Hyde St., San Francisco 94109

Physical Medicine

Rene Cailliet..........Chairman
4900 Sunset Blvd., Los Angeles 90027
Anton A. Tratar..........Secretary
2360 Clay St., San Francisco 94115
Elizabeth Austin..........Assistant Secretary
Los Angeles County Hospital
1200 N. State St., Los Angeles 90033

Preventive Medicine

Henrik L. Blum..........Chairman
P. O. Box 871, Martinez 94554
Maxwell Rosenblatt..........Secretary
Los Angeles County Health Department,
220 N. Broadway, Los Angeles 90012
Russell B. Watson..........Assistant Secretary
Orange County Health Department, Santa Ana
92701

Psychiatry and Neurology

Werner M. Mendel..........Chairman
1934 Hospital Place, Los Angeles 90033
Ames Fischer..........Secretary
Langley Porter Inst., 401 Parnassus Ave.,
San Francisco 94122
Howard S. Barrows..........Assistant Secretary
2025 Zonal Ave., Los Angeles 90033

Radiology

Victor G. Mikity..........Chairman
1200 State St., Los Angeles 90033
Vernon R. Gee..........Secretary
2020 Court St., Redding 96001
Richard P. Storrs..........Assistant Secretary
2131 West 3rd St., Los Angeles 90057

Urology

Carl K. Pearlman..........Chairman
1518 N. Sycamore, Santa Ana 92701
James L. Goebel..........Secretary
51 Loma Linda, Ross 94957
Robert O. Pearman..........Assistant Secretary
5353 Balboa Blvd., Encino 91316

Delegates and Alternates

TO THE AMERICAN MEDICAL ASSOCIATION

DELEGATES		ALTERNATES
Francis E. West, San Diego	(1966-1967)	Vincent P. Carroll,* Laguna Beach
Samuel R. Sherman, San Francisco	(1966-1967)	Albert G. Miller, San Mateo
Henry Gibbons, III, San Francisco	(1966-1967)	A. B. Sirbu, San Francisco
John M. Rumsey, San Diego	(1966-1967)	Carl E. Anderson, Santa Rosa
Eugene F. Hoffman, Los Angeles	(1966-1967)	Joseph F. Boyle, Los Angeles
Warren L. Bostick, Los Angeles	(1966-1967)	Walter H. Brignoli, St. Helena
J. B. Price, Santa Ana	(1966-1967)	Donald C. Dodds, Oakland
Ralph C. Teall, Sacramento	(1966-1967)	Robb Smith, Orange Cove
James C. Doyle, Beverly Hills	(1966-1967)	Herman H. Stone, Riverside
Wilbur G. Rogers, Glendale	(1966-1967)	Carl M. Hadley, San Bernardino
Charles B. Hudson, Oakland	(1966-1967)	Robert L. Watson, Jr., Los Angeles
John G. Morrison, San Leandro	(1967-1968)	Frederick Ackerman, Pleasant Hills
William F. Quinn, Los Angeles	(1967-1968)	Dudley M. Cobb, Los Angeles
Robert C. Combs, San Francisco	(1967-1968)	Emmett Rixford, San Francisco
Homer C. Pheasant, Los Angeles	(1967-1968)	James C. MacLaggan, San Diego
Alfred J. Murrieta, Los Angeles	(1967-1968)	Harold Kay, Oakland
Burt L. Davis, Palo Alto	(1967-1968)	Leon P. Fox, San Jose
Arlo A. Morrison, Ventura	(1967-1968)	Henry Brown, San Jose
James E. Feldmayer, Exeter	(1967-1968)	Charles Grayson, Sacramento
O. W. Wheeler, Riverside	(1967-1968)	Wilbur Bailey, Los Angeles
Malcolm C. Todd, Long Beach	(1967-1968)	Neil C. Hamel, Encino
J. E. Vaughan, Bakersfield	(1967-1968)	Joseph P. O'Connor, Pasadena

(Delegates and Alternates to the AMA are elected for terms of two calendar years. Each office is scheduled for election at the Annual Session of the second year shown above.) *Council appointee.

Other Organizations and Medical Schools

Board of Medical Examiners of the State of California

Secretary..........Shelby M. Hicks, M.D.
1021 O Street, Room A202, Sacramento 95814
San Francisco..........30 Van Ness Ave., 94102
Room 2100
Los Angeles..........107 South Broadway 90012
Sacramento..........1021 O Street, Room A547 95814

The Public Health League of California

Executive Secretary..........Ben H. Read
955 South Western Ave., Los Angeles 90006
Assistant Executive Secretary..........Al Paul Putnam
693 Sutter Street, San Francisco 94102
San Francisco..........693 Sutter Street 94102
415 474-1061
Los Angeles..........955 South Western Ave. 90006
213 731-6397
Sacramento..........1127-11th Street, 95814
916 444-7496

Department of Public Health of the State of California

Director..........Lester Breslow, M.D.
2151 Berkeley Way, Berkeley 94704
Berkeley..........2151 Berkeley Way 94704
415 843-7900
Sacramento..........1927-13th Street 95814
916 445-2264
Los Angeles..........417 South Hill Street 90013
213 620-2900

Medical Schools in California

University of California School of Medicine, Medical Center, San Francisco 94122. Dean: Stuart C. Cullen, M.D.

Stanford University School of Medicine, 300 Pasteur Drive, Palo Alto 94304. Dean: Robert J. Glaser, M.D.

University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 90033. Dean: Roger O. Egeberg, M.D.

Loma Linda University School of Medicine, Loma Linda 92354. Dean: David Burdg Hinshaw, M.D.

University of California at Los Angeles, School of Medicine, Hilgard Avenue, Los Angeles 90024. Dean: Sherman M. Mellinkoff, M.D.

University of California, California College of Medicine, 1721 Griffin Avenue, Los Angeles 90031. Dean: Warren L. Bostick, M.D.

University of California, San Diego, School of Medicine, La Jolla 92038. Acting Dean: Robert D. Tschirgi, M.D.

University of California, Davis, School of Medicine, Davis 95616. Dean: Charles John Tupper, M.D.

California Medical Education and Research Foundation

Directors: Ralph C. Teall, M.D., President; Carl E. Anderson, M.D., Vice-President; Howard Hassard, Secretary; James C. MacLaggan, M.D.; Harold Kay, M.D.; William F. Quinn, M.D.; E. B. Shaw, M.D.

Roster — CALIFORNIA COMPONENT MEDICAL SOCIETIES

Society secretaries are requested to notify California Medicine promptly when changes are indicated in their roster information

ALAMEDA-CONTRA COSTA Medical Assn., 6230 Claremont Ave., Oakland 94618. Meets quarterly.
Frederick W. Ackerman........President
2100 Monument Blvd., Pleasant Hill 93542
Herbert A. Holden........Secretary
1436 Clarke St., San Leandro 94577

BUTTE-GLENN Medical Society, P. O. Box 1008, Chico 95927. Meets Fourth Thursday.
Walter S. Mazen........President
1617 Esplanade, Chico 95926
John B. Copeland........Secretary
1423 Magnolia, Chico 95926

FORTY FIRST Medical Society, Suite 821, 1515 N. Vermont Ave., Los Angeles 90027
Paul D. Yates........President
844 Hermosa Ave., Hermosa Beach 90254
Jordan M. Phillips........Secretary
11239 S. Lakewood Blvd., Downey 90241

FRESNO County Medical Society, 1118 Divisadero St., Fresno 93721. Meets Second Tuesday, 6:30 p.m., Sunnyside Country Club.
Max Millar........President
3004 N. Fresno St., Fresno 93703
Alan T. Sortor........Secretary
1340 E. Belmont St., Fresno 93701

HUMBOLDT-DEL NORTE County Medical Society. Meets Second Thursday.
Wells C. Carey........President
2822 E St., Eureka 95501
Max Todd........Secretary
616-14th St., Arcata 95521

IMPERIAL County Medical Society, P. O. Box 518, Imperial 92251. Meets Second Tuesday, 8 p.m., Pioneer Memorial Hospital, Brawley.
O. G. Fais........President
1745 S. Imperial Ave., El Centro 92243
Elmer C. Werner........Secretary
1745 S. Imperial Ave., El Centro 92243

INYO-MONO County Medical Society. Meets Fourth Tuesday except December, January, February.
Milton R. Jones, Jr........President
Box 38, Lone Pine 93545
Robert W. Denton........Secretary
611 W. Line St., Bishop 93514

KERN County Medical Society, 2603 G Street, Bakersfield 93301. Meets Third Tuesday, 7:30 p.m., Society Office, 2603 G Street, except June, July, August.
Keith Spaulding........President
2521 "G" St., Bakersfield 93301
James Friend........Secretary
1921-18th St., Bakersfield 93301

KINGS County Medical Society. Meets Second Thursday, 7:00 p.m. Place to be announced.
James Dean........President
912 N. Douty St., Hanford 93230
Willard S. Bridwell........Secretary
1507 N. 11th Ave., Hanford 93230

LASSEN-PLUMAS-MODOC-SIERRA County Medical Society. Meets on call.
Kenneth G. Korver........President
50 N. Roop St., Susanville 96130
James McCullough........Secretary
50 N. McDow St., Susanville 96130

LOS ANGELES County Medical Assn., 1925 Wilshire Blvd., Los Angeles 90057.
Joseph F. Boyle........President
1136 W. Sixth St., Los Angeles 90017
Homer C. Pheasant........Secretary
2300 S. Hope St., Suite 300,
Los Angeles 90007

MARIN Medical Society, 1601 Second St., Suite 106, San Rafael 94901. Meets First Thursday, 7:00 p.m.
Robert Mills........President
711 D St., San Rafael 94901
William A. Galeno........Secretary
712 D St., San Rafael 94901

MENDOCINO-LAKE County Medical Society.
Frank H. Dailey........President
719 S. Dora, Ukiah 95482
Patrick R. Allanson........Secretary
765 S. Dora St., Ukiah 95482

MERCED-MARIPOSA Medical Society, P. O. Box 586, Merced 95340. Meets Fourth Thursday; meeting place changes.
Patrick J. Maloney........President
P. O. Box 586, Merced 95340
Robert J. Schiffler........Secretary
P. O. Box 586, Merced 95340

MONTEREY County Medical Society, P. O. Box 308, Salinas 93903. Meets First Thursday.
Joseph E. Turner........President
1010 Cass St., Monterey 93940
Thomas S. Elliott........Secretary
101 Maple St., Greenfield 93927

NAPA County Medical Society, P. O. Box 2158, Napa 94559. Meets Second Wednesday.
Richard G. Clark........President
3020 Beard Rd., Napa 94559
Herbert Waechlter........Secretary
980 Trancas St., Napa 94559

ORANGE County Medical Association, 300 S. Flower, Orange 92666. Meets First Tuesday, 7:00 p.m.
Wallace A. Gerrie........President
1901 Westcliff Dr., Newport Beach 92660
David I. Nielsen........Secretary
400 Westminster Ave., Newport Beach 92660

PLACER-NEVADA County Medical Society. Meets Second Wednesday.
F. Harold Johnson........President
305 Oak Ridge Dr., Roseville 95678
Arthur R. Weaver........Secretary
1212 High St., Suite 106, Auburn 95603

RIVERSIDE County Medical Association, 4175 Brockton Ave., Riverside 92501. Meets Second Monday, 7:30 p.m., Spanish Arts Gallery, Mission Inn.
Roger W. Ridley........President
6969 Brockton Ave., Riverside 92506
Richard W. Trotter........Secretary
6876 Magnolia Ave., Riverside 92506

SACRAMENTO County Medical Society, 5380 Elvas Ave., Sacramento 95819. Meets Third Tuesday, 8:30 p.m. Location of meetings varies.
Orrin S. Cook........President
1116-26th St., Sacramento 95816
Pierce A. Rooney, Jr.........Secretary
2600 Capitol Ave., Sacramento 95816

SAN BENITO County Medical Society. Meets once a month, except July and August. Time and place to be announced.
Robert D. Quinn........President
930 Sunset Dr., Hollister 95023
Roderick L. Yip........Secretary
891 Sunset Dr., Hollister 95023

SAN BERNARDINO County Medical Society, 1875 N. D St., San Bernardino 92405. Meets First Tuesday, 7:30 p.m., Harold's Club, Fontana.
Harold Hill........President
219 Cajon Ave., Redlands 92373
Nicholas P. Krikes........Secretary
25060 Base Line, San Bernardino 92410

SAN DIEGO County Medical Society, 3427-4th Ave., San Diego 92103. Meets Second Tuesday. Location of meetings varies.
Sam Peck........President
3427-4th Ave., San Diego 92103
W. C. Herrick........Secretary
233 A St., San Diego 92101

SAN FRANCISCO Medical Society, 250 Masonic Ave., San Francisco 94118.
Albert G. Clark........President
450 Sutter St., San Francisco 94108
Harry W. Sawyer........Secretary
909 Hyde St., San Francisco 94109

SAN JOAQUIN County Medical Society, 445 W. Acacia St., P. O. Box 230, Stockton 95201. Meets First Thursday, 8:15 p.m.
Thomas Huff........President
512 W. Harding Way, Stockton 95204
Dora A. Lee........Secretary
2420 N. California St., Stockton 95204

SAN LUIS OBISPO County Medical Society, P.O. Box 319, San Luis Obispo 93402. Meets Third Saturday, 7:00 p.m., Madonna Inn, San Luis Obispo 93402.
Robert Buck........President
P. O. Box 319, San Luis Obispo 93402
Billy W. Mounts........Secretary
P. O. Box 319, San Luis Obispo 93402

SAN MATEO County Medical Society, 122 El Camino Real, San Mateo 94401. Meets Third Tuesday.
Howard W. Lindsey, Jr........President
323 N. San Mateo Dr., San Mateo 94401
Martin M. Kohn........Secretary
841 W. San Bruno Ave., 94066

SANTA BARBARA County Medical Society, 300 W. Pueblo St., Santa Barbara 93105. Meets Second Monday of the month, University Club, 1332 Santa Barbara St., Santa Barbara.
T. K. Hill........President
1421 State St., Santa Barbara 93105
Casimir Domz........Secretary
317 W. Pueblo St., Santa Barbara 93105

SANTA CLARA County Medical Society, 700 Empey Way, San Jose 95128. Meets First Monday.
Stanley A. Skillicorn........President
700 Empey Way, San Jose 95128
Donald W. S. Stiff........Secretary
700 Empey Way, San Jose 95128

SANTA CRUZ County Medical Society. Meets every Second Month, Second Tuesday. Time, place to be announced.
W. W. Hoskins........President
30 Brennan St., Watsonville 95076
William Blade........Secretary
1510 Seabright Ave., Santa Cruz 95062

SHASTA-TRINITY County Medical Society. Meets Second Monday of the month. Place to be announced.
Faylon M. Brunemeier........President
2020 Court St., Redding 96001
Eugene A. Nutley........Secretary
2625 Edith Ave., Redding 96001

SISKIYOU County Medical Society. Meets Sunday on call.
Harry B. Chappell........President
201 Stagecoach Rd., Dunsmuir 96025
Robert L. Anderson........Secretary
504 Hillcrest St., Yreka 96097

SOLANO County Medical Society, 631 Tennessee St.. Vallejo 94599.
Tuesday, 8:00 p.m., at different meeting places.
Robert L. Garrett........President
1512 Napa St., Vallejo 94590
Keith E. Vincent........Secretary
2121 Redwood St., Vallejo 94594

SONOMA County Medical Society, 1625 Franklin Ave., Santa Rosa 95404. Meets Second Thursday.
John S Dunn........President
576 B St., Santa Rosa 95401
Elizabeth Buckley........Secretary
1140 Sonoma Ave., Santa Rosa 95405

STANISLAUS County Medical Society, 1125-13th St., Modesto 95354. Meets Third Tuesday of the month, 7 p.m., Hotel Covell, Modesto.
Howard Kuhl........President
1700 McHenry Ave., Modesto 95350
Jeanne I. Miller........Secretary
709-18th St., Modesto 95354

TEHAMA County Medical Society. Meets at call of President.
Hugh R. Merhoff........President
737 Washington St., Red Bluff 96080

TULARE County Medical Society, 1640 West Mineral King. P. O. Box 16, Visalia 93278.
Irving M. Schor........President
P. O. Box 1151, Visalia 93277
George Tiss........Secretary
P. O. Box 7, Visalia 93277

VENTURA County Medical Society, 3291 Loma Vista Rd., Room 40, Ventura 93003. Meets Second Tuesday each month, 7:30 p.m., Los Posas Country Club, Camarillo.
Joseph F. Gstettenbauer........President
3003 Loma Vista Rd., Ventura 93003
Franklin K. Helbling........Secretary
3291 Loma Vista Rd., Room 40, Ventura 93003

YOLO County Medical Society. Meets First Wednesday.
Benjamin R. Robinson........President
650 Third St., Woodland 95695
Corbin Young........Secretary
101 Main St., Winters 95694

YUBA-SUTTER-COLUSA County Medical Society.
John R. Heckman........President
714-14th St., Marysville 95901
Norman K. Noordhoff........Secretary
320 G St., Marysville 95901

Officers — CALIFORNIA PHYSICIANS' SERVICE — 1966-67

Officers:

Chairman of the Board	Richard S. Wilbur
Vice-Chairman of the Board	Milo A. Youel
Secretary	Wilbur G. Rogers
Treasurer	Bert L. Halter

BOARD OF *Trustees:*

Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Roberta F. Fenlon*	San Francisco
Donald R. Fitch	Glendale
Mr. Thomas Hadfield	San Francisco
Bert L. Halter	San Francisco
Donald C. Harrington	Stockton
M. M. Haskell	Long Beach
Carl Goetsch	Berkeley
Mr. Philip S. Magruder	Flintridge
Gregory C. Murray	Los Angeles
Wilbur G. Rogers*	Glendale
Mr. Paul Schrade	Los Angeles
William H. Thompson	San Mateo
Richard S. Wilbur*	Palo Alto
Milo A. Youel	San Diego

---

*Appointed by CMA Council

BOARD COMMITTEES:

Executive Committee

Richard S. Wilbur, Chairman	Palo Alto
Carl Goetsch	Berkeley
Bert L. Halter	San Francisco
Mr. Philip S. Magruder	Flintridge
Wilbur G. Rogers	Glendale
William H. Thompson	San Mateo
Milo A. Youel	San Diego

Finance Committee

Bert L. Halter, Chairman	San Francisco
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Donald R. Fitch	Glendale
Mr. Philip S. Magruder	Flintridge

Medical Policy Committee

Wilbur G. Rogers, Chairman	Glendale
Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Roberta F. Fenlon	San Francisco
Carl Goetsch	Berkeley
Mr. Thomas Hadfield	San Francisco
M. M. Haskell	Long Beach
Donald C. Harrington	Stockton
Gregory C. Murray	Los Angeles

THINK Towne when you think

GENERICS

MEPROBAMATE
Tablets
400 mg.
U.S.P.
Towne

One of hundreds
of TOWNE
QUALITY GENERICS.

In medicine and in pharmacy only the BEST is good enough. When you prescribe generically specify the manufacturer's name.

Play it safe ... Specify Towne

So your patient will consistently receive the highest quality and uniformity.

Product List available on request.

Towne PRESCRIPTION PRODUCTS DIVISION

Towne, Paulsen & Company, Inc.
Pharmaceutical Chemists Monrovia, California

The full ¼ grain of phenobarb in the formula
takes the nervous edge off the pain
...helps bring out the best in codeine

Phenaphen® with Codeine the only leading compound analgesic that calms instead of caffeinates

Each capsule contains:
Phenobarbital (¼ gr.) .... 16.2 mg.
(Warning: may be habit forming)
Aspirin (2½ gr.) ....162.0 mg.
Phenacetin (3 gr.) ....194.0 mg.
Hyoscyamine sulfate ....0.031 mg.
Codeine phosphate.............¼ gr. (No. 2),
½ gr. (No. 3), 1 gr. (No. 4)
(Warning: may be habit forming)

Contraindications: Hypersensitivity to any ingredient.

Precautions: As with all phenacetin-containing products, avoid excessive or prolonged use.

Side Effects: Side effects are uncommon—nausea, constipation, and drowsiness have been reported.

A·H·ROBINS

A. H. ROBINS CO., INC., Richmond, Va. 23220

NOW

Simplify dressing problems in those difficult areas!

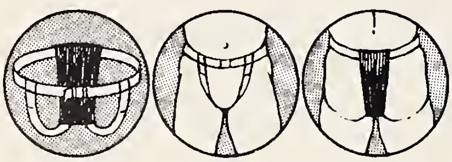
NOTE: These products have made possible the development of a flush-away sanitary napkin.*

*Patented by Mense, Inc.

Minnie Pants® PAT.

Minnie Pants is the new, more efficient method of holding dressings, sanitary napkins and tissues firmly in place in those difficult vulvar, rectal and coccyx areas, without adhesive tape, connectors or old-fashioned sanitary belts. Designed on the pressure bandage principle, Minnie Pants prevents shifting of dressings and subsequent chafing. Highly recommended for post natal patients and hemorrhoidectomies . . . and in the treatment of fistulas, cysts and urine seepage.

In cases where physicians and hospitals have used and tested Minnie Pants they have been appreciated as a more efficient method for nurses, and for patient comfort. Easy laundering!



***MENSE ANAL BELT:** The Mense Anal Belt holds any dressing firmly in place in those difficult vulvar, genital, rectal and coccyx areas without shifting. Designed on the pressure bandage principle, the Mense Anal Belt requires no adhesive tape, connectors, pins or tabs. Frees male genital organs or female vulvar area. Elastic panels completely adjustable for patient comfort.

The Mense Anal Belt is recommended for use in hemorrhoidectomies, decubitus ulcers in the coccyx area, fistulas, cysts and rectal surgery. In rectal cases with drainage the Mense Anal Belt holds dressings or even paper tissue firmly in place. Available at your surgical supply house or write . . .

*PAT.

MENSE, INC., DEPT. A
10723 FAIR OAKS BLVD., FAIR OAKS, CALIFORNIA 95628

Obesity Is Nation's Major Health Problem

Obesity has become a major health problem in the United States and a special health hazard for three obesity-prone groups, the Public Health Service reported recently.

Quoting a new PHS source book for health professionals, *Obesity and Health*, the Service said that the prevalence of obesity in this country is a source of growing medical concern because "Fat people are more likely to develop certain diseases and to die at an earlier age than people of normal weight."

Prime candidates for the development of obesity and its attendant association with certain serious disorders and possibly early death are:

1. Children whose relatives are obese: In one study, 73 per cent of 1,000 obese patients had at least one obese parent.
2. Heavily built persons who also have corpulent tendencies: Obese individuals usually have a heavier physique than their non-obese counterparts. Large-boned and thickly muscled persons, particularly adolescents, who fit this description should be watched closely.
3. Persons who are becoming less active, more sedentary: Food intake does not decrease proportionately with decrease in energy expenditure. As activity decreases, for whatever reason, the risk of developing obesity increases.

Persons who already have medical conditions that could be complicated by obesity must be especially wary of weight gain, the Service said. The onset of obesity can only complicate their disorder.

While no definite causal relationship between obesity and a variety of medical abnormalities has been found to date, the PHS pointed out that coronary heart disease and diabetes mellitus are two major diseases often associated with obesity.

It also warned that obesity compounds the disabling effect of such cripplers as arthritis and emphysema and introduces an extra hazard into pregnancy.

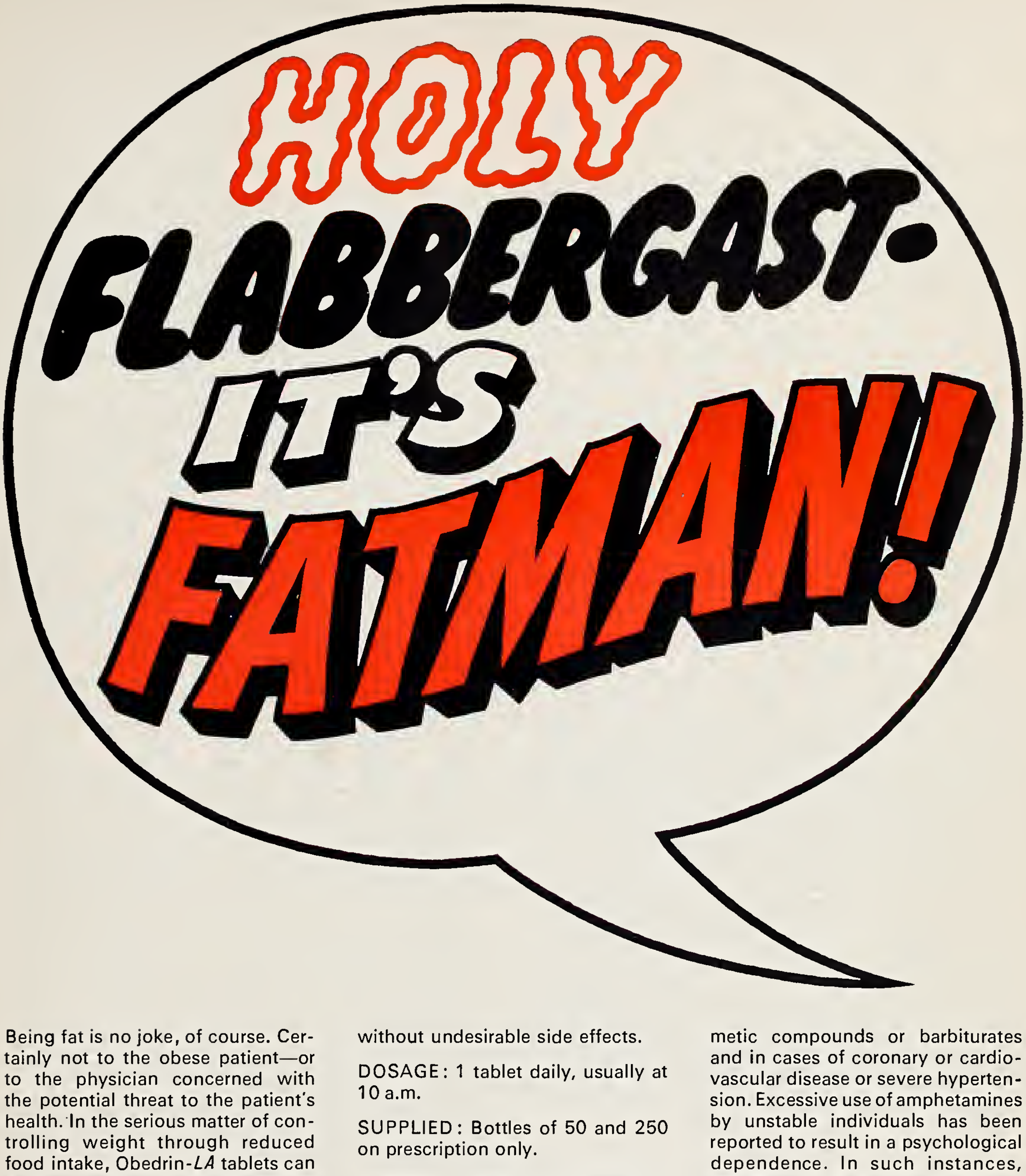
The Service said that while a substantial amount of obesity exists at every age in both sexes, obesity in children and adolescents is a particularly discouraging omen for the future.

It again quoted the source book *Obesity and Health:* "Obese children and adolescents are a major reservoir for obesity in adult life. They are more likely to remain obese as adults and to have more difficulty in losing fat and maintaining fat loss than people who become obese as adults."

Early diagnosis and the rapid initiation of preventive measures are essential if obesity, in all age groups, is to be avoided, the Public Health Service concluded.

Obesity and Health is a compendium of current information designed to assist the health profes-

(Continued on Page 36)



Being fat is no joke, of course. Certainly not to the obese patient—or to the physician concerned with the potential threat to the patient's health. In the serious matter of controlling weight through reduced food intake, Obedrin-*LA* tablets can be very helpful. Administered as directed, they trickle-release active medications in a balanced ratio to curb appetite, supplement restricted diet, sustain mood and allay anxiety without undesirable side effects.

DOSAGE: 1 tablet daily, usually at 10 a.m.

SUPPLIED: Bottles of 50 and 250 on prescription only.

CAUTION: Obedrin-*LA* should not be given concurrently with monoamine oxidase inhibitors. It should be used with caution in patients having a sensitivity to sympathomimetic compounds or barbiturates and in cases of coronary or cardiovascular disease or severe hypertension. Excessive use of amphetamines by unstable individuals has been reported to result in a psychological dependence. In such instances, withdrawal of the medication is necessary. All medication should be used with caution in pregnant patients, especially in the first trimester.

Obedrin®-LA*

"TRICKLE-RELEASE" TABLETS

Each tablet contains: Methamphetamine HCl*, 12.5 mg.; Pentobarbital*, 50 mg. (Barbituric Acid derivative; Warning: May be habit forming), Ascorbic Acid, 200 mg.; Thiamine Mononitrate, 1 mg.; Riboflavin, 2 mg.; Niacin, 10 mg.
*U.S. Pat. Nos. 2,736,682; 2,809,916; 2,809,917; 2,809,918 and pat. pend.

MASSENGILL The S. E. Massengill Company, Bristol, Tennessee • New York • San Francisco • Dallas • Chicago • Kansas City

OFFICERS

OF THE CALIFORNIA MEDICAL ASSOCIATION

(Continued from Page 5)

Committee on Health Facilities Planning

Laurence A. Mosier, Garden Grove	1967
John W. Gates, Visalia	1967
Joseph W. Telford, San Diego	1967
Jean F. Crum, Downey	1968
Glenn A. Pope, Sacramento (Chmn.)	1968
Richard Taw, Los Angeles	1968
James W. Dalton, Santa Barbara	1969
Paul C. Doehring, Glendale	1969
Pierre Salmon, San Mateo	1969

CONSULTANTS: Robert F. Schell, San Rafael; Jack Lomas, Beverly Hills.

COUNCIL COMMITTEES

Finance Committee

Harold Kay, Oakland (Chmn.); Roger C. Isenhour, San Diego; Frank C. Melone, Ontario; Joseph W. Telford, San Diego; Malcolm C. Todd, Long Beach.

Committee on Legislation

Dan O. Kilroy, Sacramento (Chmn.); Stuart C. Knox, Los Angeles; Harold E. Wilkins, Downey; John Rumsey, San Diego; Samuel R. Sherman, San Francisco.

Advisory Board to Woman's Auxiliary

James C. MacLaggan, San Diego (Chmn.); John G. Morrison, San Leandro; William F. Quinn, Los Angeles; Carl E. Anderson, Santa Rosa; Eugene Hoffman, Los Angeles.

Benevolence Fund Operating Committee

Clyde L. Boice, Palo Alto (Chmn.); Alexander Fraser, San Francisco; Elizabeth Mason Hohl, Los Angeles; Don C. Musser, San Francisco; George Wolf, Fresno; Dudley M. Cobb, Jr., Los Angeles.

HAVE YOU CHANGED YOUR ADDRESS RECENTLY?

To insure uninterrupted delivery of your copies of CALIFORNIA MEDICINE, please return this coupon properly filled out. Address CALIFORNIA MEDICINE, 693 Sutter Street, San Francisco, California 94102.

Name .... M.D
(PLEASE PRINT)

Former address:

Street..

City..

Zone..........State...

New address:

Street..

City..

State..............................Zip No.................

(Please use this coupon for address change only)

Duplicate copies cannot be sent to replace those undelivered through failure to notify this office of change of address.

Self-Mutilation by Patients

Physicians occasionally see the unusual and pitiful patient who mutilates himself.

This self-mutilation can take serious forms: In one variety, the patient believes he is infested with tiny parasites, and that the only way to get rid of them is to dig them out of his skin.

The result is a scarred and damaged skin that never heals and cannot be treated because the patient keeps re-inflicting the injuries.

Kenneth M. Halprin, M.D., a dermatologist at the University of Oregon Medical School, Portland, describes how he cares for these difficult patients in an article in a December 12 *Journal of the American Medical Association.*

The patients have the "unshakable conviction," Dr. Halprin said, that they are infested with some insect of microscopic size. They consult a physician, not to heal the wounds they inflict in searching for these insects, but to join the fight against the invading parasites.

"On the first visit, the patient invariably brings a small envelope which contains bits of dirt, skin, scales, and material fibers," Dr. Halprin said. "The patient will not be satisfied until these have been examined. Sometimes letters from laboratories may be offered as evidence that there actually is an organism present and responsible for all of the patient's difficulties.

"The delusion is so fixed and the patient so convincing that other people are led to believe that the patient is actually infested."

Helping these patients is difficult, Dr. Halprin admitted, but he suggests a "middle ground" approach.

If the physician openly declares that the patient is deluded, the result is "an angry exit with no return."

"Although it is essential not to agree with the patient's delusion, a middle ground—whereby immediate treatment of the patient's wounds is offered—should be the goal," Dr. Halprin said. This should be accompanied by making clear that no parasites are present and by "shifting the patient's attention to his inner turmoil and toward psychiatric help."

"These patients are deeply disturbed and avoid true psychosis by means of the delusion which allows them to keep some contact with reality in other areas. They therefore cling to their delusion as if their very life depended upon it—for indeed it often does."

The prospects for improvement are poor, Dr. Halprin conceded, since these patients lack the insight needed for successful psychiatric treatment.

The only successful treatment of these injured, disturbed persons, however, includes carefully steering them toward psychiatric help without destroying their elaborate mental defenses, Dr. Halprin said.

BOOKS RECEIVED

Books received by CALIFORNIA MEDICINE *are acknowledged in this column. Selections will be made for more extensive review in the interest of readers as space permits.*

ANATOMY OF THE HUMAN BODY—28th Edition—By Henry Gray, F.R.S., Late Fellow of the Royal College of Surgeons; Lecturer on Anatomy at St. George's Hospital Medical School, London. Edited by Charles Mayo Goss, A.B., M.D., Visiting Professor of Anatomy, George Washington University School of Medicine, Managing Editor of the Anatomical Record, Member of the International Anatomical Nomenclature Committee, Former President of the American Association of Anatomists. Lea & Febiger, Philadelphia, 1966. 1,448 pages, $22.50.

APPRAISAL OF CURRENT CONCEPTS IN ANESTHESIOLOGY—Volume 3—Edited and assembled by John Adriani, M.D., Professor of Surgery, Tulane University School of Medicine, New Orleans, La.; Clinical Professor of Surgery and Pharmacology, Louisiana State University School of Medicine, New Orleans, La.; Director, Department of Anesthesiology, Charity Hospital of Louisiana, New Orleans, La. 522 pages, $10.85.

ATLAS OF CRYOSURGICAL TECHNIQUES IN OPHTHALMOLOGY—By Charles D. Kelman, M.D., Associate Attending Surgeon and Director of Cryosurgery Research Laboratory, Manhattan Eye, Ear and Throat Hospital, New York, New York; Assistant Clinical Professor of Ophthalmology, New York Medical College, New York, New York; Consultant Ophthalmologist, Vassar Brothers Hospital, Poughkeepsie, New York. The C. V. Mosby Company, St. Louis, Mo., 1966. 118 pages, $18.75.

BASIC HUMAN ANATOMY—By Helen L. Dawson, Ph.D., Associate Professor of Anatomy and Histology, College of Medicine, University of Iowa. Appleton-Century-Crofts (Division of Meredith Publishing Company), New York, 1966, 332 pages, $8.95.

ENZYME KINETICS—A Learning Program for Students of the Biological and Medical Sciences—By Halvor N. Christensen, Ph.D., Professor and Chairman, Department of Biological Chemistry, The University of Michigan, Ann Arbor, Michigan; and Graham A. Palmer, Ph.D., Assistant Professor, Department of Biological Chemistry and Associate Research Biophysicist, The University of Michigan, Ann Arbor, Michigan. W. B. Saunders Company, Philadelphia and London, 1966. 124 pages, $3.50 (Paperback).

GASTRIN—Proceedings of a Conference held in September 1964; Sponsored by the School of Medicine, University of California, Los Angeles—Edited by Morton I. Grossman. UCLA Forum in Medical Sciences, Number 5. University of California Press, Berkeley and Los Angeles, 1966. 351 pages, $12.00.

HANDBOOK OF OCULAR THERAPEUTICS AND PHARMACOLOGY—Second Edition—By Philip P. Ellis, M.D., Associate Professor and Head, Division of Ophthalmology, Department of Surgery, University of Colorado Medical Center, Denver, Colorado; and Donn L. Smith, Ph.D., M.D., Professor, Department of Pharmacology, and Dean, University of Louisville School of Medicine, Louisville, Kentucky. The C. V. Mosby Company, St. Louis, Mo., 1966. 224 pages, $9.75.

HISTORY OF PSYCHOLOGY—An Overview—By Henryk Misiak, Fordham University and Virginia Staudt Sexton, Hunter College of the City University of New York. Grune & Stratton, Inc., New York and London, 1966. 499 pages, $12.50.

HUANG TI NEI CHING SU WEN—The Yellow Emperor's Classic of Internal Medicine—By Ilza Veith. University of California Press, Berkeley and Los Angeles, 1966. 260 pages, $8.00.

MANUAL OF TROPICAL MEDICINE—Fourth Edition—By George W. Hunter, III, Ph.D., Col. U.S.A. (Ret.), Lecturer, Microbiology and Biological Sciences, College of Medicine, University of Florida, Gainesville, Florida; formerly, Resident Coordinator of LSU-ICMRT and Research Professor of Medical Parasitology, Department of Tropical Medicine and Medical Parasitology, Louisiana State University, School of Medicine, New Orleans; William W. Frye, Ph.D., M.D., Sc.D. (Hon.), Professor of Tropical Medicine, Director of LSU International Research and Training Programs in Tropical Medicine; and J. Clyde Swartzwelder, Ph.D., Professor of Medical Parasitology and Head of Department of Tropical Medicine and Medica Parasitology, Co-Director, LSU International Research and Training Programs in Tropical Medicine, Louisiana State University, School of Medicine, New Orleans. W. B. Saunders Company, Philadelphia and London, 1966. 931 pages, $18.50.

OCULAR THERAPY—Complications and Management—From Symposia on Ocular Drug Complications and Management held under the auspices of the American Academy of Ophthalmology and Otolaryngology—Edited by Irving H. Leopold, M.D., Director, Department of Ophthalmology, The Mount Sinai Hospital, New York, New York; Chairman and Professor, Department of Ophthalmology, Mount Sinai School of Medicine, New York, New York. The C. V. Mosby Company, St. Louis, Mo., 1966. 157 pages, $11.00.

Your public relations problem has been our prime consideration in collection procedures during two generations of ethical service to the Medical Profession.

THE DOCTORS BUSINESS BUREAU

Since 1916

821 MARKET ST., SAN FRANCISCO 94103
GArfield 1-0460

For topical treatment of DENUDED and PAINFUL SKIN LESIONS

Anti-Pyrexol antiseptic ointment reduces pain, minimizes scarring, aids healing of burns, sunburn, scalds, lesions, wounds, and local inflammation of skin and mucous membrane. Sold through surgical supply houses. 1, 5, 10 and 50 lb. tins. Time tested—professionally since 1921. Active ingredients: Oils of spearmint, bay, wintergreen (syn.), salicylic acid, lanolin, zinc oxide, phenol .44%, ortho-hydroxyphenyl-mercuric chloride .056%, petrolatum, paraffin.

Anti-Pyrexol Benzocaine. Acutely anesthetic. Contains Benzocaine 3%. 1, 5 and 10 lb. tins.

HOSPITAL SPECIAL SIZE
ANTI-PYREXOL

EASY SPREADING
Anti-Pyrexol
KIP, INC.—LOS ANGELES 21

STACEY'S for any Medical Book in Print
Retail Stores in Palo Alto and San Francisco

NEW BOOK

STEDMAN'S MEDICAL DICTIONARY 21st ed. 1836 pages. Illustrated. 1966. Williams & Wilkins. $14.

Stacey's
326-9955
2575 HANOVER STREET
PALO ALTO, CALIFORNIA 94304

Please send me on approval:__________

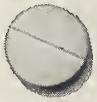
Name__________
Street__________
City__________State__________

starts fast...
keeps going...
relieves pain...

effective pain relief within 15 minutes...lasts 6 hours or more

PERCODAN relieves pain fast—usually within 15 minutes—and for prolonged periods, usually for 6 hours or more. Further, its speed and potency are *predictable* in the wide middle range of pain. PERCODAN is well tolerated and rarely causes constipation. **Usual Adult Dose:** 1 tablet every 6 hours. **Precautions:** The habit-forming potentialities of Percodan are somewhat less than those of morphine and somewhat greater than those of codeine. The usual precautions should be observed as with other opiate analgesics. Although generally well tolerated, Percodan may cause nausea, emesis or constipation in some patients. Percodan should be used with caution in patients with known idiosyncrasies to aspirin or phenacetin, and in those with blood dyscrasias. *Literature on request.*

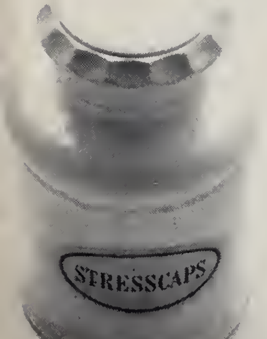
PERCODAN®



Each scored yellow Percodan Tablet contains 4.50 mg. oxycodone HCl (Warning: May be habit-forming), 0.38 mg. oxycodone terephthalate (Warning: May be habit-forming), 0.38 mg. homatropine terephthalate, 224 mg. aspirin, 160 mg. phenacetin, and 32 mg. caffeine. U. S. Pats. 2,628,185 and 2,907,768

ENDO LABORATORIES INC., Garden City, New York

Endo®



following infection

B and C vitamins are therapy: STRESSCAPS B and C vitamins in therapeutic amounts...help the body mobilize defenses during convalescence...aid response to primary therapy. The patient with a severe infection, and many others undergoing physiologic stress, may benefit from STRESSCAPS capsules.

Stresscaps®

Stress Formula Vitamins Lederle

FOR PROFESSIONAL RECOMMENDATION ONLY

Each capsule contains:

Vitamin B_1 (as Thiamine Mononitrate)	10 mg
Vitamin B_2 (Riboflavin)	10 mg
Vitamin B_6 (Pyridoxine HCl)	2 mg
Vitamin B_{12} Crystalline	4 mcgm
Vitamin C (Ascorbic Acid)	300 mg
Niacinamide	100 mg
Calcium Pantothenate	20 mg

Recommended intake: Adults, 1 capsule daily, for the treatment of vitamin deficiencies. Supplied in decorative "reminder" jars of 30 and 100; bottles of 500.

LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York

Lederle

626-6—3612

DIARRHEA
MUCOUS COLITIS
DIVERTICULITIS
SPASTIC URETERITIS
BLADDER SPASM

are relieved with.....

Trocinate®
BRAND THIPHENAMIL HCl

Minimum dosage 400 mg., q. 4 h. until relief is constant, adjust maintenance dosage.

A therapeutic blood level cannot be obtained with small dosage. Trocinate is metabolized and eliminated in the urine as harmless degradation products—a safety factor. Sixteen years of clinical usage with the absence of untoward effects establishes the safety of Trocinate. The autonomic nervous system is not involved in its prompt action.

NOW AVAILABLE IN 2 STRENGTHS,
100 mg. and 400 mg.
PINK SUGAR-COATED TABLETS

Literature and samples available.

WM. P. POYTHRESS & CO., INC.
RICHMOND, VIRGINIA 23217

Manufacturers of ethical pharmaceuticals since 1856

Obesity Is Nation's Major Health Problem

(Continued from Page 22)

sions with the obesity problems they encounter in their respective fields of activity. It was prepared by a panel of specialists and released today by the Heart Disease Control Branch, Division of Chronic Diseases. One of the eight co-authors is Reuel A. Stallones, M.D., Professor of Public Health, University of California (Berkeley) School of Public Health.

Single copies of *Obesity and Health* are available free from the Public Health Service, Department of Health, Education, and Welfare, Washington, D.C. 20201. The pamphlet sells for 60 cents a copy and is available from the Superintendent of Documents, Government Printing Office, Washington, D.C. 20402.

Arizona's Second Biennial Seminar on Prematurity Is To Be Held in February

The Seminar on Prematurity, sponsored by the Arizona State Department of Health and the State Advisory Committee on Prematurity, will be held February 25 and 26. Site of the meeting will be the Executive House Arizonian in Scottsdale.

Registration is open to all physicians and nurses interested in this problem.

Guest speakers have chosen important phases for subjects, as listed below:

SYDNEY GELLIS, M.D.—Professor of Pediatrics, Tufts University, Boston, Massachusetts.

1. "The Responsibilities of the Obstetrician and the Pediatrician to the Premature Infant."
2. "Clues to Congenital Defects in Premature Infants."

MARSHALL KLAUS, M.D.—Director of Clinical Center for Premature Infants, Stanford University, Palo Alto, California.

1. "Current Physiologic Therapy for Hyaline Membrane Disease."
2. "Touch and No-Touch Mothers in the Premature Nursery."

WILLIAM SILVERMAN, M.D.—Professor of Pediatrics, Babies Hospital, Columbia Presbyterian Medical Center, New York.

1. "Thermogenesis in the Neonate."
2. "Fetal Undergrowth."

REGISTRATION FEE: $7.50 (Includes Luncheon on Saturday). For further information, write to: Frederic W. Baum, M.D., Chief Material & Child Health Section, Room 402, Goodrich Building, 14 North Central Avenue, Phoenix, Arizona 85004.

(Advance Registration Encouraged.)

California
Medicine

Circulatory Shock

A Symposium on Advances in the Understanding of Mechanisms and Treatment

THE COMMON OCCURRENCE and the relative ineffectiveness of current modes of treatment of circulatory shock continue to be a serious problem for physicians in almost every medical or surgical specialty. In the past decade, there has been a resurgence of interest in both the mechanism of shock and its treatment, but now for the first time the emphasis is on extensive studies made at the bedside of patients in shock rather than in the experimental laboratory. This has led to the establishment of several clinical research centers for specialized study of patients in shock.

The first of these centers was established at the Los Angeles County General Hospital as a part of the University of Southern California School of Medicine. This stimulus has been in part responsible for increasing community-wide interest in shock and, in general, subjects related to the care of the critically ill.

An annual symposium on shock has been held in Los Angeles in which progress in the field of shock is reviewed by the staff of the Shock Research Unit and by prominent authorities who serve as visiting lecturers.

The fourth annual symposium, held on 9 February 1966, was attended by more than 350 physicians. The university faculty was honored by the participation of Dr. Wesley W. Spink, well known for his contributions in the field of endotoxin shock (and brucellosis) and by four outstanding surgical investigators, Dr. Sam Seeley, Dr. Lloyd MacLean, Colonel John Moncrief and Dr. David Hinshaw, in addition to members of the staff of the University of Southern California School of Medicine.

Progress in the Treatment of Shock: A Historical Perspective

SAM F. SEELEY, M.D.*

Acceptance of an invitation to discuss broad perspectives of a subject so vast as shock connotes a depth of experience and wisdom which no one person can expect to attain, but I will relate some of my observations and thoughts based on my experience as a military surgeon, some special training in surgical physiology and unusual opportunities to associate with experts in the halls of the National Academy of Sciences.

The four blood types were described in 1903, the year of my birth. About 30 years later, after I had become aware of many problems confronting military surgeons in World War I, I had the good fortune to be associated with Dr. Frank C. Mann at the Mayo Clinic and with the staff of the Army Medical School in Washington. During this time I reviewed the available literature on shock.

The symposium was held under the auspices of the Shock Research Unit and the Postgraduate Division of the University of Southern California School of Medicine, 9 February 1966. This report was compiled and edited by co-ordinator of the course, Max Harry Weil, M.D., Ph.D., director of the USC Shock Research Unit.

The work of the Shock Research Unit is supported by the John A. Hartford Foundation, Inc., New York and by Public Health Service Research Grants HE-05570 and HE-07811 from the National Heart Institute.

Submitted 11 August 1966.

Reprint requests to: Director, Shock Research Unit, USC School of Medicine, 2025 Zonal Avenue, Los Angeles 90033 (Dr. Weil).

*Brigadier General, MC, USA, Retired; Professional Associate, National Research Council, National Academy of Sciences.

We were particularly interested in experimental studies which had significance for the improvement in care of shock in the event of another world war.

In 1935, Phemister and Blalock confirmed that after burns or trauma to the extremities, the loss of fluid from the circulation was due to extravasalation with edema and exudation at the site of trauma or in relation to it. Blood transfusions were uncommon at that time. In most institutions, blood transfusions were administered by members of the surgical staff. The cause of shock and its clinical features were explained in terms of body systems rather than individual organs or more refined measurements of capillary transfer and cellular function.

My first perspective is divided into three 30-year eras. The first, beginning at the time that blood types were first described and ending with the publications of Phemister and Blalock, provided information which indicated the need for replacement of fluid, but little information was available on the manner in which fluid elements were distributed within body compartments. Very few transfusions had been administered during World War I. Acacia, one of the earliest blood substitutes, had been employed in thousands of cases but it was discarded at the end of this period because of its unfavorable effect on the liver.

The second 30-year period encompasses the advances achieved during World War II and in the Korean encounter. For the first time adequate amounts of blood became available at the battle front. The need for massive transfusion was recognized and both plasma and a number of plasma expanders were available. During this period, studies of effects of shock on individual organs and tissues were begun. A new measure of boldness was exhibited, in that observations on mechanisms were made directly on the injured man. Previously such observations were made only in animals and in the laboratory.

We are now in the third 30-year period, half of which has already passed. Highly sophisticated techniques are now brought to the bedside of the injured man. Refined instrumentation permits study of microcirculation, cellular metabolism, capillary permeability and the complicated function of even single cells, and of molecular activity within these cells. Direct measurements of pressures within the circulatory system and estimate of the deficits and by-products of cellular metabolism are now provided. A vast new field has been opened. The mechanisms of shock caused by neurogenic stimuli, trauma, hemorrhage, sepsis, anaphylaxis and other factors are likely to be explained in the near future.

There are three prerequisites for competent research in the field of shock. First is thorough familiarity with the literature of the past. Crile, Cannon, Guthrie and others seriously interested in shock, recorded basic observations which to many newcomers in the field were considered to have been discovered only recently (even in their own laboratories). Admittedly some theories have been disproved, but there should be a continuing effort to apply newer methods to improve current understanding of well-established concepts.

Secondly, we have been forced to the conclusion that most of the past studies in animals must be repeated under exacting, controlled conditions. Even then, extrapolation to man is precarious. For this reason laboratory tools are being refined and the basic scientist and the engineer are joining the clinical team.

Fulfillment of the third prerequisite is well under way. Dr. Weil and his associates have established one of the very few shock research units devoted primarily to observations in man. In this and other shock research centers, in laboratories of physiology, biochemistry, electronics, physics and other disciplines highly sophisticated studies are carried out on only one or two patients at a time. The work is done efficiently and findings are interpreted rapidly. Many lives are being saved even while the purposes of good clinical research are being achieved.

It is these multidisciplinary contributions, with development of improved methods, which will answer many of today's questions and ultimately open vistas which will provide essential information on critical functions of single cells.

Anaphylaxis and Endotoxin

WESLEY W. SPINK, M.D.*

Acute anaphylaxis is a manifestation of acquired hypersensitivity in which a generalized vascular response can predominate. The development of a concept that endotoxin shock may be anaphylactic in origin stemmed from two sets of clinical

---

Professor of Medicine, University of Minnesota Medical School, Minneapolis.

observations. First, a few hours after chronically ill patients with brucellosis had received a single dose of chlortetracycline, 50 per cent of the group developed shock. All of the patients had bacteremia due to *Brucella* melitensis. It was postulated that the shock was due to the liberation of somatic bacterial antigen (possibly endotoxin) by the antibiotic in patients with acquired brucella hypersensitivity. This manifestation of hypersensitivity could be prevented by the administration of adrenocorticosteroid. Subsequently it was shown that animals experimentally infected with brucella were more susceptible to endotoxin than normal controls and protection was afforded by steroids.

Second, some patients following catheterization of the urinary bladder exhibited the same manifestations of shock as the brucella patients, and bacteremia was usually associated with *Escherichia* coli. The prompt administration of corticosteroids, with and without pressor drugs, was beneficial.

Extensive studies in the laboratory on canine endotoxin shock fortified the concept that endotoxin shock was an expression of acquired bacterial hypersensitivity. The severity of the shock is dependent upon two factors: The dose of antigen (endotoxin) and the degree of hypersensitivity. Lethal shock was prevented by the pre-endotoxin administration of cortisol. Studies in the intact animal and with isolated strips of vessels showed that a heat-labile plasma factor (perhaps complement) was essential for endotoxin action. A decrease in complement was demonstrated in intact animals shortly after the onset of shock. The onset of hypotension was associated with a rise in circulating histamine and catecholamines, especially epinephrine. Studies by other investigators have implicated serotonin, bradykinin and proteolytic enzymes. Studies thus far do not negate the possibility of nonspecific toxic action of endotoxin on the tissues. The role of complement requires further elucidation. The nature of the bacterial antibodies needs to be defined. A more precise chemical identification of the toxic moiety of endotoxin is necessary.

Endotoxin shock is probably triggered by an immunologic mechanism but the subsequent hemodynamic dysfunction and metabolic disorder make it very difficult to distinguish between cause and effect as the shock progresses to an irreversible state. As a consequence, therapeutic agents and procedures are difficult to evaluate in a deteriorating host.

Guide Lines for the Selection of Fluids and Drugs for the Treatment of Patients in Shock

LLOYD D. MAC LEAN, M.D.*

Dissatisfaction with the treatment of shock based on clinical diagnosis alone prompted our group to establish methods of determining hemodynamic diagnosis to guide management of patients in shock. This required the measurement of arterial blood pressure, central venous pressure and cardiac output. In addition, measurements have been made of arterial blood pH, PO_2 and lactate.

On the basis of the first 30 patients studied, three hemodynamic categories emerged. In 15 of the patients, a cardiogenic cause was identified. These patients had a high central venous pressure, a low cardiac output, and improved after administration of isoproterenol in a dose of approximately 1 mg in 500 ml of dextrose 5 per cent in water. Isoproterenol was not given to patients whose heart rate exceeded 120.

The second most common hemodynamic abnormality was hypovolemia. These patients, who had low central venous pressure and low cardiac output, improved after blood volume was expanded, either with blood alone (if the hematocrit was less than 50 volume per cent), or by infusion of plasma or dextran (if the hematocrit was greater than 50 volume per cent).

In one patient peripheral pooling was the cause of shock. This patient presented with a low central venous pressure and a low cardiac output that did not respond to massive volume replacement. The central venous pressure was not increased by administration of large volumes of blood, indicating the blood was pooling in the periphery and was not being returned to the heart. The fact that this patient had a very high peripheral resistance indicated that this malignant form of pooling was distal to the resistance vessels, that is, in the capillaries and venules, the capacitance vessels.

Great reliance is being placed on the measurement of arterial blood lactate as an indication of the severity of shock and of the efficacy of treatment. Patients in whom the arterial blood lactate remains elevated have a very poor prognosis. The hemodynamic abnormality in 11 patients with septic shock was either of cardiac cause or due to deficiency of blood volume. The pattern characteristic of dogs—that is, peripheral pooling—was not seen in any of these patients.

---

*Surgeon-in-Chief, Royal Victoria Hospital, Montreal.

The success of treatment and the survival of patients in shock reflects the medical and surgical care with which the clinical diagnosis is established. We regard appropriate treatment as treatment selectively planned to reverse the hemodynamic abnormality. We infuse volumes of fluids in cases in which central venous pressure and cardiac output are reduced. If the patient is still in a state of shock after the central venous pressure has been increased to 10 cm of water, isoproterenol is used to increase the cardiac output without increasing cardiac filling pressure or precipitating heart failure. If the arterial blood lactate then remains the same or rises, there may be a place for the use of a hyperbaric chamber.

Hemorrhagic and Metabolic Defects Following Burn, with Implications For Treatment

JOHN A. MONCRIEF, M.D.*

Hemorrhagic episodes and metabolic derangements, although most frequent in the immediate postburn period, may occur at any time in the course of recovery from the injury.

The most common cause of blood loss as a complication of burn injury is massive bleeding from acute gastroduodenal ulceration—Curling's ulcer. Most often such ulcers occur after large burns and/or sepsis, but exsanguination may also occur after burns which involve as little as 10 per cent of the body or even less, and also in the absence of significant infection. Prodromal symptoms are minimal and usually there has been no pre-existing peptic ulcer diathesis. Conservative therapy is preferred for initial management but a 50 per cent gastric resection and vagotomy has been proved the only means of controlling massive hemorrhage in some patients.

Coffee-grounds hematemesis is very common in the first 24 to 36 hours after a burn. This may be closely related to but not necessarily due to local ulceration. Blood coagulation is rarely deranged except after generalized sepsis when petechiae and thrombocytopenia follow massive bacterial invasion of bone marrow. Anemia and granulocytopenia are also related to sepsis or to chronic infection.

The additional metabolic demands that result from the burn wound are dependent on size and depth of the wound and complicating disease or injury. Respiratory work is often increased two to three fold when acidosis or pneumonia complicates the course. The metabolic rate is consistently elevated to meet the requirements of wound healing in these massive injuries. Specific defects in biochemical pathways of metabolism are rarely observed.

When the vapor barrier is lost after skin burns, massive evaporative losses of water and heat greatly increase the caloric requirements. This defect persists until effective coverage of the skin is achieved. Increasing humidity decreases the heat loss but raising the room temperature is more desirable and equally effective.

Surgical manipulation and general anesthesia further reduce the patient's marginal caloric intake and compound the problem.

The Shock Research Unit at the Los Angeles County General Hospital

MAX HARRY WEIL, M.D., PH.D.*

The very complexity of shock as a clinical state attracted us to the concept of a broad-based, interdisciplinary approach to study of the phenomenon. After working for five years in the experimental laboratory, we were convinced that the information derived from the study of experimental animals could not be applied directly to patients. We therefore turned our efforts toward the development of clinical facilities for more critical examination of principles learned in the laboratory and for much more detailed study of mechanisms of shock in patients.

The shock ward at the hospital and a number of similar facilities since established at several other universities, provide a unique opportunity for detailed physiological and metabolic investigation of patients—investigations which of themselves contribute not only to academic study but in a very direct way to the improved clinical management of patients.

Since 1963 the addition of staff and machinery to operate a computer system to assist in the mechanics of acquisition of information and its translation into terms useful to clinicians has greatly advanced the scope of our clinical work.

The orderly diagnosis and treatment of patients, assisted by the availability of data acquired and computed by automated techniques, introduce a new level of efficiency in clinical management. This reflects itself particularly in the competence with which the immediate management of very critically ill patients is accomplished. We confirm that the clinical judgment inherent in the initial decisions of the physicians determines the immediate survival of the patient.

---

*Colonel, MC, US Army Surgical Research Unit, Brooke Army Medical Center, Fort Sam Houston, Texas.

*Associate Professor of Medicine, USC School of Medicine.

The computer may be looked on as a housekeeper; it is a controller of the automated techniques which assure the immediate availability of important physiological and biochemical information. The physician is provided with a highly legible numerical log by the typewriter. In addition, a record is automatically plotted for him. This record indicates the trends in the patient's course and, even more, the numerical log transmits the essential data which guide the subsequent management.

Most investigators are in agreement that the very basis of shock is failure of effective circulation of the blood. Our work of the last five years has not altered this concept. This failure of circulation is reflected in secondary metabolic changes. Most notable among these is the development of oxygen deficit, which in turn is reflected in lactate excess. This relationship between lactate excess and survival, reported about two years ago by our group[1] has been clarified by Dr. Marbach. He also has greatly simplified biochemical techniques for measurement of lactate and pyruvate, techniques of sufficient simplicity that they can be completed in a half hour in hospital laboratories. These provide an indication of the severity of damage and correlate rather well with the subsequent clinical course of the individual patient.

In the development of our program we have come to rediscover, or perhaps simply reemphasize, some general rules which help to guide the practicing physician. We recognize an obligation to the physicians who are not provided with the complexity of instrumentation nor the generosity of technical assistance available in a specialized facility such as our own. Quite apart from the facilities, however, is the recognition of critical limitations in the reserve of vital organs. The most critical reserve of any organ system is the blood volume and, more specifically, the plasma volume. A patient can easily manage with half the normal number of circulatory red cells, but a reduction of more than 30 per cent of plasma volume is life-threatening.

Volume repletion is managed in a remarkably practical fashion by the use of the central venous catheter. Dr. Zahn described an additional use of this central venous catheter. It can be a source of blood to determine the pH and PCO_2, obviating the need of repeated arterial punctures.

Doctors Hanashiro and Camarata documented the very practical implications of measuring pH and PCO_2 (or bicarbonate) routinely in critically ill patients. They also detailed the little appreciated limitations of positive pressure ventilators in wide use for maintenance of respiratory balance. Cardiac arrest is the ultimate penalty of anoxemia and respiratory acidosis.

One of the most exciting developments has been the recognition of the hematocrit gradient described by Dr. Doty [whose presentation is a later part of this symposium]. The difference between the hematocrit in the central venous blood and that in capillary blood from the fingertip provides an uncomplicated technique for determining the severity of shock. Determining the difference requires no special facilities.

Our understanding of the mechanism of shock after myocardial infarction is, in my opinion, still very primitive. From observations in autopsy proved cases, Dr. Shubin joined other workers in pointing to the important role of dysrhythmias rather than progressive heart failure in the fatal progression of shock.

Pulmonary embolization is the prototype of vascular obstruction as a cause of shock. An understanding of the relationship between the clinical features and hemodynamic defect, described by Dr. Adachi, is likely to be of increasing value as surgical methods of emergency treatment are more widely used.

Among the most satisfying areas of clinical research has been the study of the effects of various vasoactive drugs, both so-called vasoconstrictors and vasodilators, for the treatment of shock. The dilemma of vasopressor and vasodilator drugs is really not a paradox. We now recognize a spectrum of actions rather than simply a vasoconstrictor or vasodilator action.[4] We also recognize that betamimetic or their counterparts—alpha blocking or alphalytic drugs—are beneficial for improving the blood flow during the course of shock. Dr. Bradley, in addition to indicating the benefits of alphalytic phentolamine (Regitine®), explored the use of isoproterenol (Isuprel®) in the treatment of shock in patients.

As demonstrated by Mr. Whigham's report [which appears later], we investigated the role of corticosteroid in a very impressive model of hemorrhagic shock developed for the rat. Mr. Whigham and I observed striking improvement in survival. The action of the steroid under such well controlled conditions provides additional insight into the mechanism by which corticosteroids improve blood flow.

In introducing the staff to you, we point with pride to the wonderful spirit and the ease of communication that extends from physician to theoretical mathematician, from physiologist to programmer, and from biochemist to systems engineer—all part of the Shock Research Unit at the University of Southern California.

Metabolic Defects During Shock with Particular Reference to Lactic Acidosis

EDWARD P. MARBACH, PH.D.*

Since the availability and delivery of oxygen are critical in shock, the severity of oxygen deprivation is an immediate determinant of survival. Without an adequate supply of oxygen, electrons do not flow through the oxidative enzyme system, the failure causing a build-up of reduced nicotinamide adenine dinucleotide (NADH) and a depletion of oxidized nicotinamide adenine dinucleotide (NAD). Since NAD is necessary for pyruvate to enter the Krebs cycle, the overall effect of oxygen depletion is the increase in NADH and pyruvate. By mass action the increase of NADH and pyruvate causes the build-up of lactate by way of the lactic dehydrogenase reaction.

In an attempt to arrive at an index of reversibility of shock, an enzymatic technique for measuring lactate and pyruvate using small blood samples was recently developed.[2] This procedure for lactate and pyruvate is simple, rapid, accurate and feasible for routine laboratory use. The work of Broder and Weil, which relates excess lactate to survival, shows that a lactate excess from 0 to 2 mM per liter carries less than a 20 per cent likelihood of fatal outcome; 2 to 4 mM is a twilight zone, and when lactate excesses exceed 4 mM, 80 per cent of cases terminate fatally (Chart 1). Lactate excess is a helpful index. The problem before us is the further exploration of other metabolites or enzymes in the oxidative electron transfer system of intermediary metabolism which would reflect the deficit of oxygen delivery to vital tissues promptly.

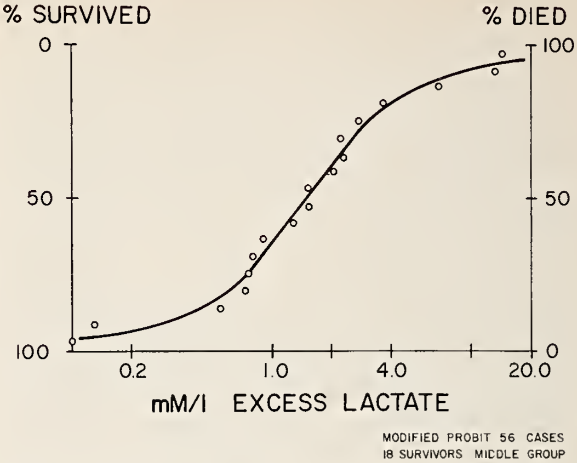


Chart 1.—Excess lactate related to the probability of survival of patients in shock. (Reproduced with permission of Broder and Weil, "Excess lactate: An index of reversibility of shock in human patients," Science, 143: 1457-1459, March 1964.)

Central Venous Blood for Monitoring pH and PCO_2 in the Critically Ill Patient

RICHARD L. ZAHN, M.D.*

The importance of knowledge of both blood pH and carbon dioxide content for competent evaluation and treatment of acid base disorders is now generally recognized. In critically ill patients, and especially patients in shock, the physical trauma of arterial puncture necessary to obtain reliable blood samples has discouraged wide application of pH and PCO_2 measurements on arterial blood.

We have previously recommended the routine employment of a central venous catheter in the management of critically ill patients. This catheter has the added advantage of providing a relatively simple means for obtaining reliable samples of blood comparable to arterial blood for repetitive analysis of pH and PCO_2.

A total of 101 paired arterial and central venous blood samples from 32 patients in the Shock Research Unit were analyzed. The pH of arterial blood ranged from 6.86 to 7.70 units (mean 7.45, and median 7.47). Venous pH ranged from 6.85 to 7.64 units (mean 7.439, and median 7.46). The correlation coefficient of arterial and venous measurements was 0.978 (Chart 2). Close correspondence between carbon dioxide tension in arterial and central venous blood was also observed. Arterial PCO_2 ranged from 22 to 100 mm of mercury (mean, 44.85, and median 44.0). In venous blood the PCO_2 ranged from 16 to 100 mm of mercury (mean, 47.0, and median 45.0). The correlation coefficient between paired measurements of PCO_2 on arterial and venous blood was 0.962.

---

*Research Associate, Dept. of Medicine and Assistant Biochemist, Los Angeles County General Hospital.

*Affiliating Resident in Surgery, Orange County Hospital.

We conclude that blood samples from a central vein provide a reliable indication of blood pH and carbon dioxide tension with values comparable with those obtained on arterial blood. Technical ease without undue medical risk of arterial puncture makes this an attractive technique for monitoring acid base changes in critically ill patients.

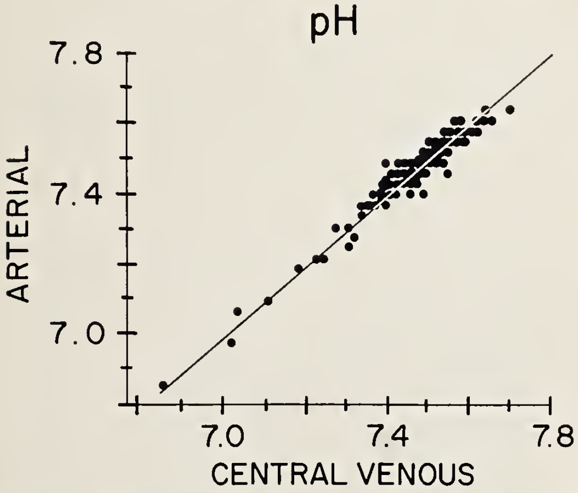


Chart 2.—Comparison of the central venous and arterial blood pH. (Reproduced with permission from Weil and Shubin, "Diagnoses and Treatment of Shock, The Williams and Wilkins Company, Baltimore, to be published, 1967.)

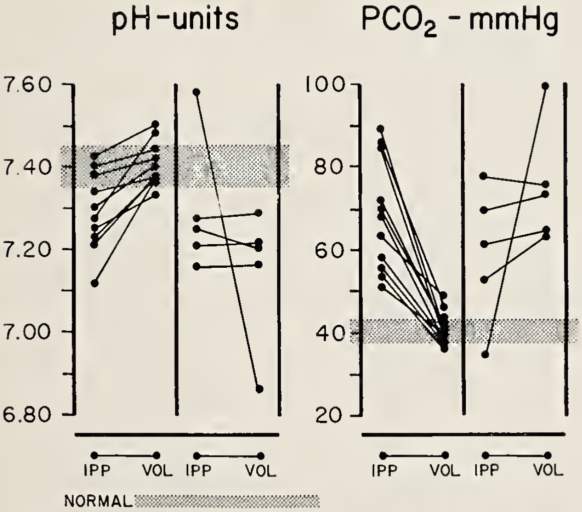


Chart 3.—Comparison of effects of an intermittent positive pressure ventilator (IPP) and a volume controlled ventilator (VOL) on pH and PCO_2. The first column in each group shows successful outcome and the second column failure to maintain adequate carbon dioxide elimination.

Ventilatory Failure as a Major Clinical Cause of Fatal Progression of Shock

PAUL K. HANASHIRO, M.D.*

Of the first 589 patients observed in the Shock Research Unit, 39 per cent required mechanical ventilatory assistance and of these 91 per cent died.

A detailed study was undertaken of the effects of ventilators with reference to their effects on arterial blood pH, PCO_2, PO_2 and arterial oxygen saturation. The volume-controlled ventilator, but not the pressure-controlled ventilator, was effective in improving gas exchange in 11 of 16 cases in which the intermittent positive pressure apparatus had failed. Only one patient became worse and in this instance spontaneous respiratory efforts, which had not been adequately suppressed, countered the action of the mechanical ventilator. In the remaining four patients, ventilatory status was unchanged (Chart 3).

The present study confirms the need for the wider availability of volume-controlled ventilators. Decrease in pulmonary compliance with increased resistance to airflow limits the output of the intermittent positive pressure ventilators. Volume controlled ventilators which overcome the greatly augmented resistance are necessary for management of such cases.

These observations also emphasize the usefulness of measuring pH and PCO_2 routinely for monitoring effectiveness of ventilation in critically ill patients.

A Study of 150 Instances of Cardiac Arrest with Implications for Treatment

SAMUEL J. CAMARATA, M.D.,† EDWARD C. BRADLEY, M.D.,‡ AND MAX HARRY WEIL, M.D., PH.D.

In seriously ill patients, particularly those with circulatory shock, respiratory and metabolic rather than cardiac abnormalities were the causes of cardiac arrest in the majority of cases.

In a review of 150 episodes in patients ranging from 25 to 86 years of age, the routine and repetitive measurements of pH, PCO_2, PO_2, O_2 saturation and bicarbonate before and after cardiac arrest indicated the special importance of acidosis as a predisposing condition. In 80 per cent of this group, pH ranged from 6.61 to 7.35 before arrest. In the majority, increases in PCO_2 and concurrent though lesser decreases in PO_2 indicated predominance of ventilatory defects.

---

*Instructor in Medicine, USC School of Medicine.

†Affiliating Resident in Surgery, Orange County Hospital.

‡Assistant Professor of Medicine, USC School of Medicine.

Cardiac arrest was more frequently related to drugs, asphyxia or myocardial causes in patients who did not have acidosis. The potentially detrimental action of aminophylline was apparent. In 14 cases, cardiac arrests occurred in immediate temporal relationship to intravenous administration of the drug to these critically ill patients (Chart 4).

Comparison of the Capillary and Central Hematocrit as an Indication Of Circulatory Shock

DONALD B. DOTY, M.D.,* AND
M. H. WEIL, M.D., PH.D.

Measurement of the microhematocrit on samples of blood obtained simultaneously from the fingertip and the central venous pool or an artery provides an indication of the extent to which peripheral blood flow is reduced and as such serves as an index of circulatory shock.

A study was designed to compare the microhematocrit determined on samples of blood obtained simultaneously from the arterial, central venous and capillary pools. These hematocrit values were correlated with measurement of arterial blood pressure, cardiac output, and circulation times. Serum lactic acid, pyruvic acid and urine output were determined to assess the efficiency of peripheral blood flow. The complete analytical procedure was accomplished on 92 occasions in 40 consecutive patients. The arterial hematocrit and the central venous hematocrit were essentially the same ($r=.99$). The capillary hematocrit, however, was as much as 10 volume per cent greater than the central venous hematocrit. The greatest differences were observed in patients with circulatory shock (Figure 1).

Comparison of the capillary and central venous hematocrit thus provided a gradient of hematocrit. A hematocrit gradient of 2 volume per cent or greater occurred during circulatory shock. Objective evidence of decreased peripheral blood flow was documented in these patients.

This bedside test is attractively simple, requires no special equipment or personnel and may be repeated with ease at frequent intervals. Its value compares favorably with measurements currently used to define the severity of shock and monitor the effects of treatment.

---

*Resident in Surgery, Los Angeles County General Hospital.

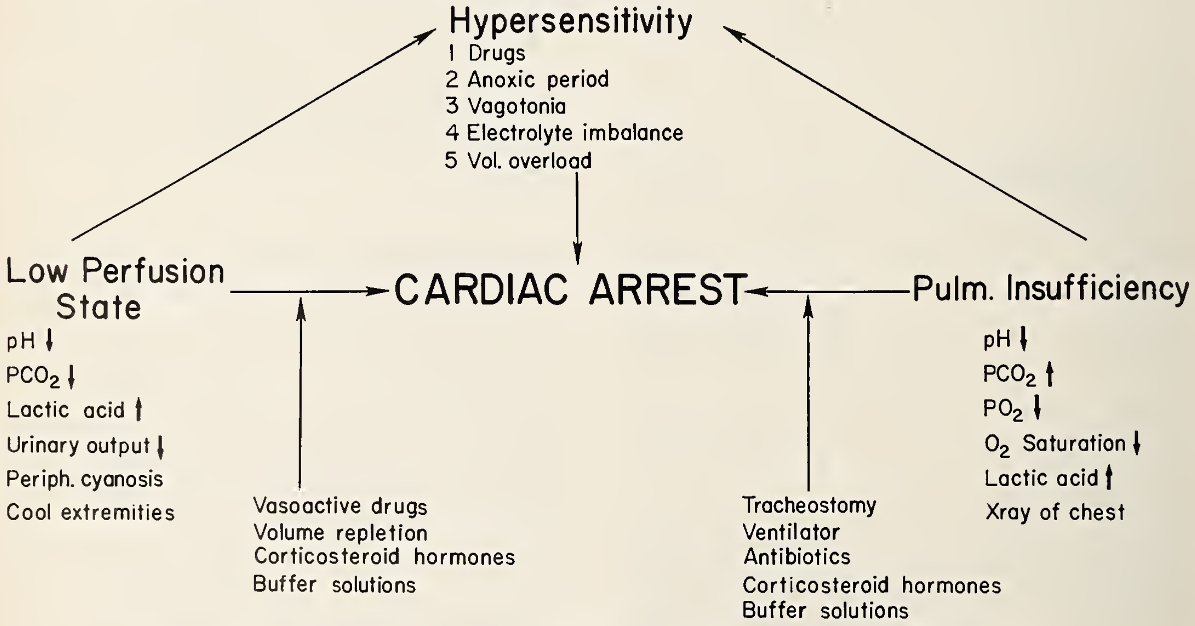

Hypersensitivity
1 Drugs
2 Anoxic period
3 Vagotonia
4 Electrolyte imbalance
5 Vol. overload
Low Perfusion State
CARDIAC ARREST
Pulm. Insufficiency
pH ↓
PCO2 ↓
Lactic acid ↑
Urinary output ↓
Periph. cyanosis
Cool extremities
Vasoactive drugs
Volume repletion
Corticosteroid hormones
Buffer solutions
Tracheostomy
Ventilator
Antibiotics
Corticosteroid hormones
Buffer solutions
pH ↓
PCO2 ↑
PO2 ↓
O2 Saturation ↓
Lactic acid ↑
Xray of chest

Chart 4.—The observed causes and present rationale for management of cardiac arrest.

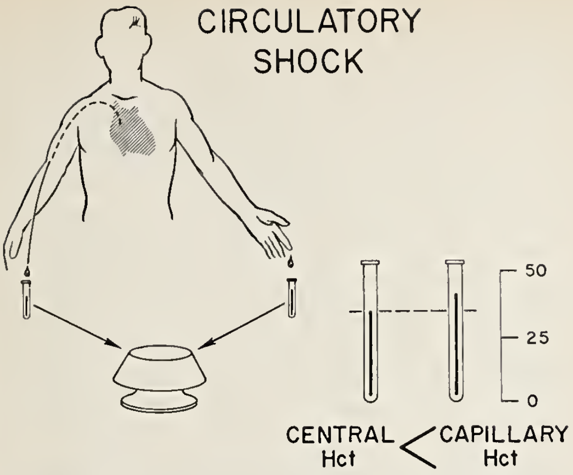


Figure 1.—Comparison of the capillary and central hematocrit serves as an index of circulatory shock.

Myocardial Infarction and Shock

HERBERT SHUBIN, M.D.*

The hemodynamic defect in patients who are in shock after acute myocardial infarction includes a reduction in arterial pressure and cardiac output and an increase in peripheral arterial resistance. In serial studies of 16 patients with acute and extensive myocardial infarction, the hemodynamic function improved after vasoactive drugs, such as metaraminol (Aramine®) and levarterenol (Levophed®), were administered during the initial period of 12 to 24 hours.

Twelve patients died. The typical couse of patients is illustrated in Chart 5. Following initial treatment with metaraminol or levarterenol the cardiac index and arterial pressure increased. Cardiac output was quite well maintained. Since the peripheral vascular resistance was normal or increased, so-called vasomotor collapse was not the cause of shock. Moreover, there was no evidence of congestive heart failure for central venous pressure was not elevated except as a terminal event.

The terminal course was primarily due to changes in cardiac rhythm. Within minutes before death, atrial tachycardia, flutter and fibrillation, sinus bradycardia, atrioventricular block and ventricular tachycardia appeared. These phenomena were followed by a rapid and precipitous decrease in arterial pressure and cardiac index and by increases in central venous pressure. Vasoactive agents were ineffective for hemodynamic functions at this stage. Death followed failure to control or to correct the progressive cardiac arrhythmia.

Hemodynamic Studies on Clinical Shock Following Pulmonary Embolism

ROY T. ADACHI, M.D.,* AND E. C. BRADLEY, M.D.†

Circulatory shock is a feature in approximately one-fourth of clinical cases of pulmonary embolism. Recognition of pulmonary embolism as the underlying cause of circulatory shock is often difficult, but with the wider use of surgical techniques for the treatment of this condition in emergency, prompt recognition has assumed even greater practical significance.

From studies recently completed in the Shock Research Unit, we now recognize that shock of this type does have characteristic features which are helpful in the clinical diagnosis. Five cases in which occlusion of major pulmonary artery was documented were studied. Mean arterial pressure was reduced, as was the cardiac index, except in one patient who was pregnant. The velocity of blood flow was reduced, as evidenced by the increased mean circulation time. The most consistent

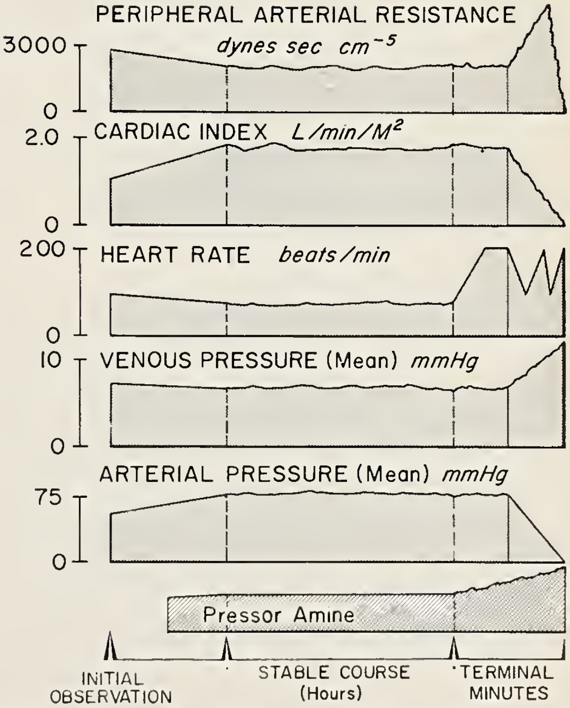


Chart 5.—Typical course of patients with myocardial infarction and shock.

---

*Assistant Professor of Medicine, USC School of Medicine.

*Affiliating Resident in Surgery, Orange County Hospital.
†Assistant Professor of Medicine, USC School of Medicine.

TABLE 1.—*Hemodynamic Effects of Isoproterenol*

	Control	*Test*	*Significance of Paired Differences*
Arterial Pressure			p
Systolic (mm Hg)	88	109	<0.02
Diastolic (mm Hg)	47	53	<0.2
Mean (mm Hg)	61	70	<0.1
Cardiac Index (L/min/M^2)	2.4	3.4	<0.001
Mean Circulation Time (sec)	20.5	15.5	<0.001
Resistance (dynes sec cm^{-5})	1,390	1,020	<0.001
Pulse Rate	97	109	<0.001

The values presented are the mean values for the group of 14 patients. Test values are the results obtained in 23 trials of isoproterenol infusion.

and we believe the most significant observation was an elevation in central venous pressure in the absence of congestive heart failure.

Hyperventilation was a prominent clinical sign, but the arterial oxygen saturation was not usually reduced. Respiratory alkalosis gave way to metabolic acidosis as the low perfusion state persisted. Lactic acid accumulation reflected the inadequacy of blood flow necessary to meet the oxygen requirements of actively metabolizing tissue. Pertinent measurements are summarized in Chart 6. The difference between values observed and those established as normal values were all significant at the 0.1 per cent level ($p<0.001$).

In summary, circulatory shock caused by pulmonary embolism should be suspected when the sudden onset of hypotension is associated with hyperventilation and elevation of central venous pressure.

Treatment of Circulatory Shock With Isoproterenol

EDWARD C. BRADLEY, M.D.,* AND
M. H. WEIL, M.D., PH.D.

Current practices, including the wide use of so-called "vasopressor" agents, for treatment of shock are being revised. We found that isoproterenol, an adrenergic drug with primary "vasodilator" activity, had beneficial hemodynamic actions. In dogs it consistently increased venous return and decreased arterial resistance. Its use for treatment of hemorrhagic shock in rats resulted in a highly significant increase in survival. We subsequently explored the hemodynamic actions of isoproterenol in patients. Tabulation of hemodynamic actions included 23 trials in 14 patients. Shock followed sepsis in 10, was due to cardiac cause in one, to neurologic cause in two and to pulmonary embolism in one. Isoproterenol was infused in average amounts of 3.8μg per minute for an average duration of 43 minutes. Cardiac index was augmented in each instance. The mean value increased from 2.4 L per minute per square meter of body surface to 3.4 L per minute per square meter of body surface ($p<0.001$). Arterial blood pressure was increased by an average of 9 mm of mercury. Heart rate increased from 97 to 109 beats per minute. The volume per beat increased from an average of 41 to 53 ml ($p<0.001$). Shortened dye circulation times and increase in the skin temperature of extremities indicated improvement in systemic and regional blood flows (Table 1). Present data confirm previous observations that isoproterenol increased cardiac output and systemic flow of blood when used for treatment in selected cases of circulatory shock.

Effect of Corticosteroids on the Progression of Hemorrhagic Shock in Rats

HOWARD WHIGHAM, B.S.*

Pharmacological amounts of corticosteroids effectively improve survival in experimental hemorrhagic shock in rats. With a refined and predictable technique for producing hemorrhagic shock in four

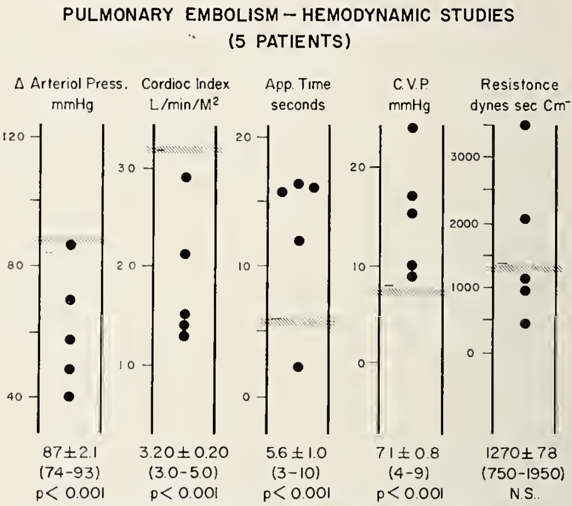


Chart 6.—Hemodynamic observations in five patients with massive pulmonary emboli. Normal values for this laboratory are indicated at the base of each column and the cross bars are the *mean* normal value for each hemodynamic measurement. (Reproduced with permission from Weil and Shubin, "Diagnoses and Treatment of Shock," The Williams and Wilkins Company, Baltimore, to be published, 1967.)

---

*Assistant Professor of Medicine, USC School of Medicine.

*Research Assistant, USC School of Medicine.

animals simultaneously, our laboratory was provided with a model by which animals were bled into a reservoir maintained at a pressure of 30 mm of mercury for 240 minutes. Two hours after reinfusion, mean intra-arterial pressure was increased to 77 mm of mercury but oxygen consumption was reduced to 40 per cent of normal. However pH (7.23 units), bicarbonate (14 mEq per liter) and hematocrit (50.9 volume per cent) were all essentially unchanged. Both aldosterone and glucocorticoid improved survival. The best results were obtained with the synthetic analogue, dexamethasone. After intra-arterial injection of 8 mg of dexamethasone per kilogram of body weight at completion of the bleeding period, oxygen uptake during the next two hours was increased to 104 ml per hour per kilogram of body weight (95 per cent of normal), blood pressure to 101 mm of mercury, pH to 7.31, bicarbonate to 19 mEq per liter and hematocrit to its control value. Blood lactate remained at levels about 4 mM in both control and treated animals. All control animals died within 72 hours but all corticosteroid treated animals survived. In patients in circulatory shock, dexamethasone increased cardiac output and decreased peripheral resistance.[3]

The present study indicates that glucocorticoid reverses hemoconcentration, increases oxygen uptake and reduces lactate excess and acidosis, which are indicative of tissue anoxia. Previously reported from several laboratories, including our own, was improvement in effective blood flow after corticosteroid therapy.

We now confirm on the basis of metabolic studies that the beneficial effect of dexamethasone is through an action by which oxidative metabolism is improved.

Application of Computer Techniques To Treatment of the Critically Ill Patient

MARSHALL A. ROCKWELL, JR., B.A.*

At the Shock Research Unit, techniques for use of digital computers in the treatment of the critically ill have been developed in two phases.

The first phase involves use of the computer to aid in gathering data simultaneously from two critically ill patients. An IBM 1710 computer system and the transducers which function in relation to it are programmed to perform these functions. The system has been in routine operation for over a year. It measures and reports 26 different variables, including temperature at six points, blood pressure at four points, urine output, heart rate, respiratory rate and other variables as often as every two minutes. Cardiac output and nine associated values are measured and reported at intervals ranging from one to 15 minutes.

Continuing effort is directed to improve organization and interpretation of the data so that the computer will supply critical information to assist in the decisions made by the physician at the bedside. In fact, the physician is to be provided with a simulator with which he can "test" and hence "predict" the effects of various forms of treatment, for the purpose of achieving optimal therapy without the delay of "trial and error" exploration on the very critically ill patients.

ACKNOWLEDGMENT: The staff of the Shock Research Unit gratefully acknowledges the devoted efforts of P. R. Manning, M.D., and the staff of the Postgraduate Medical Education Division; also the financial assistance of Winthrop Laboratories, Wyeth Laboratories, and The Upjohn Company and grants from Merck Sharp & Dohme and Smith Kline & French.

GENERIC AND TRADE NAMES OF DRUGS

Metaraminol—*Aramine.*
Levarterenol—*Levophed.*
Phentolamine—*Regitine.*
Isoproterenol—*Isuprel.*

REFERENCES

1. Broder, G., and Weil, M. H.: Excess lactate: An index of reversibility of shock in human patients, Science, 143:1457-1459, March 1964.

2. Marbach, E. P., Goodell, D. E., and Weil, M. H.: Rapid determination of lactate-pyruvate during shock, Fed. Proc., 24:341, March-April 1965.

3. Sambhi, M. P., Weil, M. H., and Udhoji, V. N.: Acute pharmacodynamic effects of glucocorticoids: Cardiac output and related hemodynamic changes in normal subjects and patients in shock, Circulation, 31:523-530, April 1965.

4. Weil, M. H., and Shubin, H.: Diagnosis and Treatment of Shock. With contributions by various authors and a foreword by Wesley W. Spink, Williams & Wilkins Company, Baltimore. To be published in 1967.

---

*Senior Programmer, USC School of Medicine.

The Role of External Irradiation In the Treatment of Transitional Cell Carcinoma of the Bladder

A Review of Fifty Cases

GEORGE JACK, M.D., AND FRANZ BUSCHKE, M.D., *San Francisco*

■ *Of 100 patients with carcinoma of the bladder seen in the Section of Therapeutic Radiology, University of California, San Francisco, between 1957 and 1962, 59 were accepted for radiation treatment. Fifty had transitional cell carcinoma and were treated with supervolt therapy (1 mev or cobalt-60).*

Two types of tumors were again found suitable for external irradiation: Papillary carcinomas Grades II and III, as long as they have not, or at least have not massively, invaded muscle; and undifferentiated carcinomas, Grade IV, regardless of degree of extension through the pelvis. The former type, if single, is treated by irradiation for the first recurrence after one attempt with radical transurethral resection. In the presence of multiple lesions at the first examination, radiation therapy is given immediately. The latter type is treated by radiation therapy without any attempt at surgical removal.

Of 37 patients, Stages A to C, treated more than three years ago, 14 (38 per cent) lived more than three years and eight (22 per cent) had no cystoscopic or clinical signs of active disease and had normal bladder function. Of 23 patients treated more than five years ago, eight were alive after five years (35 per cent) and four (17 per cent) remained controlled by radiation therapy alone, with normal bladder function.

No major complications were observed. In particular, no fibrosis of the bladder occurred. Doses ranged from 5,000 r in five and a half weeks to 6,000 r in seven weeks.

A close cooperation between urologic surgeons and radiotherapists during recent years permits long-range treatment planning from the time of diagnosis, which is essential in the effective therapy of carcinoma of the bladder.

NO ONE TECHNIQUE is best in the treatment of carcinoma of the bladder. Management of each case requires close collaboration between the urologic surgeon and the radiation therapist. Before any treatment is begun, joint consideration of all details of the individual case is essential. This implies cystoscopic examination by both urologist and radiation therapist, bimanual palpation in deep anesthesia and review of all available microscopic material, both past and present, by pathologist, urologist and radiation therapist.

Staging is based on the findings at cystoscopy and palpation, and on the presence or absence of muscular invasion (and the degree of invasion) in the microscopic material. Most pathologists are aware of the importance of determination of histologic grade and of tumor invasion as seen in the microscopic material. Pathologic interpretation, however, can only be as complete and reliable as the sampling of the submitted material permits. Even if the invasion of muscle is clinically suspected or obvious, it may be impossible to substantiate the suspicion in the microscopic material. The limitations of grading of microscopic differentiation and of staging must be recognized. A small sample may not be representative of the entire disease. Different portions of the same tumor or different tumors in the same bladder may show different degrees of differentiation. Where there are such differences of degree, the more undifferentiated portion determines the prognosis, but the more differentiated portion may determine the response to radiation therapy. Likewise, the staging—meaning the extent of the invasion of the bladder wall or beyond the bladder wall, or both—can, of course, not be determined as accurately by cystoscopic, microscopic and palpatory examination as it can be by examination of the gross surgical specimen. Nevertheless, rather accurate estimates are possible for experienced observers. Both grading and staging are essential for the planning of treatment as well as for interpretation of results.

When radiation therapy is planned, attention must be given to the presence or absence, and to the degree of, bladder infection, particularly in those areas not involved by tumor, for infection may critically influence both the radiation response and the tolerance of treatment. In the presence of infection, careful treatment by the appropriate antibiotics is essential before radiation therapy is begun. Treatment is not tolerated in the presence of obstruction that interferes with adequate drainage. In such instances, resection of an obstructive portion of the prostate or an obstructive portion of the tumor must precede radiation therapy. This is necessary even though the urologist may protest that such procedure in the presence of viable neoplasm in the bladder may lead to implantation of malignant cells. Without adequate drainage, radical treatment is not tolerated. The patient's general condition must be good enough to support this radical form of therapy. The strain on the patient's economy is considerable—frequently as great and sometimes greater than that of surgical intervention.

During treatment, attention to symptoms and proper administration of antispasmodics and analgesics is necessary. Excessive frequency of urination and discomfort may lead to loss of sleep and deterioration of the general condition to a degree that would force discontinuance of therapy.

The experience of the last 25 years[1] has shown rather conclusively that two types of transitional cell carcinoma of the bladder are suitable for external irradiation:

1. The moderately, undifferentiated tumors, Grades II and III, as long as they have not deeply invaded the muscle.

2. The very undifferentiated anaplastic tumors, Grade IV; these may still be controlled even in the presence of very far advanced disease. They are not suitable for surgical treatment because of their biological character, which leads to massive invasion of the pelvic lymphatic structures.

Technique

Frequently too much emphasis is placed on details of treatment technique. The technique must of course be adjusted to the particular quality of radiation used and to the peculiarities of the individual disease. That medium volt therapy is not suitable for carcinoma of the bladder has long been recognized. It necessitates the use of multiple fields which, together with lateral scattering, leads to unavoidable "hot spots." Radiation therapy of carcinoma of the bladder, therefore, has progressed only since supervoltage has become available. In transitional cell carcinomas, Grades II and III, the

---

From the Section of Therapeutic Radiology, Department of Radiology, University of California School of Medicine, San Francisco.

Partially supported by a grant (T1 CA 5177) from the National Institutes of Health.

Dr. Jack is supported as a Trainee (Radiation Therapy) by National Cancer Institutes Grant T1 CA 5177.

Presented before the Section on Radiology at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: Department of Radiology, U.C. Medical Center, San Francisco 94122 (Dr. Jack).

TABLE 1.—*Modification of Marshall's Classification* of Stages of Carcinoma of the Bladder: M.D. Anderson Hospital*[2]

Superficial:	Stage O	—Limitation of tumor to *mucosa.*
	Stage A	—Extension into *lamina propria.*
	Stage B_1	—Extension into *superficial muscle.*
Deep:	Stage B_2	—Extension into *deep muscle.*
	Stage C	—Extension into fat, *serosa,* or perivesicle space.
Metastatic or Fixed:	Stage D_1	—Involvement of prostate, palpable extension to pelvic wall, or to anterior abdominal wall.
	Stage D_2	—Extrapelvic spread.

*Bullous edema and hydronephrosis not staged.

risk of lymph node involvement is not great as long as the tumor has not massively invaded the bladder muscle and thus reached the circumferential lymphatic channels in the bladder wall. When this has occurred, control of the lesion in its primary site is not likely. Therefore, for this type of carcinoma inclusion of peripheral pelvic nodes does not appear to be the most important problem. The three-field techniques (suprapubic and posterior oblique), rotational therapy (which attempts to limit the volume treated with the cancerocidal dose to the bladder and its immediate neighborhood), and techniques covering the entire pelvis are equally effective. The importance of rotational therapy for the purpose of protecting the rectum should not be overemphasized. The anterior rectal wall in men is so closely related to the bladder that it cannot be protected without the risk of insufficient coverage of the tumor. With the techniques mentioned, however, we have never seen rectal reactions of clinical significance. (One of the authors has seen severe rectal damage as well as fibrosis of bladder in patients treated during the early days of supervolt therapy with doses now recognized as excessive.) In the very undifferentiated Grade IV carcinomas, it is preferable to cover homogeneously the entire pelvis because of the lymphatic involvement, which, owing to the great radiosensitivity of this tumor, may still be controllable.

The bladder dose should not exceed 6,000 r, or at the very most, 6,500 r with protraction of approximately 900 r per week. In this way, it is possible to avoid radiation fibrosis of the bladder consistently. More and more it is being recognized that increasing the dose beyond this level, or more rapid treatment, does not increase the likelihood of control of the disease, but does significantly increase the incidence of fibrotic changes in the bladder and other pelvic structures to a degree which brings us to the point of diminishing return. In the experience of one of the authors over a period of more than 25 years,[1] all those patients who remained controlled had received doses not exceeding 5,000 or 5,500 r. There had been in his experience since 1939 no cases of fibrosis of the bladder following such doses.

Results

From 1957 through 1962, 100 patients with carcinoma of the bladder were seen in consultation in the Section of Therapeutic Radiology, University of California San Francisco Medical Center. Of these, 59 were accepted for treatment. The main reasons for rejection were (a) previous irradiation to the pelvic area; (b) debilitated condition; (c) negative cystoscopic examination; or (d) recommendation of surgical treatment. One important reason for the high rejection rate (40 per cent) is our close cooperation with the urologists: We are called to be present at every cystoscopic examination when tumor is suspected. Many of these patients, obviously, are not candidates for irradiation. Of the 59 accepted for treatment, nine were excluded from this series, four with squamous cell carcinomas, one with adenocarcinoma and three with multiple papillomas. The ninth patient had transurethral resection one month before radiotherapeutic consultation without demonstrable tu-

TABLE 2.—*Results of Supervoltage Irradiation of Transitional Cell Carcinoma of Bladder (1957-1962)*

				Survival		*Controlled**	
Stage	*No.*	*Course Complete*	*Course Incomplete*	*3 yrs.*	*5 yrs.*	*3 yrs.*	*5 yrs.*
Superficial	24	19	5	12/24	7/16	6/24	3/16
A B_1	....	....	....	50%	43.8%	25%	18.8%
Deep	13	11	2	2/13	1/7	2/13	1/7
B_2 C	....	....	....	15.4%	14.2%	15.4%	14.2%
Metastatic	13	9	4	0/13	0/6	0/13	0/6
D_1 D_2	....	....	....	0.0%	0.0%	0.0%	0.0%
Totals	50	39	11	14/50	8/29	8/50	4/29
				28%	27.6%	16%	13.8%

*"Controlled" means that the patient had no recurrence following irradiation and lives free of demonstrable disease with normal bladder function.

mor at the time of our cystoscopic examination, and was probably cured by this procedure. This patient has remained free of symptoms for more than five years.

More than half of the 50 patients with transitional cell carcinomas who were treated had had several transurethral resections.

The staging of these cases is retrospective after review of all available records, roentgenograms and microscopic material. The staging (Tables 2 and 4) must therefore be interpreted with reservation. In case of doubt, the particular case was classified in the less advanced stage. Marshall's staging as modified at the M.D. Anderson Hospital was used[2] (Table 1).

Of the 39 patients given complete courses (Table 2), tumor doses ranged from 5,000 r in five and a half weeks to 6,000 r in seven weeks, and were delivered by one of three techniques: A central suprapubic field complemented by two posterior oblique fields; larger opposed pelvic fields for advanced disease; and a few arc rotations with cobalt-60 beginning in 1962. In the earlier years, 1,000 kv was used, but since 1960, cobalt-60 teletherapy had been used exclusively.

Seven patients, Stages A to C, did not tolerate treatment (Table 2). All had had multiple transurethral resections. Of these seven, however, three survived five years, but only one had been free of tumor at any time during that period (Table 5). The tumors in the three survivors had been classified as Stage A, Grade II, and the contributions from radiation therapy were minimal. Four patients with Stage D tumors were treated for palliation (Table 2) and received incomplete courses of approximately 2,500 r to the midpelvis in three to four weeks.

Of the 37 patients with tumors in Stages A to C (Table 3), in whom control could possibly be expected, 23 are available for five-year analysis. Of these, eight (35 per cent) survived five years, and four (17 per cent) remained controlled by radiation therapy alone. "Controlled" means that the patient lives free of demonstrable disease by cystoscopic and clinical examination and with normal bladder function. Radiotherapy in three of the four uncontrolled survivors was incomplete (as mentioned previously) and the fourth patient required a surgical procedure for persistent tumor seven months after irradiation (Table 5). Of the 37 patients available for three-year analysis (Table 3) 14 (38 per cent) survived and eight (22 per cent) remain controlled by radiotherapy. Some patients with histologically proved invasion of both superficial and deep muscle are controlled at three-year and five-year intervals (Tables 2 and 5).

TABLE 3.—*Transitional Cell Carcinoma of Bladder, Stages A to C Results of Supervoltage Therapy 1957-1962*

	3 Years	*5 Years*
Survival	14/37 37.8%	8/23 34.9%
Controlled*	8/37 21.6%	4/23 17.4%

*"Controlled" means that the patient had no recurrence following irradiation and lives free of demonstrable disease with normal bladder function.

Patients with Stage D disease were accepted for palliation. Generally, therapy was tolerated poorly and treatment was given more commonly for pain secondary to tumor. The longest period of palliation in a Stage D_1 tumor was 10 months, the patient having received more than 5,000 r to the midpelvis in eight weeks. Attempts to "keep the ureters open" or significantly reduce hydronephrosis were unsuccessful. Hematuria was not a prominent indication for palliation, as the dose necessary for any lasting effect could be neither tolerated nor delivered in a reasonable enough time. In a few patients of this advanced group, prostatic invasion was evident only on histologic examination, but still the tumors were not controlled by therapeutic doses. When Stages A to D are considered (Table 2), the per cent of salvage at three-year and five-year intervals falls in proportion to the number of advanced cases added.

No major complications occurred and there were only a few instances of mild proctitis and increased dysuria, which usually subsided before treatment ended. Patients with significant bladder symptoms before treatment responded well to antibiotic and antispasmodic medications.

Those of the 23 patients who were not cured by complete courses usually died within 12 months after treatment. Most of the patients were uremic at

TABLE 4.—*Stage/Grade Relationships in Carcinoma of Bladder 1957-1962 Supervoltage Therapy*

	Superficial			*Deep**		*Metastatic*†	
	O	*A*	*B_1*	*B_2*	*C*	*D_1*	*D_2*
Grade I		..	..	..	..	..	..
Grade II		10	3	1	1	2	..
Grade III		2	8	4	2	4	4
Grade IV		..	1	4	1	3	..

*Stage C—Biopsy evidence of penetration of wall in 1 patient only.

†D_1 cases represent extensive pelvic disease. In the D_2 stage, 3 of 4 had linguinal nodes, another a nodule in the abdominal wall (excised).

TABLE 5.—*Three and Five Year Survivors, Transitional Cell Carcinoma of Bladder, Supervoltage Therapy*

Age	*Sex*	*Previous Surgical Procedure**	*Stage*	*Grade*	*Treatment*	*Results*
60	M	TUR (1)	A	II	1,000 kv, opposed pelvic fields. Incomplete, 2,200 r in 3 weeks.	Positive TUR 4 months later. Same area. Living and well more than 5 years.
72	M	TUR (2)	A	II	1,000 kv, 3 fields. Incomplete. 3,500 r in 8 weeks.	Multiple positive TURs over next 7 years.
68	M	TUR (2)	A	II	1,000 kv, 3 fields. 5,100 r in 5 weeks.	Mild cystitis. No tumor 7 years.
74	M	TUR (2)	A	III	1,000 kv, 3 fields. 4,600 r in 7 weeks.	Positive TUR 7 months later. Same area. Living and well 6 years.
73	M	TUR (2)	A	III	1,000 kv, 3 fields. 5,500 r in 8 weeks.	Living and well 6 years.
72	M	TUR (1)	A	II	1,000 kv. 3 fields. Incomplete. 1,750 r in 2½ weeks.	Stroke 4 years later. No bladder symptoms in 6 years.
60	M	TUR (1)	B_2	III	1,000 kv, 3 fields. 5,300 r in 7 weeks.	Living and well 5 years.
50	M	TUR (2)	B_1	III	Cobalt 60, 3 fields. 5,400 r in 8 weeks.	Living and well 5 years.
68	M	TUR (1)	B_2	IV	Cobalt 60, 3 fields. 5,100 r in 5½ weeks.	Living and well 4 years.
70	M	TUR (2)	A	II	Cobalt 60, 3 fields. 5,100 r in 5 weeks.	Multiple positive TURs same area next 2 years. No symptoms following 2 years.
72	F	TUR (1)	B_1	IV	Cobalt 60, 140° arc. 5,500 r in 7 weeks.	Positive TUR same area 5 months. Living and well 4 years.
74	M	TUR (3)	A	II	Cobalt 60, 180° arc. 5,100 r in 7½ weeks.	Living and well 3½ years.
63	M	TUR (1)	B_1	III	Cobalt 60, 180° arc. 5,600 r in 7 weeks.	Positive TUR same area 1½ years later. Living and well following 2 years.
69	M	TUR (1)	B_1	III	Cobalt 60, opposed pelvic fields. 6,100 r in 8 weeks.	Positive tumor in 6 months. Cystectomy. Living and well 3 years.

*TUR—Transurethral resection (number of operations shown in parentheses).

the time of death. Although necropsy was carried out in only a few cases, death was most commonly attributed to pyelonephritis and uremia. Extrapelvic metastasis was suspected in approximately one-third of this group, but proved in only five patients. Five patients died of other causes within 15 months after treatment. These included two with coronary occlusion and recurrent bladder symptoms, another after cystectomy for recurrent tumor treated one year previously, and two who killed themselves after symptoms and proved disease had recurred.

During recent years a very close relationship has developed between the Section of Therapeutic Radiology and the Division of Urology. The radiotherapists are notified every time a cystoscopic examination is scheduled and the presence of tumor is suspected. The radiotherapist has an opportunity to inspect the bladder, perform palpation in deep anesthesia, review the microscopic material and then discuss the situation with the urologist. In this way, it has been possible to determine the stage of the disease more accurately and to improve long-term planning for the individual patient.

At the urologists' initiative, it is now our policy in Grades II and III carcinoma to accept a patient for radiation therapy after one failure of transurethral resection. Radiation therapy is no longer approached as the last resort. In the presence of multiple lesions of this grade, radiation therapy is begun immediately. The very undifferentiated carcinomas (Grade IV) are treated by irradiation exclusively without consideration being given to surgical procedure other than biopsy.

After radiation therapy, regular cytologic studies are done. Cystoscopic examination is repeated at three- to four-month intervals during the first year. When positive signs of tumor are present more than six months after irradiation, consideration is given to cystectomy.

REFERENCES

1. Buschke, F., and Jack, G.: Twenty-five years' experience with supervoltage therapy in the treatment of transitional cell carcinoma of the bladder, Am. J. Radiol., to be published.

2. Miller, L. S., Crigler, C. M., and Guinn, G. A.: Supervoltage irradiation for carcinoma of the urinary bladder, Radiology, 82:778-785, 1964.

A Family Planning Clinic In a County Hospital

JOHN N. MILLER, JR., M.D., *Sacramento*

■ *In Sacramento County a family planning clinic was organized and its services made available, within defined limits, to women eligible for county hospital service. In the first year of operation, one morning a week, the clinic served 542 women, examining them, giving advice and helping in the selection of means of contraception. Three hundred and fifteen patients selected oral contraceptives and 211 chose to use an intrauterine device. The problems with the intrauterine device were minimal. At one point 32 per cent of the people taking oral contraceptives were failing to return for refilling their prescription. Experience with the operation of the clinic showed shortcomings in the plan that was being used and pointed the way to improvements.*

THE INCREASING public focus on the population explosion has provided the necessary encouragement and impulse to provide family planning services in facilities that are paid for in part or whole by various branches of government. In Sacramento County the Board of Supervisors chose the County Hospital to provide such a service and the hospital administrator, through the director of professional services, selected five physicians in the private practice of medicine to advise, to make policy and to supervise a clinic for that purpose. These physicians represented Obstetrics and Gynecology, General Practice and the Roman Catholic faith. The initial directions of the Board of Supervisors were that the services would be provided only to married women or those about to be married, with the exception that the services could also be made available to others if two of the physicians agreed there was medical need. In addition, the patients needed to be eligible for county hospital service as determined by the Social Service Department. The orientation, education and instruction would be provided by Public Health Department nurses. Nevertheless, public health nurses and social workers in the Department of Social Welfare were instructed not to initiate discussion regarding family planning facilities.

After several meetings, the Medical Advisory Board for the Family Planning Clinic, composed of the five physicians and the Director of Medical Services, established medical objectives, policies and standards for the Family Planning Clinic: The clinic would provide information and clinical services for eligible patients, compatible with good medical practice and with their beliefs and needs, equal to services available in private practice. Medical care in the clinic would provide maximum assistance in reducing unwanted sterility and unwanted pregnancy. Consultation would be provided to determine medical need for family planning to patients not meeting social service criteria for referral to the clinic.

The clinic met one day a week at 8:00 a.m. At

Presented before the Section on Obstetrics and Gynecology at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: 5301 F Street, Sacramento 95819.

that time an orientation class preceded the provision of service by the staff of the clinic. The class was conducted by a public health nurse or other qualified person designated by the Director of Medical Services, and it covered the various methods of contraception, including rhythm. Instruction on the rhythm system preceded description of the other methods so that patients who did choose not to be informed of them could leave. Each patient was encouraged to form an opinion as to which type of contraception would be most acceptable to her.

Following the orientation, the patients were referred to the service area of the clinic where they discussed their choice of contraceptive with a physician who respected their wishes if they were compatible with good medical practice, but in no case did he recommend a method not of the patient's choice if it was contrary to their religious beliefs. Then, after history-taking, an examination of the breasts, abdomen and pelves and preparation of a smear of cervical exudate for cytologic examination, the patient was either further instructed in rhythm or other method she had chosen.

Following this procedure the patient was referred back to the public health nurse for individual instruction in the method chosen. In addition, written instructions for each of the methods were prepared by the Advisory Board, and each patient was given the sheet dealing with the method she had selected. Follow-up appointments appropriate to the method were made. Each patient was also given calendar cards to provide a record of menses, spotting and, in the case of tablets, the days on which they were taken.

Birth control counseling and the prescription and dispensing of devices are the responsibility of the Family Planning Clinic, and while other clinics in the area may point out the advisability of seeking Family Planning service, none will provide service to patients.

The clinic was opened 16 July 1964, staffed by a paid county physician and a voluntary consultant, who in most cases was a member of the Medical Advisory Board. At the outset the clinic was scheduled to have 10 new patients at each weekly session, and also follow-up patients, the number depending on a previously determined schedule relating to the particular method involved. Soon the schedule was increased to call for 15 new patients at each meeting, but rarely could the staff deal with that many. The waiting list grew and soon there was a wait of two months before an interested woman could be scheduled for her first session. One unfortunate result was that as the list grew longer the number of failed appointments increased.

In the first full year of operation, 542 new patients were seen. The majority chose either oral tablets or an intrauterine device. There were always a number of patients using foam or oral contraceptive until the time came for insertion of the device. All intrauterine devices were inserted within seven weeks postpartum or in the five to seven days following the onset of a menstrual period. For obvious reasons both from the standpoint of ease of insertion and the presence of bleeding these times were used, based on protocol.

All techniques are used as referred to by Tietze[4] as "cafeteria system." All the materials dispensed to patients were purchased by the county hospital with the exception of intrauterine devices, which were provided by the State Department of Health, Bureau of Maternal and Child Health, under a cooperative study with the Population Council.

The number of patients who decided upon oral contraceptives was 315. At first one kind of oral contraceptive was prescribed for all of them. A second kind was available for those who had untoward reactions from the first, mainly breakthrough bleeding but occasionally other minor symptoms also. About 15 months after the clinic was opened, a survey revealed that 32 per cent of patients were more than two months past the limit of their supply of tablets and had not returned for refill prescriptions. Recent figures from the Chicago Planned Parenthood Association show continued use by from 87.5 per cent to 70.2 per cent between the thirteenth and the eighteenth month.[2]

Two hundred and eleven patients elected to use the intrauterine device. In one case the coil was twice expelled, and the patient then switched to oral contraception. After a year of oral contraception she requested a diaphragm and jelly. Two patients were dropped from the study because of hysterectomy for unrelated causes. Reinsertion was necessary in seven instances, in five because of expulsion of the device and in two because of absence of the thread in the cervical os. There was one pregnancy with coil in place. This patient was delivered of a healthy infant and the loop. There was one definite case of pelvic inflammatory disease with no previous history. There were two other questionable cases of inflammatory disease.

Since the clinic does not function as a part of the department of obstetrics and gynecology, it is probable that we are not fully aware of all complications.

There are a number of things which are apparent after the establishing of such a clinic that might be of assistance to other counties. In the first place, in a county with large rural and urban areas, a single central clinic operating in the county hospital one day a week does not satisfy the need for all who are eligible for its services. An educational program to acquaint eligible persons with the availability of the service was carried on by the local Planned Parenthood Association, but this probably could be done more effectively by the public health nurses and the social workers from the County Welfare Department. The Board of Supervisors now has approved letting welfare workers and health department workers initiate discussion of family planning, provided the discussions do not conflict with religious or moral convictions. One further handicap to the operation of a complete family planning service is that under the definition of eligibles the clinics cannot advise or prescribe for unwed mothers.

There is little question that providing contraceptive advice to mothers who seek it is only a part of complete medical care. Of course, family planning service in a county hospital should be a part of the regular obstetrical and gynecologic service rather than a separate entity. This is the way the service is performed at Bellvue Hospital in New York[3] where it provides for patient instruction, undergraduate and graduate teaching and contraceptive research. Likewise, this is the recommendation of the American Medical Association Committee on Human Reproduction.[1] Even then such a clinic fails to provide family planning service for persons not dealt with in private practice. It is apparent that there is significant need for providing family planning services as part of total medical care in a county hospital. At the same time, this type of service cannot provide for those people who are not undergoing current care in the institution. Some other clinic that is set up outside the hospital and that is more readily available to persons requiring its service, such as the satellite well-baby clinics operated by the Public Health Department, would better serve this purpose. The availability to the patient of family planning services should be convenient on a geographic and transportation basis. Moreover, it should be borne in mind that in persons with limited knowledge regarding reproduction, the motivation for seeking the services of a clinic is limited. Some means of motivation, therefore, should be a part of any complete family planning service.

REFERENCES

1. Committee On Human Reproduction—American Medical Association: The control of fertility, J.A.M.A., 194:462, 25 October 1965.

2. Frank, R., and Tietze, C.: Acceptance of an oral contraceptive program in a large metropolitan area, Am. J. Obst. Gynec., 93:122, 1 September 1965.

3. Lehfeldt, H.: The first five years of contraceptive service in a municipal hospital, Am. J. Obst. Gynec., 93: 727, 1 November 1965.

4. Tietze, C.: The clinical effectiveness of contraceptive methods, Am. J. Obst. Gynec., 78:650, 1959.

North American Blastomycosis in California

Diagnosis of a Case and a Survey with 28 California Observed Cases

DONALD E. CASAD, M.D., AND WILLIAM J. WALDMANN, M.D., *Fresno*
NORMAN E. LEVAN, M.D., *Los Angeles*, AND
ROYAL H. SORENSEN, B.S., *Fresno*

■ *North American blastomycosis is not seen often in California. An exhaustive survey uncovered records of only 28 cases. It is believed that this disease does not occur naturally in California and that a careful study of the history will reveal that a residence or visit to a known endemic area can be found.*

A chart summarizing the results of complement fixation and skin tests for coccidioidomycosis, North American blastomycosis, and histoplasmosis in the 28 cases is included. Use of all three antigens is recommended in cases where there is a difficult diagnostic pulmonary problem, even though there is occasional occurrence of cross-reactions between the three antigens. Finally, an appeal is made to be aware of other mycotic diseases non-edemic to this state.

THE CLINICAL similarity between North American blastomycosis and coccidioidomycosis, as well as some other pulmonary and skin conditions, is such that the former may be overlooked. This can be particularly true in areas endemic for coccidioidal infection where the more common disease is found so routinely that the possibility of the other condition may scarcely become a consideration. The occasional microscopic similarity between the parasitic phase of *Coccidioides immitis* and *Blastomyces dermatitidis* may also contribute to the confusion unless laboratory personnel are completely familiar with the morphological differences and are continuously alert to the possibility that the other may occur.

With the increase in population migration of today the fact that a fungal agent does not occur naturally in an area is not good reason for not thinking of it as a possibility in a patient, wherever examined. Adequate techniques for the diagnosis of North American blastomycosis are available, yet there is good evidence to indicate that failure or long delay in diagnosis of North American blastomycosis does occur.

A patient with North American blastomycosis was observed at the Veterans Administration Hospital, Fresno, after misdiagnosis elsewhere for several years. Moreover, the disease could very easily

---

From Veterans Administration Hospital, Fresno, and the University of Southern California School of Medicine, Los Angeles.

Submitted 22 March 1966.

Reprint requests to: Medical Service—Veterans Administration Hospital, 2615 Clinton Avenue, Fresno 93703 (Dr. Casad).

have been missed by us, not because it was masked or atypical, but because the true diagnosis had never been considered. This case led us to conduct a survey of 21 of the largest hospitals and clinics in California and approximately 300 practicing dermatologists in the state.* The survey brought reports of 27 other cases, which, we believe, must represent nearly all the cases observed in California since about 1940. Table 1 shows general information about these cases. Since all 28 cases could not be published in a single periodical, brief summaries of five representative cases, including that of the patient we observed are presented.†

CASE 1.—A 52-year-old white man, a jackhammer worker, was admitted to Veterans Administration Hospital, Fresno, on 13 June 1963, with a history of shortness of breath that had gradually increased in severity for a year and a half. Other symptoms included a cough which at times produced scanty yellow sputum, loss of weight, wheezing and weakness. A skin lesion had been present on the forehead for several months. The patient had been in a general hospital from 15 December 1962 to 24 January 1963. He was at first considered to have active tuberculosis, yet a smear and a culture of sputum were negative for the disease. Lung cancer was considered. Bronchoscopy and supraclavicular node dissection were done and no evidence of malignant disease was found. Another physician diagnosed pneumoconiosis and referred the patient to our hospital. The patient had come from the Middle West to California in 1950 but had made a visit to Arkansas in 1959. He had had pneumonia as a child, again at age 17, and "dust pneumonia" in 1955.

On physical examination, wheezing was noted in the lungs, with bilateral high-pitched rhonchi, particularly pronounced over the right lung. There was increased sound of whispered voice in the right upper chest. There was a small circumscribed ulcerated lesion on the right forehead (Figure 1), and a small papule on the left forehead.

The routine tests revealed no abnormalities. An x-ray film of the chest showed an extensive lesion in the right upper lobe with nodular areas of increased density associated with cavitation. There were pronounced interstitial inflammatory changes and pleural reaction. Not much reaction was observed in the right lung, but evidence of old basal pleuritis was noted. Emphysematous areas were also present. The heart and mediastinum were shifted to the right (Figure 2).

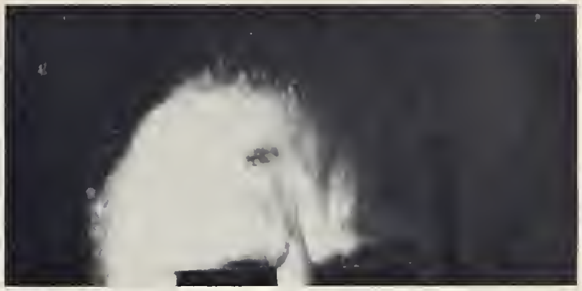


Figure 1.—Ulcerated lesion on forehead of patient in Case 1.

Studies of sputum showed the presence of spherical forms which on potassium hydroxide wet mount were mistakenly considered to be *C. immitis*. When these forms were more critically examined, it was concluded that the spherules of *C. immitis* were not acceptable because: (1) All forms were smaller than mature *C. immitis* spherules, and (2) there was no evidence of spherule endosporulation. Continued search finally demonstrated single-budding forms (Figure 3). This proved to be the key to the diagnosis and revealed all the errors of the past.

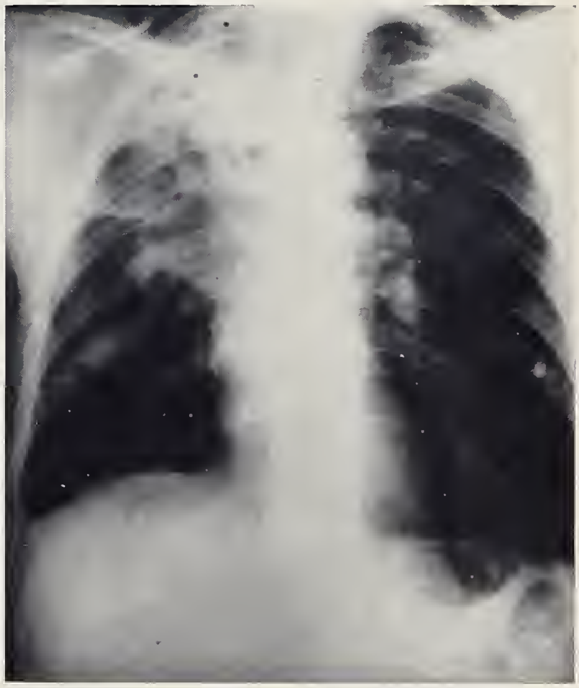


Figure 2.—(Case 1) X-ray film taken early in his hospital course, showing involvement of the right upper lobe.

---

*The majority, 296 dermatologists, were listed in the Pacific Dermatological Registry. Four of these merely had San Francisco APO addresses and were not practicing in California. Several other physicians were contacted who were not on the list. One case was furnished by an internist and two of the dermatological cases were furnished by one of us (N.E.L.).

†Mimeographed reports of all cases are available for interested persons.

TABLE 1.—*Clinical and Epidemiologic Data in 28 Cases of North American Blastomycosis Occurring in California*

Case Number	Duration Approx. Years	Year of Diagnosis	Age	Sex	Race	Probable Area of Acquisition	First Observed as Blastomycosis In California	*Involvement					Site of Diseased Tissue Leading to Diagnosis
								Lung	Skin	Genito-Urinary	Lymph Nodes	Other	
1	4+	1963	52	M	Cauc.	Ark.	VAH Fresno	1	2	..	..	..	Lung
2	6+	1952	31	M	Negro	La.	VA TB Ctr. So. Calif.	1	..	2	..	3	Lung
3	1−	1954	50	M	Cauc.	L.A. Gen. Hosp.	L.A. Gen. Hosp.	..	1	..	..	..	Thumb
4	1−	1950	39	M	Cauc.	Nev.?	So. Calif. Derm.	..	1	..	..	..	Skin face
5	1−	1958	38	F	Cauc.	Ark.	Fresno, Calif. Intern.	..	1	..	..	..	Skin thigh
6	6+	1961	49	M	Cauc.	Chicago	Med. Ctr., L.A.	..	1	..	..	..	Face skin
7	11+	1954	35	M	Cauc.	Okla.	San Joaquin Derm.	..	1	..	..	..	Skin neck
8	20+	1930	32	M	Jap.	Unknown	No. Calif. Derm.	..	1	..	..	..	Skin thigh
9	1+	1956	33	M	Cauc.	Ill.	VAH No. Calif.	1	2	3	..	..	Lung
10	1+	1951	20	M	Negro	Tex.	So. Calif. Co. Hosp.	..	2	..	..	1	Spine
11	35+	1940	59	M	Cauc.	Iowa or Wy.	VAC So. Calif.	2	1	..	..	..	Skin arm
12	7+	1962	?	M	Cauc.	Wisc.	So. Calif. Derm.	2	1	..	..	..	Skin neck
13	1+	1948	45	M	Cauc.	Minn.	U. Med. Ctr. No. Calif.	2	..	..	1	..	Ing. L.N.
14	20+	1942	38	F	Cauc.	N. Dak.	No. Calif. Med. Ctr.	..	..	..	1	2	Spine
15	1+	1959	32	M	Negro	La.	VAC L.A. Calif.	..	1	..	..	..	Skin arm
16	1−	1951	23	M	Cauc.	N. Car.	L.A. Calif. Derm.	1	2	..	..	..	Skin face
†17	3+	1958	22	M	Negro	Ark.	So. Calif. Co. Hosp.	2	1	..	..	..	Skin neck
18	2−	1954	47	M	Cauc.	? Died, no rcd.	L.A. Co. Gen. Hosp.	..	1	..	..	2	Systemic
19	3+	1955	61	M	Cauc.	Ark.	VAH So. Calif.	2	3	4	1	5	Skin node
20	1+	1949	61	M	Cauc.	Iowa	So. Calif. Derm.	2	1	3	..	..	Skin thumb
21	26+	1939	35	M	Latin Am.	Tex. or Ariz.	No. Calif. Derm.	..	1	..	..	..	Skin thigh
22	8+	1955	34	M	Negro	Ark.	VAH So. Calif.	2	1	..	..	..	Skin face
23	1?	1953	38	M	Cauc.	Ark.	San Joaquin Derm.	..	1	..	..	..	Skin arm
24	1+	1937	59	M	Cauc.	Ark.	No. Calif. Med. Ctr.	2	1	3	..	..	Skin thigh
25	1−	1954	35	M	Cauc.	R.I.	VAC So. Calif.	..	1	..	..	..	Skin neck
26	5+	1947	68	M	Cauc.	Nebr.?	U. Med. Ctr. No. Calif.	2	1	..	..	..	Skin buttock
27	13?	1943	33	F	Cauc.	Tex.?	No. Calif. Derm.	..	1	..	..	..	Skin arm & leg
28	2+	1947	49	M	Cauc.	Ill.	L.A. Co. Gen. Hosp.	..	1	..	..	..	Skin foot

---

*The numbers represent the order in which North American blastomycosis was found in the various organs.
†A case published in Ann. Int. Med., 51:780-789, 1959 (Ref. No. 2).

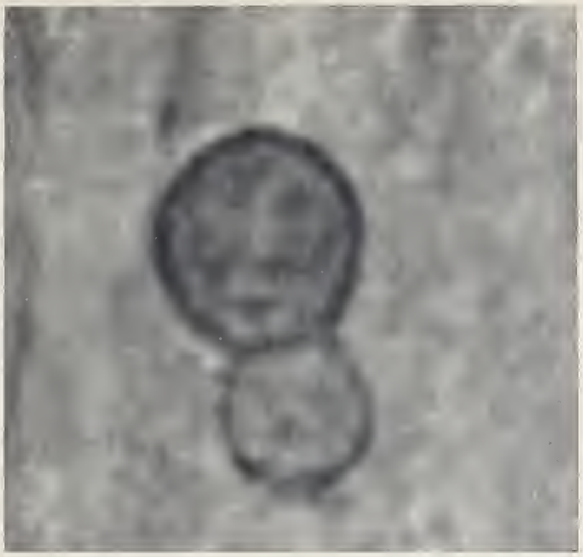


Figure 3.—(Case 1) Typical single-budding forms of spherules observed in specimen taken directly from the lesion (×5,000).

Results of the skin test at this time were negative for intermediate purified protein derivative (PPD) and positive for blastomycin and histoplasmin. The result of a complement fixation test for blastomycosis YP was 4 plus at 1:16. Culture of material from the lesion on the forehead as well as the sputum produced *Blastomyces dermatitides.* All cultures for acid-fast bacilli were negative. The patient was given 2.0 gm of amphotericin B which produced improvement clinically as well as by x-ray observation (Figure 4). Serological tests with blastomycin became negative. Repeated sputum cultures no longer grew *Blastomyces.* When last seen 7 December 1965 the patient was doing well.

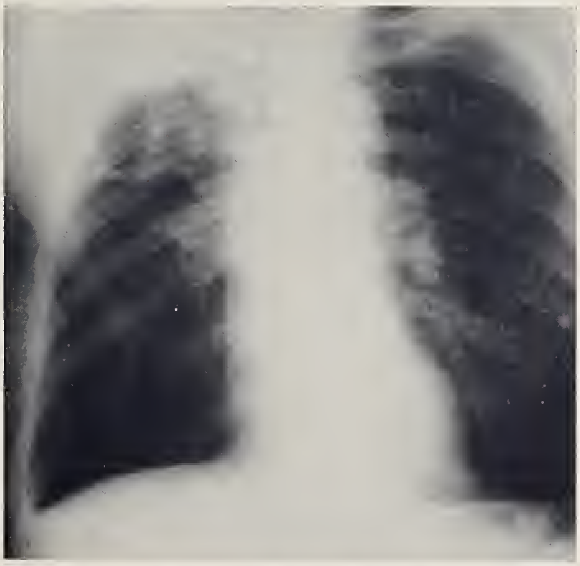


Figure 4.—(Case 1) X-ray film of chest after treatment, showing evidence of some improvement.

TABLE 2.—*Summary of Complement Fixation (CF) and Skin Test (ST) for the Three Mycotic Diseases, Blastomycosis, Histoplasmosis and Coccidioidomycosis, in the 28 Cases Diagnosed as North American Blastomycosis in California*

Case No.	*Blastomyces* Comp. Fix	*Blastomyces* Skin Test	*Histoplasma* Comp. Fix	*Histoplasma* Skin Test	*Coccidioides* Comp. Fix	*Coccidioides* Skin Test	Probable Duration of Disease at Time of Test
1	+	+	−	+	−	−	4
2	+	+	−	−	−	−	4
3	−	+	−	−	−	−	LT 1
4	+	−	+	−	0	0	LT 1
5	−	−	−	0	−	0	LT 1
6	−	−	0	0	0	0	5
7	+	−	0	0	0	0	9
8	−	+	0	0	0	0	Over 30
9	−	−	0	0	0	0	2
10	−	+	0	0	0	0	4
11	−	−	0	0	0	0	Over 30
12	−	+	0	0	0	0	6
13	+	+	0	0	0	0	10
14	0	−	0	−	0	−	10
15	0	+	0	+	0	−	LT 1
16	0	+	0	+	0	0	LT 1
17	+	0	−	0	−	0	2.5
18	+	0	+	0	0	0	2
19	+	0	0	0	0	0	1
20	0	−	0	0	0	0	3
21	0	+	0	0	0	0	2
22	0	0	0	0	0	0	5
23	0	0	0	0	0	0	6
24	0	0	0	0	0	0	2
25	0	0	0	0	0	0	LT 1
26	0	0	0	0	0	0	5
27	0	0	0	0	0	0	10
28	0	0	0	0	0	0	3

+=Positive; −=Negative; 0=Not done or no record of test can be found; LT=Less than.

CASE 7.*—A 35-year-old white male bakery truck driver, a native of Georgia, spent his childhood in Missouri, later moved to Oklahoma and served in the Army in Texas and Louisiana. He was first seen by one of us (N.E.L.) in the San Joaquin Valley 11 August 1954. Early in 1954 he had noted two lesions (one on the back of the neck and one on the left forearm) with pustules which slowly enlarged. He had visited Oklahoma in June 1954, staying on a farm there for one month.

Smears from pustules in the border of the lesion showed double-contoured single-budding spherical cells about 12 microns in diameter, microscopically diagnosed as the organisms of North American blastomycosis. Similar organisms could be seen in tissue sections. An x-ray film of the chest on 22 September 1954 showed some fibrosis that was thought to represent a resolving pulmonary process.

---

*The case numbers correspond to the case numbers as listed in Tables 1 and 2.

Treatment consisted of diethylstilbestrol, potassium iodide and fractional roentgen therapy. While x-ray films of the chest on 17 November 1954 and 16 August 1955 showed the lung fields to be clear, on 12 February 1960 a 7 mm circumscribed density in the fifth anterior interspace was noted. Sputum cultures and guinea pig inoculations were negative for North American blastomycosis. On 11 February 1960 there was a new circular lesion on the scalp. On 12 May 1960 complement fixation was negative for blastomycosis. Although he did not return for follow-up, later information revealed that from mid-June of 1961 to January 1962 the patient was in a Veterans Administration Hospital in Oklahoma. The clinical record there showed that he had had another ulcer, this one on his left heel, of about a year's duration. For this he was treated with amphotericin B. On 8 November 1961 an otolaryngologist who was called into consultation reported "a granulomatous infiltration of epiglottis and larynx, conceivably due to systemic disease." The patient was later admitted to a Veterans Administration Hospital in Los Angeles after illness with influenza, and remained there from 11 June 1964 to 10 November 1964. During that time it was noted that systemic blastomycosis had flared again. Biopsy of the lesions of the epiglottis showed multiple granulomas consistent with blastomycosis. Skin tests at that time with blastomycin were negative and positive with tuberculin. On 8 July 1964 a complement fixation reaction to blastomycin was 4 plus at 1:2, 4 plus at 1:4, 2 plus at 1:8 and negative at 1:16. The patient was still considered to have active disseminated blastomycosis. Intravenous amphotericin B, 2.6 gm, was given. On 23 July 1964 cultures of biopsy material grew *B. dermatitidis*. At that time the patient looked and felt well.

Comment: This case was first diagnosed by one of us, lost to follow-up and subsequently came to light again as a result of our survey. One wonders about other such cases in which the skin is affected, which we now recognize as disseminated disease. The fact that many of the patients are lost to follow-up does not necessarily mean cure. If they could be followed along, how many of these would show further dissemination? For this reason the need for accurate identification is apparent so that when the patients show up in other clinics a continuity of clinical records can be maintained. This case also emphasizes that seemingly innocuous cutaneous lesions thought until recent years to represent local infection are part of the disseminated disease. As with coccidioidomycosis, the skin lesions can be followed, preceded or accompanied by lesions in other organs, so that the need for systemic therapy in such cases must be considered even when the only lesion observed is an isolated small cutaneous granuloma.

CASE 8.—A 32-year-old Japanese man, a native of California, had done mechanical and ranch work. By 1930 he had already been under the private care of a Northern California dermatologist for several years because of widespread granulomatous eruptions. The condition was eventually diagnosed as North American blastomycosis. A culture taken on 13 August 1958 was said to be consistent, microscopically, with *B. dermatitidis*.

He had received potassium iodide and other local treatment over the years, and more recently blastomycin vaccine, all without benefit. He was admitted to a private hospital in Northern California on 10 July 1960 and at that time he said that as far back as age seven he had had a skin lesion that slowly progressed to involve the thigh and lower abdomen. No abnormality was noted on an x-ray film of the chest. A complement fixation test for North American blastomycosis was negative, a skin test positive. In some areas of the body there had been spontaneous healing of the lesions. While in the hospital he was given amphotericin intravenously. The patient's condition improved on administration of amphotericin. "By 20 August 1960," according to a report from the private hospital, "it was noted that the skin had not cleared but it was felt that the patient had had enough Fungizone® (Amphotericin B) for the moment." He was discharged with plans for further treatment later if necessary. There was no further record of this patient.

Comment: We have no knowledge of how extensively this patient may have traveled outside of California if indeed he ever left the state, nor do we know just when the primary infection began. The skin lesion had been noted since age seven. During World War II, some Japanese were in detention camps in areas in which North American blastomycosis is endemic, such as Arkansas. However, this patient's first recollection of skin lesions went back to a time much earlier than the war. An attempt to find this patient again during our survey failed. The record is so incomplete that a conclusion that the infection was acquired in California is not warranted.

CASE 2.*—A 31-year-old Negro kitchen worker, a native of Texas, was admitted to a large Veterans Administration Hospital in Southern California 28 February 1952, soon after coming to California. He had been ill for two and a half or three years. Illness probably started in Shreveport, Louisiana. The symptoms were intermittent fever, chest pain and cough productive of a large amount of sputum. An x-ray film of the chest was interpreted as showing chronic pneumonia with pleural effusion. No response to multiple antibiotics was noted. Exploratory thoracotomy was done 27 May 1952 and a chronic granuloma of the right lung, considered inoperable, was observed. The causative organism was not discovered. Numerous studies for fungi, acid-fast bacilli, viruses and parasitic organisms were made without success, yet tuberculosis was considered most probable. There appeared to be some response to streptomycin and para-amino-salicylic acid. The patient then began to complain of vague abdominal pains, and ascites developed. Paracentesis was carried out repeatedly and on one occasion the intestinal wall was accidentally punctured. Partial intestinal obstruction developed. At abdominal exploration 10 December 1953, an extensive granulomatous process involving the abdomen and peritoneum was observed but the site of obstruction could not be found. The patient was then transferred to a Veterans Administration Center. Acute epididymitis developed and when epididyectomy on the right was done 17 July 1953, a chronic granuloma was observed in that area also. The appearance of the microscopic sections of all operative specimens was suggestive of tuberculosis, but no organisms were seen. Laparatomy was carried out again 12 November 1953, and many small granulomas scattered throughout the abdomen and peritoneal surfaces were seen. At many points there were obstructions due to adhesions. On microscopic study of sections of operative specimens, blastomycosis was noted, and on review the slides from the first hospital were also found to contain North American blastomycosis. A specimen of serum was sent to the Army Medical Service Graduate School, Washington, D.C., and a report was received that it had fixed complement with blastomycin to a titer of 1:32 but was negative for histoplasmin and coccidioidin antigens. Fluid that had been withdrawn by paracentesis 27 September 1953 was now (three months later) reported to be growing *B. dermatitidis*. A blastomycin skin test was then performed and the result was positive. A course of nystatin (Mycostatin®) was given, without benefit. On 4 January 1954 stilbamidine was given, 2-hydroxystilbamidine being unobtainable. First 50.0 mg was given intravenously and then the dose was raised to 150.0 mg daily until 9 January 1954. The patient died 11 January 1954. At postmortem examination, generalized blastomycosis, partial intestinal obstruction and deposits of amyloid in the spleen, kidney and lymph nodes were observed. On 6 January 1954 *B. dermatitidis* was seen growing in cultures of pus taken from the abdomen.

Comment: Although many studies were made for fungus over nearly two years that this patient was in hospital, North American blastomycosis had been consistently missed. Once the idea of North American blastomycosis occurred as a possibility, the diagnosis was easily established on several grounds. No skin lesions ever developed in this case, but generalized dissemination from the lung to the abdomen and epididymus did occur. This was a case in which the diagnosis might have been made directly from early examination of fluid from the lung if proper consideration had been given to it. Had skin dissemination occurred, a dermatological diagnosis might have been made.

CASE 18.—A 47-year-old white man who was admitted to a large county hospital in Southern California 16 June 1954 had been well until hemoptysis developed in mid-1952. He was on the road to recovery when, not long before admittance, he became dyspneic and then had facial paralysis and retinal hemorrhage. At the time of admittance he was believed to have had a cerebral vascular accident, but an x-ray film of the chest showed bilateral granulomatous inflammation of the lungs. His condition progressively deteriorated. On 3 August 1954 a scalene node biopsy was done. The same day, the patient died. Sputum cultures started after admission were reported 12 July 1954 as growing a fungus (later identified as *B. dermatitidis*). At autopsy, granuloma and chronic inflammation of brain, lungs and liver were observed. Histological examination showed granulomatous lesions with refractile bodies consistent with *B. dermatitidis*. Blood taken at autopsy fixed complement with blastomycin at 1:30, and with histoplasmin at 1:8.

---

*This case together with six others from California, numbers 2, 5, 11, 12, 14 and 19 in Table 1, were included in a review of 198 cases of North American blastomycosis in Veterans Administration Hospitals. In addition, Cases 3 and 10 have been reported elsewhere in the literature.[2,5]

It was learned that the patient had been born in California, but the clinical records contained no information as to residence or travel elsewhere.

Comment: During the autopsy in this case, one of the assistants pricked his finger with a knife and contracted primary dermal blastomycosis (Case 3, Tables 1 and 2), the only typical primary dermal infection we found in this survey.[5] The difference between the primary cutaneous mycotic syndrome and extension from dissemination is striking and distinctive.

Discussion

Mycological Features

In almost all the cases in this survey there were long delays in specific diagnosis. In only a few was it made during the primary pulmonary phase; in most not until dissemination to the skin had occurred. Our preconditioned willingness to diagnose coccidioidomycosis after a rather perfunctory direct sputum examination started us along the same path of error that others had gone. It was not until a more critical look was taken that the error was discovered. Schwartz and Baum[3] commented on the confusion in differentiating between the several fungal diseases.

It is likely that the careful following of special mycological techniques may hold the key to success. In the case we observed (Case 1) mature growth and the development of identifying characteristics of the fungus on culture took a month. The routine fungus plates had actually become dehydrated and discarded before the special cultures yielded North American blastomycosis. Thus, a special consideration for the protection of cultures from dehydration and contamination should be provided.

Immunological Considerations

For us, in this area endemic for coccidioidomycosis, the use of immunological diagnostic procedures for this disease have become routine. These tests have aided greatly in early diagnosis (a change from a negative to a positive skin test reaction is an example). Furthermore, in the serological tests we have laboratory aids which will help in following the progress of the disease and determining its severity. We have also come to rely on these tests as gauges of response to therapy. Hence we were eager to learn if there existed a relative significance in these tests when applied to North American blastomycosis. We also wondered about occurrence of cross-reactions between this and other deep mycotic infections with regard to both the skin hypersensitivity and the serological tests.

In Case 1 reported herein, the skin test for histoplasmin was more positive than for blastomycin. We believe that this was not a cross-reaction but rather was dual specific dermal sensitivity for both antigens. The complement fixation test was positive at 1:16 for blastomycin only.

Experienced investigators (Busey and coworkers[1]) in an endemic area for blastomycosis state that in proven cases positive serologic reaction occurs in 48 per cent of cases and skin reactivity in an even smaller proportion (41 per cent).

A summary of the immunological results of the survey of the 28 cases is shown in Table 2. In only three were complete skin and complement fixation tests done for coccidioidin, histoplasmin and blastomycin. Five patients had complete complement fixation tests for all three antigens. Both complement fixation and skin tests were done in 13 cases. In only three were reactions to both tests positive. In two there was positive complement fixation and negative skin test reaction. Four patients had positive skin test reaction and negative complement fixation. Four had negative reaction to both.

In seven of the 28 cases, a result for histoplasmin complement fixation was reported. It was positive in two. It is of special interest that in one of the two positive results, the titer was higher for *H. capsulatum* than for blastomycosis. In this case, the skin test for histoplasmin was negative. In the other case in which complement fixation was positive, no skin test was reported.

Results of complement fixation for coccidioidin were reported in only five cases; in none was the result positive. In five cases coccidioidin skin tests were reported and in none of them was the result positive. This does not imply that coccidioidin tests do not cross react. We know that they sometimes do.[4]

In many of these cases diagnosis was made by dermatologists from histological results. Reports of immunological procedures were not found in the records of many cases which were available to us. Whether this means that the tests were not performed or simply that they were not included in the record summaries, we cannot determine.

It seems apparent that at the times these tests were performed the conditions were usually not optimal. In most cases they were done late, after dissemination, and when the disease state had become chronic and localized to the skin. If the tests had been done early in the course of illness, might not the results have been better, perhaps paralleling those observed in like circumstances in coccidioidomycosis?

We have come to believe that the use of all three antigens in difficult diagnostic pulmonary lung problems should be recommended both for serological and dermal tests. Case 2 is a good example of cases in which this would be helpful: If all the tests had been used in that case, the diagnosis should have been made much earlier, with benefits in evaluation of both diagnosis and prognosis.

Not until more consistent application of these diagnostic techniques have been observed can the worth of using the immunological criteria as a guide to therapy be determined. One should not overlook, however, that there may be instances in which a histoplasmin or coccidioidin skin test may be more strongly positive in a case of blastomycosis than is a blastomycin test (Case 1). It should be borne in mind also that the complement fixation tests for other deep mycotic infections at times show higher titers than for blastomycosis itself (Case 4).

We continue to wonder if a more organized use of all of these immunological tests would not eventually approach the significance we have seen in their use in the diagnosis of coccidioidomycosis.

GENERIC AND TRADE NAMES OF DRUGS

Amphotericin B—*Fungizone.*
Nystatin—*Mycostatin.*

REFERENCES

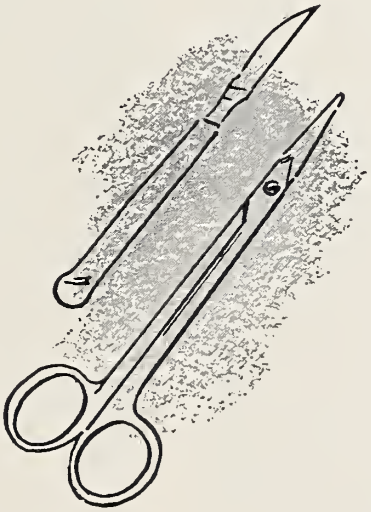
1. Busey, J. F., Baker, R., Birch, L., Buechner, H., Chick, E. W., Justice, F. K., Matthews, J. H., McDearman, S., Pickar, D. N., Sutliff, W. D., Walkup, H. E., and Zimmerman, S.: Blastomycosis. I. A review of 198 collected cases in Veterans Administration Hospitals, Am. Rev. Resp. Dis., 89:659-672, 1964.

2. Carmody, E. J., and Tappen, W.: Blastomycosis meningitis: Report of a case successfully treated with amphotericin B, Ann. Int. Med., 51:780-789, 1959.

3. Schwartz, J., and Baum, G. L.: Blastomycosis, Am. J. Clin. Path., 21:999-1029, 1951.

4. Smith, C. E., Saito, M. T., and Beard, R. R.: Histoplasmin sensitivity and coccidioidal infection. I. The occurrence of cross-reactions, Am. J. Public Health, 39:722, 1949.

5. Wilson, J. W., Cawley, E. P., Weidman, F. D., and Gilmer, W. S.: Primary cutaneous North American blastomycosis, Arch. Dermat. and Symph., 71:39-45, 1955.



Systemic Agents in the Management Of Acne

RONALD M. REISNER, M.D., *Torrance*

■ *Topical therapy including incision of pustules and injection of corticosteroids into nodular and cystic lesions remains the mainstay of the management of acne.*

Systemic agents, including diuretics, corticosteroids, broad spectrum antibiotics and progestin-estrogen combinations are significant and valuable additions to the therapy of resistant pustulocystic acne. They are, however, not without side effects and they should be reserved for carefully selected patients for whom they may, when used with discretion, produce gratifying results with relatively low risk.

UNDERSTANDING OF THE complex interrelationship among the various factors influencing the development of acne has improved with increased study of the fundamental pathogenesis of this common and intriguing disease. Despite this growing body of knowledge, therapy for acne remains to a great extent empiric, with the rationale for a given modality often following rather than preceding its use. For many years, topical therapy has remained the mainstay of the management of acne. Topical measures have included cleansing of the skin with a variety of agents, the application of an almost endless series of drying and peeling lotions which consist primarily of various combinations of sulfur, resorcin and salicylic acid, desquamation of the skin with carbon dioxide slush or ultra violet light, mechanical extraction of closed comedones (whiteheads), and open comedones (blackheads), incision and drainage of pustules and fluctuant cysts and, more recently, the injection of various corticosteroids directly into non-fluctuant cystic and nodular lesions.

Such measures, although tedious and time-consuming, have often provided good control of this eventually self-limiting disease while waiting for the active process to become quiescent. However, the more severe and disfiguring forms of pustular and cystic acne with their sometimes disastrous emotional impact on the developing teen-age boy or girl and their severe residual scarring, both physical and psychological, have often eluded control with topical agents alone. Although the use of dermabrasion has been partially successful in reversing the permanent scarring changes following upon severe pustulocystic acne, a far more preferable approach is prevention of the scarring by adequate control of the original disease process.

The development of several newer means of systemic therapy has helped improve the management of these more severe forms of the disease. Most significant among them is the long-term administration of chemotherapeutic agents, particularly the broad spectrum antibiotics, and the use of

---

From the Department of Medicine, Division of Dermatology, Harbor General Hospital, Torrance, and U. C. School of Medicine, Los Angeles, University of California Center for the Health Sciences, Los Angeles.

Supported in part by a grant from the Elliott and Ruth Handler Foundation.

Presented before the Section on Dermatology at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: Division of Dermatology, Harbor General Hospital, 1000 West Carson Street, Torrance 90509.

estrogen-progestin combinations either together or sequentially. Before proceeding to a more detailed consideration of these agents, I would like to comment briefly on the influence of other systemically administered agents in the management of acne.

Diet

In a broad sense, alterations in diet may be considered as systemic therapy, and in the management of acne there is a long list of dietary restrictions that have been proposed at various times, including such foods as chocolate, cheese, milk, nuts, fats, carbohydrates, shellfish. Although many dermatologists still incriminate chocolate[32] as an offender, there is growing acceptance for the view expressed by Hopkins in 1958[21] that "We have no reliable evidence that the food one eats has anything to do with outbreaks of acne . . .," and it would seem unnecessary at this time to impose dietary restrictions on most patients with acne.

Vitamin A

Although a venerable member of the anti-acne armamentarium, vitamin A has never been unequivocally shown to be of value in the management of any form of acne, and a recently carefully controlled double-blind study[1] comparing the results of vitamin A administered orally in a dose of 150,000 units daily for 12 weeks and a lactose placebo, revealed no difference in effectiveness between the two. An important point emphasized in this study was the difficulty of accurately assessing the influence of treatment on the course of acne by clinical impression alone. The more objective evidence provided by serial photographs compared by a panel of qualified observers at a later date proved more accurate and reliable, and at the same time highlights the difficulty of making objective reproducible clinical assessments in a therapeutic trial in a disease subject to spontaneous exacerbation and remission. There does not seem to be a firm place for vitamin A in the management of acne at this time.

Diuretics

Many female patients are noted to have a flaring of acne during five to 10 days preceding the onset of menses. These patients often also have a gain in weight during the same time.[31] Since it is known that there is no alteration in sebum production during the various phases of the menstrual cycle,[31] it would seem reasonable to postulate a possible relationship between premenstrual retention of fluid and exacerbation of acne. And indeed in many such patients the administration of a diuretic during a period of from five to 14 days before menses has proven effective in eliminating or decreasing the severity of these premenstrual flares. The thiazide diuretics are widely used for this purpose, although any effective diuretic well tolerated by the patient should be equally effective. Double-blind studies are needed for final evaluation of this modality.

Corticosteroids

Although corticosteroids may themselves produce a papulopustular follicular acneform eruption, they do appear to have an effective role in reducing the inflammatory lesions of severe cystic acne. However, double-blind studies are also needed here. Because of the hazardous side effects associated with corticosteroid administration, use of them should be reserved for selected severe cases of cystic acne with extensive involvement unresponsive to other modes of therapy. Treatment should be of the shortest duration possible, with a suggested initial daily oral dose in the range of 20 to 30 mg of prednisone or its equivalent, with gradual withdrawal preferably within a month or so, although in exceptional cases long-term treatment may be necessary, with its concomitant increase in risk of side effects.

Chemotherapeutic Agents

Since Andrews[2] reported the successful use of systemically administered antibiotics in the management of acne in 1951, a number of studies* have been published reporting the good therapeutic results obtained in the management of pustular and cystic acne by use of a variety of antibiotics and sulfonamides. These have included several controlled double-blind studies.[20,40,49] Such double-blind studies are especially important in evaluating therapy in a disease such as acne which is subject to repeated spontaneous exacerbations and remissions. Most of the recent interest in antibiotic therapy for pustular and cystic acne has centered around the tetracyclines,† and it is with this family of broad spectrum antibiotics that the remainder of this discussion will be concerned.

Although, as noted previously, a number of uncontrolled studies have been reported demonstrating the value of the tetracyclines in pustular and cystic acne, there has been some conflict of opinion as to their value expressed in several recent controlled studies.

---

*Reference Nos. 4, 9, 10, 11, 20, 22, 40, 45, 46, 49.

†Reference Nos. 9, 10, 12, 16, 20, 31, 38, 39, 40, 49.

Hicks,[20] Stewart[40] and Wansker[49] have all published double-blind controlled studies showing the apparently unequivocal value of orally administered tetracyclines in many patients with pustulocystic acne. The matched placebo-treated and tetracycline-treated patients studied by Freinkel and coworkers[16] showed similar clinical results, and in both groups a reduction in free fatty acids of the surface lipids paralleled clinical improvement. On the other hand, Smith and coworkers[34] in England and Crounse[12] here in the United States both failed to find any significant differences between placebo and tetracyclines in controlled double-blind studies of the treatment of acne. The general preponderance of opinion, however, favors the tetracycline group of broad spectrum antibiotics as active and useful agents in the management of pustulocystic acne.

In view of some disagreement as to their efficacy on the one hand and, on the other, their apparent ability to control severe pustulocystic acne with very small doses over a long period, it is of interest to inquire into the mechanism by which the tetracyclines exert their apparently favorable effect. Although the precise and final answer to this question is not yet known, a fairly convincing web of circumstantial evidence has been developed which points strongly toward at least one probable mode of action.

In general, patients with acne produce more sebum than do normal persons, although acne does not develop in all persons with high sebum levels.[30] It has also been demonstrated that the injection of whole sebum into the skin results in a pronounced lymphocytic inflammatory response and that sebum with the free fatty acids removed excites only a minimal inflammatory response.[41,44] In contrast, the injection into the skin of free fatty acids derived from sebum results in a decided inflammatory response similar to that caused by whole sebum, with rupture of pilosebaceous follicles and other histologic changes comparable with those occurring in acne.[44] This strongly suggests that the free fatty acids in sebum are largely responsible for the inflammatory reaction in acne. However, the presence of free fatty acids is not alone sufficient to produce inflammation in acne, since levels of free fatty acids in surface lipids are essentially the same in groups with and without acne.[16] Other factors as yet unelucidated appear to be necessary for free fatty acids to incite acne.

It is further known that there is little or no free fatty acid in lipids of intact sebaceous cysts[25] which suggests that the lipids of freshly secreted sebum must undergo hydrolysis during their passage through the follicular canal to the surface of the skin where sebum is found to contain up to 23 per cent free fatty acid.[19] Although there are several possible sources of such lipolytic activity, Scheimann and coworkers[36] have presented evidence that skin bacteria are a major source of such lipolytic activity.

Descriptions of the organisms comprising the cutaneous bacterial flora in acne have in part been somewhat conflicting; but two recent studies[37,38] have clearly demonstrated that two organisms constitute the overwhelming majority of the bacteria found in all forms of acne. Both are Gram-positive. One is an aerobe, *Staphylococcus albus,* and the other an anaerobic diptheroid, *Corynebacterium acnes.*

It has further been shown that injection of living *Corynebacterium acnes* into sterile steatocystomas (intact sebaceous cysts) results in rapid proliferation of the organism accompanied by the production of "products which irritated the lining, leading to leakage and rupture."[23] This in turn gives rise to a pronounced inflammatory reaction resembling an inflamed acne cyst. In view of the predominance of *Corynebacterium acnes* among the follicular bacteria and its probable role in the production of free fatty acids by hydrolysis of the esterified fatty acids of sebum in the follicle and on the surface, it seems reasonable to speculate that the "products" resulting from the injection of living *Corynebacterium acnes* into the sterile steatocystomas might well be free fatty acids.

Puhvel and coworkers[33] have shown that levels of antibody to *Cornyebacterium acnes* are elevated in the serum of patients with acne; whereas levels of antibody to *Staphylococcus albus*[34] are not; and this may reflect a direct involvement of *C. acnes* in the development of acne.

Accordingly, a reasonable working hypothesis might suggest that the inflammatory lesions of pustulocystic acne are due in part at least to the release of free fatty acids in the follicular ducts as a result of the lipolytic activity of *Corynebacterium acnes on sebum,* and that in susceptible persons these free fatty acids leak through the follicle wall, resulting in the clinical findings of pustules, cysts and inflammatory nodules.

Since *Corynebacterium acnes* is known to be highly sensitive to the broad spectrum antibiotics, including the tetracyclines,[29] a suitable test of this hypothesis would be to correlate the influence of the tetracyclines on the clinical course of acne with their influence on sebum free fatty acid levels. Just such a critical study was undertaken by Freinkel and coworkers,[16] and the results support the hypothesis noted above.

They found that administration of tetracycline orally to adult patients resulted in a qualitative alteration in the composition of sebum, the total quantity produced remaining unchanged. The change observed was a reduction in the concentration of free fatty acids, and it correlated well with amelioration of the inflammatory process and clinical improvement of the patient's acne. Although the study did not clearly demonstrate the basis for this activity of tetracyclines, the evidence previously cited strongly suggests that they act by reducing the number of *Corynebacterium acnes* organisms on the skin, and hence, their lipolytic activity.

The tetracyclines suppress *Corynebacterium acnes* on the skin as long as they are administered, but the organisms multiply rapidly after these agents are discontinued, usually reaching pretreatment levels within approximately two weeks.[18] Clinical relapse of patients with pustulocystic acne correlated well with rising values for free fatty acids in sebum ater discontinuation of tetracycline therapy,[16] further suggesting a relationship between the number of *Corynebacterium acnes* organisms, the amounts of free fatty acids and the clinical manifestations of acne.

Thus there is a rationale (developed, to be sure, long after the first clinical observations of its activity) to account for the efficacy of the tetracyclines in pustulocystic acne; and development of this rationale has illuminated and been illuminated by a better understanding of fundamental pilosebaceous pathophysiology.

Despite their effectiveness, the tetracyclines and other antibiotics should not, of course, be used in the routine management of all patients with acne, but should be reserved for the more severe, extensive and recalcitrant forms of the disease which do not yield to conventional therapy as alluded to above. In such patients, gratifying results may be observed within two to three weeks after the initiation of therapy and may be maintained as needed for months or even years, often with extremely low dosages. A typical regime consists of the use of a tetracycline in doses of 250 mg orally four times daily for the first one to three weeks until a positive result is obtained. This is followed by a gradual reduction of the dose to the lowest dose which is sufficient to maintain control of the acne. In some patients this may be as little as 250 mg every second or third day. In females who experience premenstrual exacerbations, uncontrolled with diuretics, an increase in the tetracycline dosage during the 10 to 14 days preceding the onset of menses to a level greater than the maintenance dose may bring about improved control.

Although the tetracyclines have been remarkably free of serious toxicity despite prolonged usage, a number of side effects are known to be associated with their use. Some of these side effects are trivial and do not ordinarily necessitate discontinuation of treatment. Others are grave and even life-threatening and represent absolute contraindications to tetracycline therapy for acne.

Among the serious complications are the development of severe and at times fatal liver damage in pregnant women given large doses of tetracycline in the presence of preexisting renal disease such as pyelonephritis.[51] The susceptibility of women to this complication may be further enhanced during the latter half of pregnancy because of a physiological decrease in clearance from the liver during this period.[8] Another problem associated with tetracycline therapy in the face of preexisting renal disease is the development of azotemia associated with decreased renal excretion of tetracycline, bringing about higher blood levels thus enhancing inhibition of protein synthesis and presenting the already damaged kidney with an increased nitrogenous load.

Another significant problem associated with long continued administration of tetracycline is discoloration of both temporary and permanent teeth.[47] This yellow-brown pigmentation may result from tetracycline administered from the fourth fetal month to the twelfth year of life.[24,53] Enamel hypoplasia has been noted in some, but not all, children with discoloration of the teeth due to tetracycline.[53] Reversible retardation of bone growth has also been observed in prematures receiving tetracycline.[7]

Thus it is apparent that tetracycline therapy is completely contraindicated for the management of acne in pregnant women and in children below the age of 12 years, as well as in patients with preexisting renal or hepatic disease, especially in view of the usually long-term nature of such therapy.

Allergic reactions to tetracycline are rare[27] although anaphylactoid reactions have been reported.[13,15] With the exception of demethylchlortetracycline in which the incidence approaches 20 per cent,[5] photosensitization reactions are not common with tetracyclines.[5,6,26] Careful history coupled with clinical follow-up should avoid or promptly identify these side effects.

Nausea, vomiting, diarrhea and superinfection with bacteria and yeasts are all uncommon complications in otherwise healthy persons[27] and the debilitated patient who would be more susceptible to these problems is not likely to be a candidate for long-term tetracycline therapy for acne.

The incidence of intercurrent infections resistant to treatment appears to be no higher in acne patients treated with antibiotics for long periods than it is in comparable patients not so treated.[3,46]

Finally, since degraded tetracyclines may produce severe interference with renal tubular function, only "in date" drug which has not been subjected to extremes of heat and humidity should be used.[14,50]

Thus the tetracyclines have at present a secure place in the management of carefully selected patients with recalcitrant pustulocystic acne, but their use must be carefully supervised, and although at times it may seem convenient, refillable prescriptions should obviously not be dispensed.

Progestin-estrogen therapy

The androgen-stimulated increase in sebaceous gland activity occurring at puberty which provides the necessary but not sufficient basis for the development of acne vulgaris may be antagonized effectively by sufficient levels of estrogen. The precise mechanism of this activity is not established, but it is thought to be due to inhibition of ovarian androgen.[43] To exert this effect, doses well in excess of normal physiologic replacement amounts are required—at least 0.06 mg of ethynyl estradiol daily.[31] However, the pronounced menstrual abnormalities and menometrorrhagia resulting from employment of such doses has made estrogen alone an unsatisfactory therapeutic agent in many patients who might otherwise have benefited from such therapy. Because of feminization, estrogen therapy is, of course, not applicable to male patients.

The more recent development of a large series of progestin-estrogen combinations for contraceptive use has provided a group of compounds which have both the sebum suppressive effect desired for control of acne and good control of menstrual cycling.[42,43] There are a wide variety of such agents—one recent publication[35] lists over 70—but evidence indicates that their favorable effect in acne is due almost entirely if not entirely to the sebum-suppressive effect of the estrogenic moiety.[43] Quantitative sebum determinations demonstrate that clinical improvement and decrease in sebum levels are well correlated and that cyclic progestin-estrogen therapy for approximately two to five months is required to produce sebum suppression and clinical improvement in most women with acne,[43] the majority responding within the first three cycles.[28]

Such therapy should not be undertaken without adequate examination before it is begun, and careful observation during therapy is necessary.

An adequate pretherapy examination should include, at a minimum[17,35,48]

(1) History to rule out previous thromboembolic phenomena, thrombophlebitis, cerebrovascular accident, or estrogen dependent neoplasms;

(2) Physical examination including bimanual pelvic examination and speculum examination of cervix and vaginal vault to rule out malignant disease and fibroid tumors of the uterus (in young women with a virginal introitus, rectal examination is ordinarily preferred); breast examination to rule out carcinoma of the breast and, in the light of recent evidence,[48] routine ophthalmologic examination;

(3) Laboratory examination to include routine blood cell count, urinalysis, cytologic examination of cervical exudate, and a liver function test.

If results from these studies are within normal limits, selected women with recalcitrant pustulocystic acne unresponsive to the measures previously discussed may be considered for cyclic progestin-estrogen therapy of the older combined type or the more recent sequential type. Although there may be a flare of the acne during the first one or two cycles, control can ordinarily be expected within three to four cycles, occasionally five.[28,31,42,43] If no improvement is noted by the end of the fifth cycle, it is unlikely to occur with continued use of the compound although increased dose levels may achieve control.

These agents are not without side effects, which should be carefully watched for. They include:

- Nausea and vomiting. These are the most frequently encountered side effects and often disappear with time. They may be minimized by taking the drug at bedtime.[17,28,35,42,43]
- Breakthrough bleeding. The incidence of this side effect may be diminished by regularity of tablet intake[17] and usually can be controlled by increasing the dosage.[35]
- Weight gain. This may be secondary to fluid retention and it may spontaneously remit or require diuretic therapy.[17,31,35]
- Mastalgia and breast engorgement. This usually improves with change to a preparation with less estrogen.[17]
- Dizziness, headache, abdominal pain, pelvic pain, fatigue and nervousness have all been occasionally noted but are difficult to evaluate.[17,35]
- Chloasma. This occasionally becomes enough of a cosmetic problem to require discontinuation of the drug.[17,28,43]
- Increased bromsulfalein retention. This has been occasionally noted but to date has not been shown to be clinically significant and may disappear despite continued therapy.[17]
- Thrombophlebitis. Although this has been the focal point of much interest, most observers seem to feel that there is no causal relationship between the use of progestin-estrogen combinations and thrombophlebitis.[17,35]
- Neuro-ophthalmological complications. A recent survey by Walsh and coworkers[48] suggested the possibility of the association of progestin-estrogen therapy and the occurrence of a variety of problems, including pseudotumor cerebri, stroke syndromes, intraocular vascular lesions and severe headaches of the migraine type. The investigators emphasized the preliminary nature of their findings and concluded that no firm relationship between the observed pathologic changes and the use of the progestational drugs was established, but that further study is warranted. In the interim it would appear prudent to be aware of the possibility and to withhold therapy from patients who develop during therapy or who give a previous history of cerebrovascular disease, partial or complete visual loss, proptosis, diplopia, migraine, papilledema, retinal vascular lesions or thromboembolic phenomena.
- Irregular menses, amenorrhea and infertility of varying and in some instances prolonged (12 to 18 months) duration have recently been reported following withdrawal of therapy with progestin-estrogen combinations.[52] This may be of particular importance to a young woman who has not yet had her family.

Despite this long list of side effects the actual incidence of difficulty has been remarkably low, considering the vast number of women using these compounds for contraception.[17,28,35] However, acne does not carry the risk that pregnancy may in selected situations, and therefore patients must be chosen carefully and this group of drugs reserved for patients with recalcitrant acne.

REFERENCES

1. Anderson, J., and Stokoe, I. H.: Vitamin A in acne vulgaris, Brit. Med. J., 5352:294-96, 3 August 1963.

2. Andrews, G. C., Domonkos, A. N., and Post, C. F.: Treatment of acne vulgaris, J.A.M.A., 146:1107, 1951.

3. Baer, R. L., and Witten, V. H.: Editorial Comment in Year Book of Dermatology, 1960-1961, Yearbook Medical Pub., Chicago, 1961, p. 67.

4. Brandt, R., and Beyer, A.: Controlled study of sulfadimethoxine (Madribon) in infected acne, Ohio Med. J., 56:1649-52, December 1960.

5. Cahn, M. M., Levy, E. J., and McMillen, J. A.: Nature and incidence of photosensitivity reaction to demethyl chlortetracycline, Arch. Derm., 84:485-89, September 1961.

6. Clendenning, W. E.: Complications of tetracycline therapy, Arch. Derm., 91:628-32, 1965.

7. Cohlan, S. Q., Bevelander, G., and Tiamsic, T.: Growth inhibition of prematures receiving tetracycline, Am. J. Dis. Child., 105:453-61, May 1963.

8. Combes, B., Shibata, H., Adams, R., Mitchell, B. D., and Trammell, V.: Alteration in sulfbromophthalein sodium removal mechanisms from blood during normal pregnancy, J. Clin. Invest., 42:1431-42, September 1963.

9. Cornbleet, Theodore: Long-term therapy of acne with tetracycline, Arch. Derm., 83:414-16, March 1961.

10. Cornbleet, Theodore: Comparison of demethylchlortetracycline and tetracycline in acne, Arch. Derm., 89:204-06, 1964.

11. Cronk, G. A., Naumann, D. E., Heitzman, E. J., Marty, F. N., McDermott, K. J., and Vercillo, A. A.: Tetracycline hydrochloride in treatment of acne vulgaris, Arch. Derm., 73:228-35, March 1956.

12. Crounse, Robert G.: The response of acne to placebos and antibiotics, J.A.M.A., 193:906-10, September 1965.

13. Editorial: Anaphylactic reaction to tetracycline, J.A.M.A., 192:992, 1965.

14. Editorial: Effects of tetracycline and degradation products, J.A.M.A., 194:143, 1963.

15. Fellner, M. J., and Baer, R. L.: Anaphylactic reaction to tetracycline in a penicillin-allergic patient, immunological studies, J.A.M.A., 192:997-98, 1965.

16. Freinkel, R. K., Strauss, J. S., Yip. S. Y., and Pochi, P. E.: Effect of oral tetracycline on fatty acids of human sebum, New Engl. J. Med., 273:850-54, 14 October 1965.

17. Goldzieher, Joseph W.: Newer drugs in oral contraception, Med. Clin. North Amer., 48:529-45, March 1964.

18. Goltz, R. W., and Saemandus, K.: Oral tetracycline

treatment on bacterial flora in acne vulgaris, Arch. Derm., 93:92-100, January 1966.

19. Haahti, E.: Major lipid constituents of human skin surface with special reference to gas chromotographic method, Scand. J. Clin. Lab. Invest., 13:1-108, 1961.

20. Hicks, J. H.: Demethylchlortetracycline: A double-blind study in the treatment of acne with attention to side effects noted, Southern Med. J., 55:357-60, April 1962.

21. Hopkins, H. H.: Management of acne in adolescents, Quart. Rev. Pediat., 13:10, 1958.

22. King, W. C., and Forbes, M. A.: Use of tetracycline in treatment of acne vulgaris: Clinical study, Southern Med. J., 49:875-79, August 1956.

23. Kirschbaum, J. O., and Kligman, A. M.: The pathogenic role of corynebacterium acnes in acne vulgaris, Arch. Derm., 832-33, December 1963.

24. Kline, A. H., Blattner, R. J., and Lumin, M.: Transplacental effect of tetracyclines on teeth, J.A.M.A., 188:178-80, 13 April 1964.

25. Nicolaides, N., and Wells, G. C.: On biogenesis of free fatty acids in human skin surface fat, J. Invest. Derm., 29:423-33, 1958.

26. Orentreich, N., Harber, L. C., and Tromovitch, T. A.: Photosensitivity and photo-onycholysis due to demethylchlortetracycline, Arch. Derm., 83:730-37, May 1961.

27. Ory, E. M., and Yow, E. M.: The use and abuse of the broad spectrum antibiotics, J.A.M.A., 185:273-79, 27 July 1963.

28. Palitz, L. L., Milberg, I. L., and Kantor, I.: Enovid for acne in the female, Skin, 3:243-45, August 1964.

29. Pochi, P. E., and Strauss, J. S.: Antibiotic sensitivity of Corynebacterium acnes (Propionibacterium acnes), J. Invest. Derm., 36:423-29, June 1961.

30. Pochi, P. E., and Strauss, J. S.: Sebum production, casual sebum levels, titratable acidity of sebum and urinary fractional 17-ketosteroid excretion in males with acne, J. Invest. Derm., 43:383-88, 1964.

31. Pochi, P. E., and Strauss, J. S.: Treatment of acne; from Modern Treatment, edited by D. J. Demis, J. A. Rider, and H. C. Moeller, Hoeber Medical Division, Harper and Row, New York, 1965, pp. 847-59.

32. Proceedings of the XII International Congress of Dermatology, Excerpta Medica Foundation, New York, 1963, vol. 2, p. 999.

33. Puhvel, S. M., Barfatani, M., Warnick, M., and Sternberg, T. H.: Study of antibody levels to Corynebacterium acnes, Arch. Derm., 90:421-27, October 1964.

34. Puhvel, S. M., Warnick, M. A., and Sternberg, T. H.: Levels of antibody to Staphylococcus epidermidis in patients with acne vulgaris, Arch. Derm., 92:88-90, July 1965.

35. Roland, Maxwell: Progestagen Therapy, Charles C Thomas, Springfield, Illinois, 1965.

36. Scheimann, L. G., Knox, J., Shes, D., and Rothman, S.: Role of bacteria in formation of free fatty acids on human skin surface, J. Invest. Derm., 34:171-74, 1960.

37. Shehadeh, N. H., and Kligman, A. M.: The bacteriology of acne, Arch. Derm., 88:828-31, December 1963.

38. Smith, M. A., and Waterworth, P. M.: The bacteriology of acne vulgaris in relation to its treatment with antibiotics, Brit. J. Derm., 73:152, 1961.

39. Smith, M. A., Waterworth, P. M., and Curwen, M. P.: A controlled trial of oral antibiotics in the treatment of acne vulgaris, Brit. J. Derm., 74:86-90, March 1962.

40. Stewart, W. D., Maddin, S., Nelson, A. J., and Danto, J. L.: Therapeutic agents in acne vulgaris. I. Tetracycline, Canad. Med. Assoc. J., 89:1096-97, 1963.

41. Strauss, J. S., and Kligman, A. M.: Pathologic dynamics of acne vulgaris, Arch. Derm., 82:779, 1960.

42. Strauss, J. S., and Pochi, P. E.: Effect of Enovid on sebum production in females, Arch. Derm., 87:366-68, March 1963.

43. Strauss, J. S., and Pochi, P. E.: Effect of cyclic progestin-estrogen therapy on sebum and acne in women, J.A.M.A., 190:815-19, 30 November 1964.

44. Strauss, J. S., and Pochi, P. E.: Intracutaneous injection of sebum and comedones—Histological observations, Arch. Derm., 92:443-56, October 1965.

45. Stritzler, C., and Lawrence, F.: Long-term oleandomycin therapy for acne, Arch. Derm., 81:958-61, June 1960.

46. Sulzberger, M. B., Witten, V. H., and Steagall, R.: Treatment of acne vulgaris: Use of systemic antibiotics and sulfonamides, J.A.M.A., 173:1911-15, 27 August 1960.

47. Wallman, I. S., and Hilton, H. B.: Teeth pigmented by tetracycline, Lancet, 1:827-29, 21 April 1962.

48. Walsh, F. B., Clark, D. B., Thompson, R. S., and Nicholson, D. H.: Oral contraceptives and neuro-ophthalmologic interest, Arch. Ophth., 74:628-40, November 1965.

49. Wansker, B. A.: Antibiotics and pustulocystic acne: A long-term double-blind evaluation, Arch. Derm., 84:146-48, July 1961.

50. Wegienka, L. C., and Weller, J. M.: Renal tubular acidosis caused by degraded tetracycline, Ann. Int. Med., 14:232-35, August 1964.

51. Whalley, P. J., Adams, R. H., and Combes, B.: Tetracycline toxicity in pregnancy, J.A.M.A., 189:357-62, 3 August 1964.

52. Whitelaw, M. J., Nola, V. F., and Katman, C. F.: Irregular menses, ammenorrhea and infertility following synthetic progestational agents, J.A.M.A., 195:780-82, 28 February 1961.

53. Witkop, C. J., and Wolf, R. O.: Hypoplasia and intrinsic staining of enamel following tetracycline therapy, J.A.M.A., 185:1008-11, 28 September 1963.

How Community Cardiovascular Disease Programs Can Aid the Private Physician

J. GORDON BARROW, M.D., *Atlanta*

THE SECOND National Conference on Cardiovascular Diseases, after a careful study of the need for community programs in the control of heart disease, published recommendations emphasizing the necessity for greater use of community facilities and ancillary medical personnel if physicians are to continue to give the best possible medical care to the rapidly increasing population. I will point out a few ways in which community programs can aid the private physician in the care of patients with cardiovascular disease.

Rheumatic Fever, Rheumatic Heart Disease, Congenital Heart Disease

It is estimated that one school child in every two hundred has either rheumatic or congenital heart disease and that only about half of them are known to a physician. Therefore, adequate case-finding is a primary objective of the community health effort. School health programs have been very productive in finding new cases of heart disease in the community. There has been much interest in screening of children in the schools by tape recording of heart sounds for interpretation by a cardiologist, or by other means involving the use of automatic equipment. Thus far, however, this has not proved more satisfactory than the ordinary school physician with his stethoscope, and it must be considered an experimental approach.

There are several observations which I feel should be made about school health programs. It is not enough merely to identify the murmur and send a note home asking the mother to take the child to a private physician. If this happens, probably less than half of the children are actually followed up by a physician, and many of them are labeled by their own mothers as having heart disease when in reality they may have only a functional murmur. I would strongly urge that every school health program screening for heart disease have associated with it a secondary screening program where the child with a significant murmur is reexamined by a cardiologist who evaluates the murmur before sending the child back to a private physician. In this way about eight out of 10 children found to have murmurs in the routine screening can be labeled as having only functional murmur, and the private physician is spared the necessity for having to see the child. Therefore, only the children with organic heart disease are referred, or returned, to private physicians for further care.

When heart disease is found by the private physician, it is important, after his examinations are completed, that he notify the school of his findings and any recommendations he may wish to make as to restriction of the child's activity. A thorough understanding of exactly what the child can and cannot do is particularly important in such conditions as cyanotic congenital heart disease.

If advanced diagnostic procedures or surgical therapy are needed and the patient cannot afford private medical care, there are now in most states facilities (such as cardiac clinics for indigents and Crippled Children's Program clinics) through which the needed care can be supplied regardless of inability to pay.

In Georgia we have found it very helpful to utilize public health nursing services in all of our cardiovascular disease programs. As to those pro-

---

The author is Director of the Cardiovascular Disease Control Service, Georgia Department of Public Health.

Presented before the Section on Preventive Medicine and Public Health at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: Georgia Baptist Hospital, Atlanta, Georgia 30312.

gram areas mentioned above, it has been our experience that public health nurse follow-up of patients scheduled for cardiac catheterization, angiocardiography or surgical operation cuts down on the number of broken appointments. Since a broken appointment for cardiac catheterization ties up the diagnostic laboratory team usually for an entire day, it can be very expensive and wasteful. The public health nurse can also make arrangements for transportation to and from the diagnostic center. She can explain to the parents exactly what is going to be done to the child and can be most helpful in relieving their fear of the procedure. Another valuable service the public health nurse can provide is the gathering of blood donors for patients who are to have open heart surgical procedures.

Genetic counseling is coming into increasingly greater importance in the care of children with congenital heart disease. In a substantial number of cases cardiac lesions are being found to be genetically determined; and whether or not the parents of the child with mongolism, for example, should have more children is a matter that can be decided only by genetic study of the mongoloid child and of the parents to determine the exact chromosome abnormality. I am sure that proper genetic counseling will soon become a community service in many areas and will be available to physicians for the counseling of private patients.

Since many children with rheumatic and congenital heart disease require special school counseling and later vocational guidance because they will be unable to do strenuous jobs, they should be directed toward more sedentary occupations early in life. If this objective is to be met, close liaison between private physicians and school counseling and vocational guidance leaders is imperative.

Many states now have rheumatic fever secondary prevention programs which supply prophylactic medication free of charge or at cost to patients whose physicians have prescribed long-term prophylaxis against recurrent rheumatic fever. A weakness in all of these programs, however, has been the inability to keep children from neglecting prophylaxis after a time. It is our experience that a year after the drug is prescribed, fewer than a third of the patients are still taking it unless follow-up visits have been made to the home. The follow-up can be done by Heart Association volunteer workers who are properly trained in the importance of prophylaxis and are given appropriate literature; or, better still, it can be done by professional public health nurses who go into the home at regular intervals and discuss with the mother the necessity of prophylaxis and make sure the child continues medication as instructed by his physician.

Primary prevention has not yet reached the stage where it should have widespread application in all communities, but certainly anyone who has ever had so much as a questionable history of rheumatic fever should have culture studies done any time he has a sore throat. If Group A hemolytic streptococcus is found, the patient should be treated with 10 full days of penicillin therapy. The establishment of local or regional facilities for this culturing, whereby the private physician can have prompt reports on the results, has been another area of community activity during the past few years.

Because many patients with active rheumatic carditis require long periods of bed rest, and since most authorities agree that this is best carried out in the patient's own home (unless in seriously deprived circumstances), facilities for home-care and follow-up of acute rheumatic fever cases must be available in the community. Again, the public health nurse is an excellent source of help. Home-bound teachers also render a necessary service since the child will be away from school for a period of three weeks to one year and it is important that he not fall far behind in his classwork. When home-bound teachers are not a part of the community resources, we have found it possible to locate retired teachers in the community who in almost every instance are only too happy to give their services without pay to provide tutoring for these children. The local Heart Association is a good group to call upon for assistance in finding a tutor when such services are needed.

Hypertension

A recent survey of a Georgia county, involving a random sample of the entire county population with better than 96 per cent participation, has shown that 17 per cent of the population over 17 years of age, excluding those age 65 and over, have significant diastolic hypertension (using the World Health Organization criteria of 165/95 mm of mercury). Follow-up has shown that in many of these persons the pressure returned to normal on repeated visits, the abnormality having been due merely to excitement of the survey. Yet there are perhaps 10 per cent who do have significant degrees of hypertension. The interesting facts are that only one-third of these patients were aware that they had hypertension, and that of those who did know, less than one-third were receiving any medical therapy for high blood pressure. Since excellent control of this disease can be achieved with drugs, there is every reason to think it both practical and timely to begin community case-finding in this field in an attempt to get under treatment all persons in the community with significant hypertension.

We are experimenting with the feasibility of routine home visits by the public health nurse who takes the patient's blood pressure and reports it to the physician as an aid to him in managing the case. Preliminary results indicate that this approach is most successful and probably should be a very important addition to our armamentarium against hypertension.

Stroke

The recent National Stroke Congress focused attention on the problem of stroke in our communities and reemphasized the importance of rehabilitation of patients after stroke. It has been found that many patients with stroke, once they are discharged from the hospital, discontinue all physical therapy and regress very soon into a state of inactivity and bed rest. This, of course, leads to the complications which are so feared in stroke—contracture, arthritis of the affected joints, decalcification of the bone, resulting kidney stones, decubitus ulcer and other complications caused by neglect.

Harder to measure, but equally important, are the effects on the morale of the patient and his family. If, when the patient leaves the hospital, there can be continued follow-up at home by public health or other home nursing services or similar community facility, with regular physical therapy consultation and regular exercises given at least twice a day by members of the family, we have found that recovery from stroke is rapid and in many cases amazing. Eighty-five per cent of the patients who live to leave the hospital can be restored to self-care in the activities of daily living, thereby releasing family members to return to work. Moreover, 15 per cent of the patients themselves can be returned to gainful employment of some kind. Almost fantastic is the change in spirit and attitude of both patient and family when a positive approach is made to stroke rehabilitation rather than acceptance of defeat and "tender loving neglect."

Coronary Heart Disease

Recent epidemiologic studies of populations have identified some of the major factors which lead to proneness to coronary heart disease in individuals. These are identified as hypertension or diabetes, cigarette smoking, sedentary occupations and lack of recreational exercise, a high fat diet and elevated serum lipid levels. Personality type has also been implicated by many. Apparently it is possible to modify significant numbers of the "coronary proneness" factors, and preliminary studies in prevention programs have made it clear that successful modification of these factors reduces the risk of coronary heart disease by at least 25 per cent. Since this is a major health problem today, we feel that a widespread community education program concerning the coronary proneness factors should be undertaken and that private physicians should make every effort to modify these risk factors when they are found in routine physical examinations.

Another important recent advance in the field of coronary heart disease has been the installation of intensive care units for persons with heart disease in our acute care hospitals. This shows promise of significantly modifying the risk of death from a heart attack while the patient is in the hospital, and we urge all physicians to investigate the use of such a unit in their own hospitals. Associated with this, but also important in other respects, is cardiopulmonary resuscitation and the teaching of resuscitation techniques to hospital personnel and other persons, such as electrical linemen, policemen, firemen and ambulance drivers, who are likely to be confronted with situations in which these techniques are needed. Cooperation of the physicians in the community in teaching proper techniques of cardiopulmonary resuscitation is necessary if we are to avoid accidents by improper application of these potentially dangerous techniques.

Congestive Heart Failure

A common end result of all types of heart disease is congestive heart failure, and although we now have drugs that make management of this disease easier than heretofore, recent studies in community programs aimed at controlling congestive heart failure have demonstrated the value of regular home health nursing visits to follow-up patients with congestive heart failure. The Dade County, Florida, experience in which the number of patient days in the hospital was reduced from 904 in the control group to 542 in the treatment group, and the Durham County, North Carolina, study in which the number of hospitalizations in the control group was 26 and in the follow-up group four, point up the importance of good follow-up of patients in congestive failure. During her visits with the patient, the nurse not only can weigh the patient and watch for increasing signs of congestive failure, but she can also evaluate results of diuretic therapy and watch for signs and symptoms of digitalis intoxication, hyponatremia and the like which she can report to the physician. With this service, the number of visits to the physician's office can be greatly reduced and acute pulmonary edema is less likely to develop.

An Exercise Program for Shoulder Disability

DAVID RUBIN, M.D., *Los Angeles*

■ *In disabling conditions of the shoulder the involvement is either primary or secondary in origin. The primary disabilities are usually caused by sudden direct trauma or repeated minor traumata. Bursitis, tendonitis, and minor tears of the rotator cuff may result. The disabilities which arise secondarily are more difficult to treat and may have prolonged sequelae. Cervical radiculitis, infectious mononeuritis, cardiac disease, and cerebrovascular disease are common sources for secondary shoulder involvement. Pain and limitation of motion with loss of function are the presenting symptoms.*

The selection of a proper program of therapy is determined by the diagnosis. Primary conditions are responsive to the usual physical measures of moist heat, ultrasound, and the common exercises. Injections of local anesthetics or steroids and use of deep x-ray therapy may also be effective. The disabilities which arise secondarily are more difficult to treat and often become chronic and intolerably painful. In the author's experience, the most effective treatment for the secondarily induced shoulder problem is the combined use of moist heat of mild intensity, manual mobilization, neuromuscular facilitation, and a suitable home program of appropriate exercise.

SHOULDER DISABILITY with pain and loss of function is a common problem in the practice of medicine, especially in general practice, orthopedic medicine, industrial medicine and physical medicine. Unless the disability arises by direct and severe trauma sufficient to induce a fracture or dislocation, the resultant pain and loss of function occurs either by involvement of the soft tissues in and around the shoulder or by secondary extension from adjacent anatomic regions. Whether the cause is primary or secondary, pain and loss of function are the complaints that bring the patient to the physician.

History-taking and examination, both clinical and radiologic, may or may not provide a positive diagnosis, and in most instances the laboratory tests are of little value. Among the conditions often considered are bursitis, tendonitis, rotator cuff tear, pericapsulitis and "frozen shoulder," and it is not uncommon for a patient to receive two or three different diagnoses from different physicians.

Primary Disabilities

Primary shoulder disability resulting from repeated traumata with ordinary wear-and-tear activity results in the development of pain with an in-

---

Presented before a Joint Meeting of the Sections on Physical Medicine and Industrial Medicine and Surgery at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: 10921 Wilshire Boulevard, Los Angeles 90024.

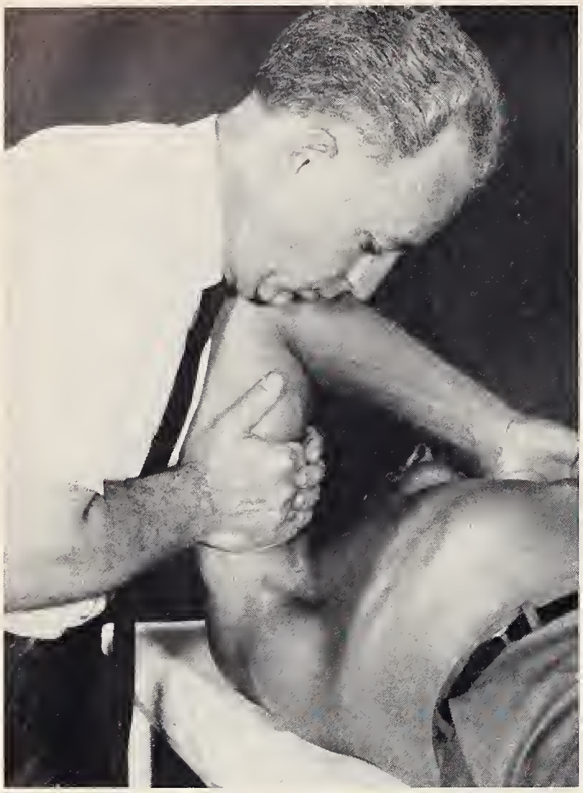


Figure 1A.—Lateral distraction.

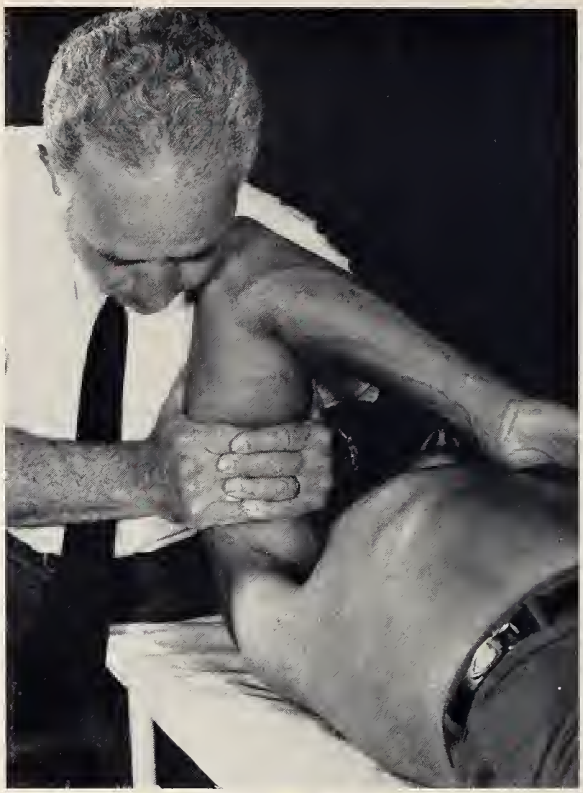


Figure 1B.—Superior distraction.

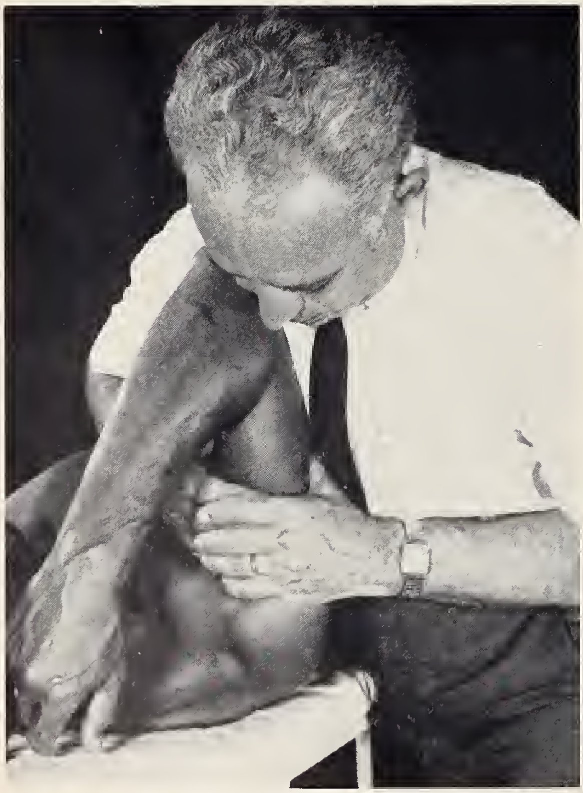


Figure 1C.—Inferior distraction.

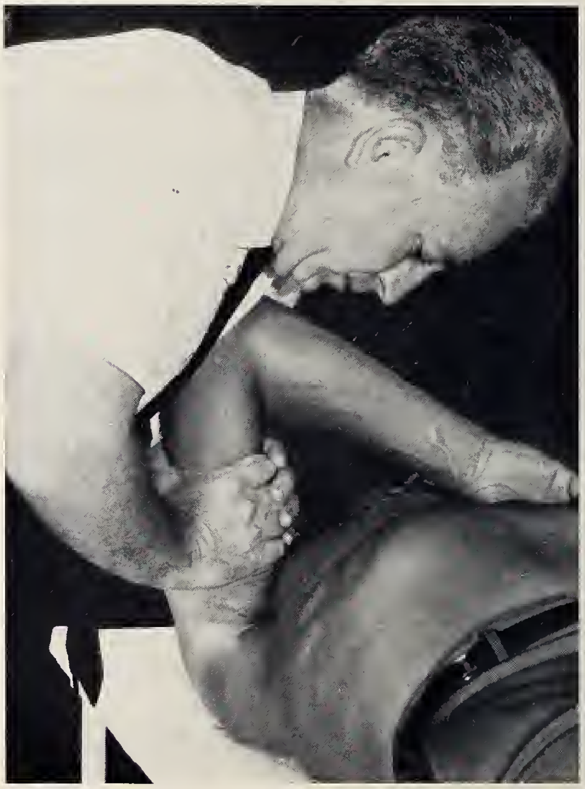


Figure 1D.—Posterior distraction.

sidious loss of shoulder motion. In other instances direct trauma may lead to more severe pain and a more rapid loss of function. In such cases a definitive diagnosis may determine the most effective and shortest therapeutic approach. The injection of steroids into localized areas of tenderness and pain, such as the subdeltoid bursa or the supraspinatus tendon, may eliminate the problem easily. Early application of heat or cold, ultrasound and simple exercises (Codman[1]) may be rapidly effective in relieving the pain and restoring normal motion. In more resistant cases, x-ray therapy may also be useful in alleviating inflammation and reducing pain, thus enabling the patient to recover a more normal although frequently incomplete range of motion.

Secondary Disabilities

Shoulder disability resulting from other than direct trauma or disease is more difficult to treat and may have prolonged sequelae. Common causes of secondary involvement of the shoulder are cervical radiculitis, brachial plexus stretch injuries (thoracic outlet syndrome), cardiac disease, cerebrovascular disease, infectious mononeuritis, and post-inoculation mononeuritis. In the first two categories cited, shoulder pain and loss of mobility may arise either with or without motor weakness of the shoulder musculature.

The shoulder disabilities which are secondarily induced are frequently misdiagnosed and unsuccessfully treated, and often they become chronic, intolerably painful and a source of great irritation to the patient. Sleep is frequently disturbed and job efficiency may be decidedly impaired. Routine treatment, including injections, various forms of heat, ultrasound, the usual exercises and medication, is usually attempted but often does not either relieve the pain or restore the lost motion. It is not unusual for such therapy to increase rather than decrease the disability, leaving the patient frustrated and psychologically depressed.

An Exercise Program

Passive Exercise

Passive exercise consists of gentle mobilization and manipulation of the shoulder to stretch and relax the capsule and adjacent muscles. The maneuver may be painful, but usually for only a short period. The movements are performed by placing

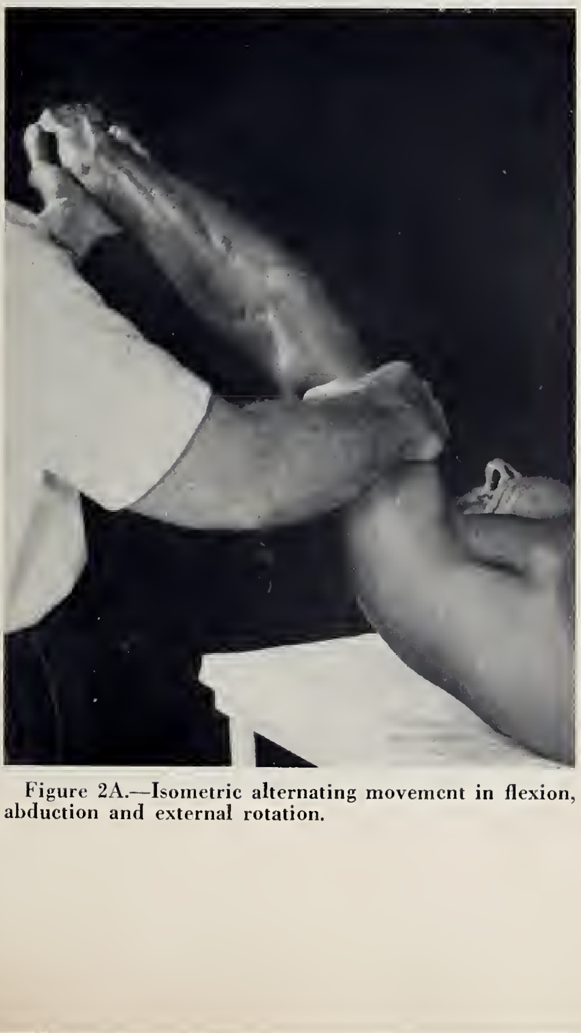


Figure 2A.—Isometric alternating movement in flexion, abduction and external rotation.

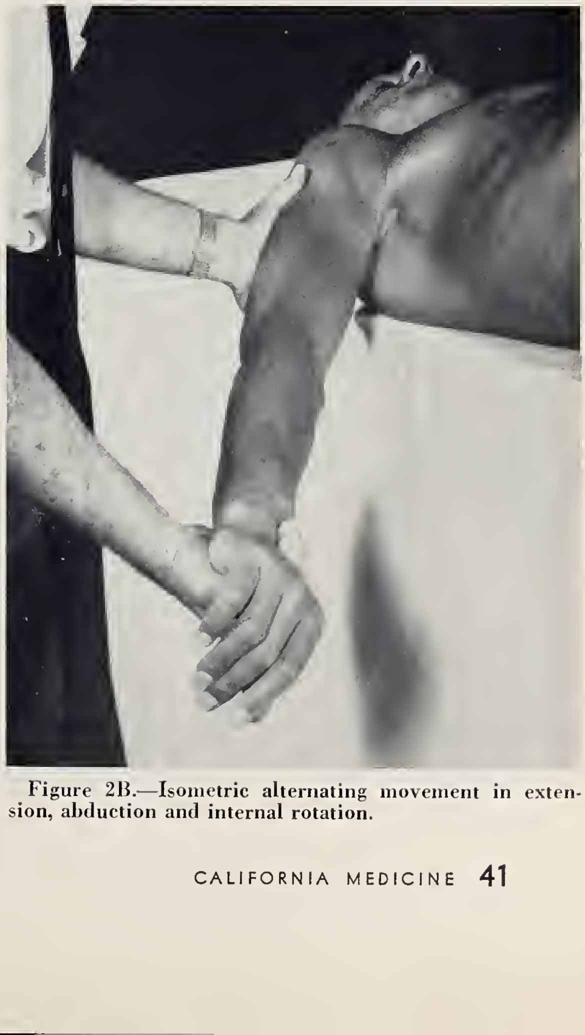


Figure 2B.—Isometric alternating movement in extension, abduction and internal rotation.

the patient on his back and stretching the head of the humerus away from the glenoid fossa.[3] The movements are gentle but firm, and pain of short duration may occur as tightening and gentle distraction are applied. Four distinct manipulative (distraction) movements are attempted—namely, lateral, superior, inferior, and posterior (Figure 1A, B, C and D).

Manual Resistive Exercise and Neuromuscular Facilitation Techniques

Following passive mobilization and gentle manipulation, the use of isometric, manually-applied resistive exercises in the normal patterns of motion for the upper extremity (Figure 2) will often result in a remarkable increase in painless range of motion. When these exercises are properly carried out, the patient has minimal pain during the exercise and is therefore able to cooperate in alternately contracting and relaxing the agonist and antagonist muscles about the shoulder joint. Spiral patterns of motion are utilized to increase first the forward flexion-abduction-external rotation pattern (Figure 2A), and later the extension-abduction-internal rotation pattern (Figure 2B). These are skillfully applied techniques which facilitate a slow, progres-

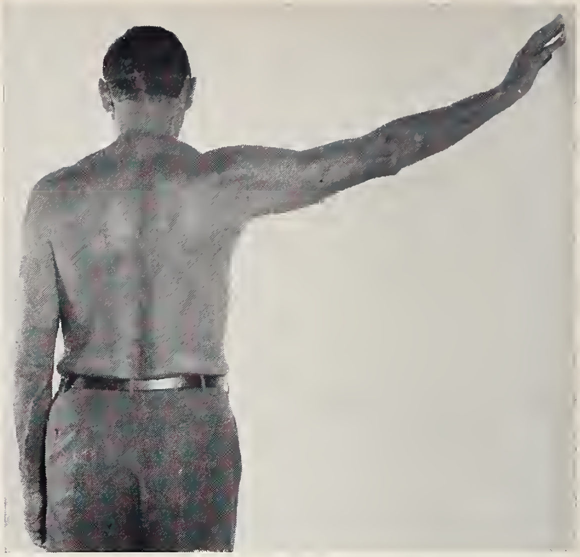


Figure 3B.—Abduction, wall climbing.

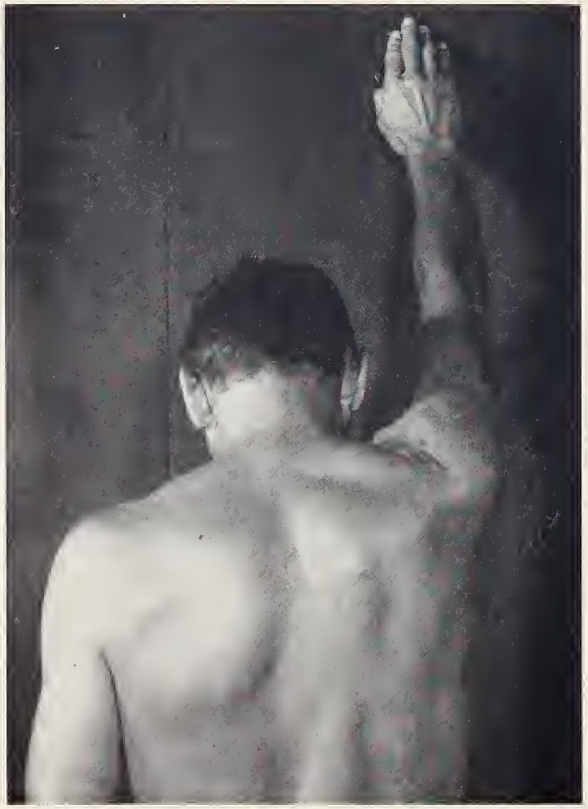


Figure 3A.—Forward flexion, wall climbing.

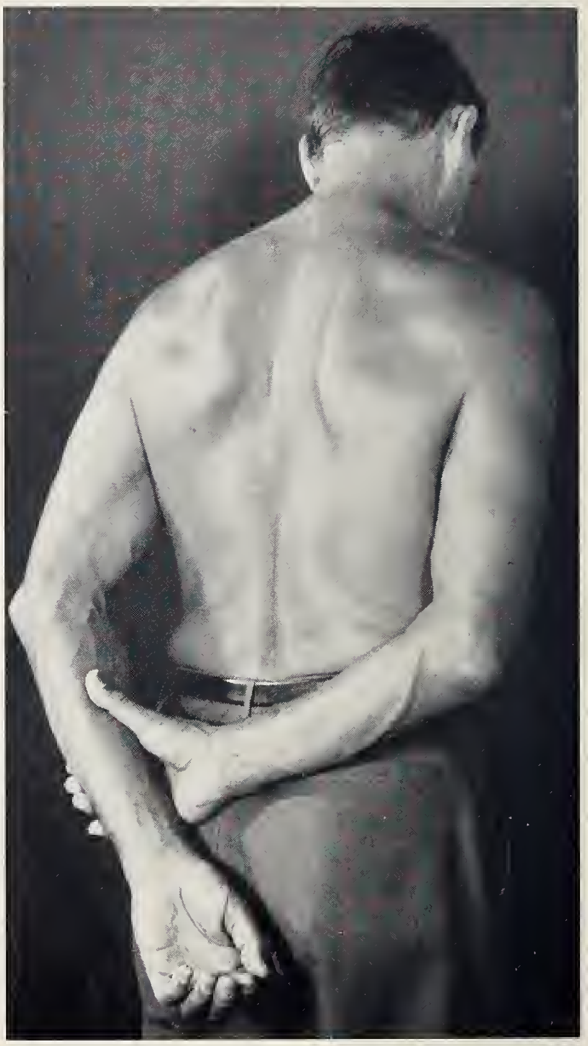


Figure 3C.—Internal rotation, abduction maneuver.

sive relaxation of the tight muscular and ligamentous structures and restore an increased and painless (or less painful) range of motion.[2]

The prior application of moist heat and ultrasound to the involved shoulder may relax the soft-tissues and promote general relaxation which will facilitate passive mobilization, manipulation and resistive exercise. As the range of motion increases and the pain diminishes, shoulder wheel exercises, which serve to increase isotonic strength and psychologically stimulate the patient to greater voluntary effort, may be quite helpful.

Home Exercises

The formal treatment program of passive mobilization, manipulation and manual resistive exercise two or three times a week is supplemented by a series of home exercises which are performed three to four times daily. These are, at first, forward flexion and gentle abduction, with the hand on the affected side placed on a wall for support to minimize pain. The patient is instructed to face the wall and, with the extremity outstretched and the elbow straight, to "climb" the wall with his fingers. He slowly moves toward the wall as the degree of forward flexion increases (Figure 3A). This is repeated several times. He is then instructed to stand at right angles to the wall and to repeat the maneuver with the extremity in abduction (Figure 3B). In this case, he side-steps toward the wall as the degree of abduction increases. Mild to moderate pain must be felt at the shoulder as it is stretched; otherwise the effective range of motion will not be increased.

If the shoulder disability has resulted from radiculitis of cervical origin, the patient must be instructed to keep his head flexed slightly while performing these exercises, otherwise he may have recurrent radicular pain.

Rotation exercises are also a part of the home program but they are not begun until forward flexion and abduction have improved and are relatively painless in a useful range of motion. At the appropriate time, instruction in rotation exercises is outlined and demonstrated. First, the patient is asked to place the uninvolved extremity behind his back and to grasp the wrist of that extremity with the hand of the involved extremity (Figure 3C). Then he slowly slides the hand toward the elbow of the unaffected extremity until moderate pain in the involved shoulder limits further movement. This is repeated three to five times several times daily.

By use of these three steps—passive exercise (mobilization and manipulation), manual resistive exercise and a home exercise program—in a definite time sequence, a majority of patients can be helped over a period of time (varying from one to four months) to overcome painful and functionally limiting shoulder disabilities. However, unless the patient is adequately instructed and has full knowledge of the nature of the problem, the course to be expected and the physical activities to be avoided, the treatment may fall short of the desired goal—namely, restoration of complete and painless motion and function. Shoulder disabilities which are secondarily induced are extremely challenging to the patient and to the physician. They require a very precise, carefully supervised and meticulously administered program for a good result.

REFERENCES

1. Codman, E. A.: The Shoulder, Thomas Todd Co., Boston, 1934.

2. Kabat, Herman; Neuromuscular Reeducation for Paralysis, Chap. XXII in Physical Medicine in General Practice, 3rd Ed., Bierman, W., and Licht, S., Paul B. Hoeber, Inc., New York, 1952.

3. Mennell, John McM.: Joint Pain, Little, Brown and Company, Boston, 1964.

Unsuspected Carcinoma Found Histologically After Minor Anorectal Operations

LEWIS GRODSKY, M.D., *San Francisco*

■ *The unsatisfactory results of radical operations in advanced anal cancer could be bettered by earlier diagnosis of the malignant lesion. The possibility of cancer should always be kept in mind when treating any of the common chronic anal diseases. Preliminary biopsy studies should be done more frequently if suspicious lesions are present and all tissues removed during minor anorectal operations should always be examined by a pathologist. Each surgical specimen should be labeled to show the site from which it was removed. Follow-up care of the patient would be simplified if the exact site of cancer origin could be identified.*

A recent ten-year survey of minor anorectal surgical procedures at the University of California Medical Center showed clinically unsuspected anal cancer found in 2 per cent of cases on tissue examination by the pathologist. The cancers were generally early and of the keratinizing squamous cell and nonkeratinizing varieties.

ANAL CANCER is relatively uncommon; it accounts for less than 1 per cent of all the malignant lesions that afflict the body. The prognosis is still unsatisfactory for the patient with an advanced anal cancer, despite radical surgical excision including extended lymphadenectomy. The results would be improved if diagnosis could be made earlier, before the malignant anal lesion becomes invasive. Often the lesion is not recognized promptly, possibly because of the infrequency of anal cancer, but usually because early lesions are masked by the more prominent, common, benign anorectal disease.

Clinically unsuspected malignant changes have been found postoperatively in specimens obtained at hemorrhoidectomy, anal fistulectomy and fissurectomy, and from excised anal condylomata. Early anal cancers will be found more frequently before they become disseminated if we keep in mind the possibility of cancer in all patients with long-standing chronic anal disease, if we routinely obtain preliminary biopsy studies of any suspicious area, and if we submit all excised anal tissues for pathologic examination to detect possible malignant changes.[1-18]

A recent study was made at the University of California Medical Center of all patients operated on for minor anorectal disease during the period 1954 to 1965 (Table 1). On pathologic examination of the surgical specimens, undiagnosed anal canal and perianal cancers were found in 2 per cent of these cases. The cancers were generally early growths and included squamous cell, columnar cell, and cloacogenic tumors. Bowen's and Paget's disease have also been found in postoperative tissues.[5-7] Other surveys have shown a similar incidence of cancer not detected until minor operation was done.[3,4,18]

Published reports have indicated an association between the common chronic benign anorectal diseases with attending infection, and the subsequent

---

From the Department of Surgery, University of California School of Medicine, San Francisco.

Presented before the Section on General Surgery at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: 2211 Post Street, San Francisco 94115.

TABLE 1.—*Unsuspected Anal Cancers Found After Minor Operation, University of California Medical Center, 1954-1964*

						Carcinoma, Varied Type	
	No. of Cases	*Squamous Cell*	*Columnar Cell*	*Cloacogenic Cancer*	*Basal Cell*	*Bowen's Disease*	*Paget's Disease*
Hemorrhoidectomy	526	4	1	3	0	1	1
Anal Fissurectomy	64	0	0	1	0	0	0
Anal Fistulectomy	63	1	0	1	0	0	0
Totals	653	5	1	5	0	1	1

anal cancer development of various types. This association has been noted in chronic infected anorectal fistulas and sinuses, lymphopathia venereum, condylomatas and other long-standing perianal and anal canal lesions.[2,10,12,13,15-17] Proof of this relationship cannot always be completely established.[1]

The following representative case reports illustrate some of the problems encountered in cases in which clinically unsuspected cancers were found by the pathologist after minor anorectal surgical procedures.

Reports of Cases

CASE 1.—A 42-year-old Negro woman had hemorrhoidectomy in March 1962 for chronic prolapsing hemorrhoids which had been causing increasing disability. An indurated component was present in the right anterior prolapsing hemorrhoid, but preliminary biopsy was not done. Radical ligature and excision hemorrhoidectomy was performed and the hemorrhoids were dissected to the anal sphincters. The pathologic diagnosis of the excised right anterior hemorrhoid was keratinizing squamous cell cancer with local invasion into the subcutaneous tissues (Figure 1). The right anterior wound bed was then revised in width and depth, and the subjacent anal sphincter muscles were excised. Pathologic examination of the new specimen showed no residual malignant changes. No recurrence was noted on continued observation of the wound site. (Last examined in December 1966.)

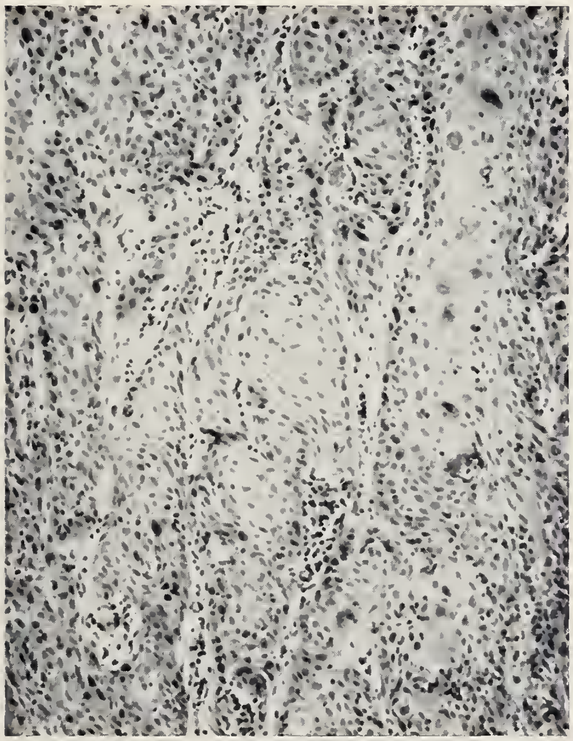


Figure 1.—(Case 1) Keratinizing squamous cell cancer with epithelial pearl formation found in an excised hemorrhoid (×300).

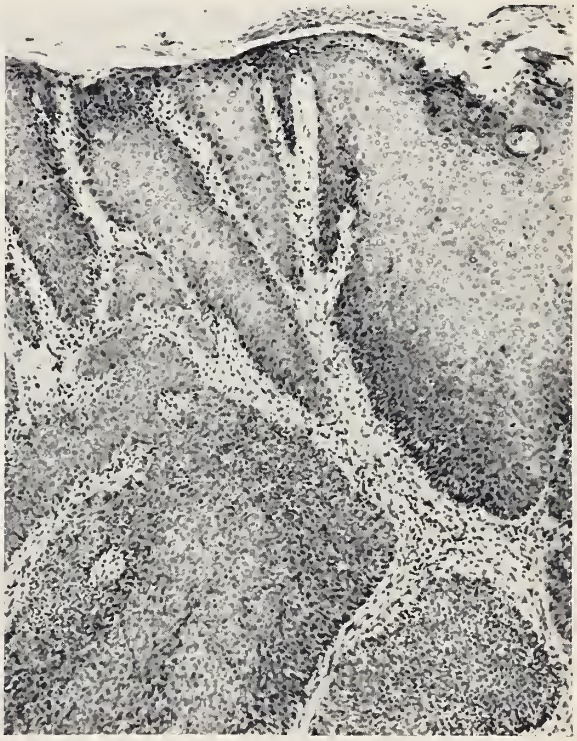


Figure 2.—(Case 2) Excised hemorrhoid showing leukoplakic changes and squamous cell anaplasia with dermal invasion (×125).

Case 2.—A 53-year-old white woman had chronic prolapsing bleeding hemorrhoids for more than six years. Proctologic examination in November 1962 showed advanced hemorrhoidal disease with diffuse leukoplakic surface changes. A preliminary biopsy was not done. Ligature and excision hemorrhoidectomy was performed. Three unmarked surgical specimens showing leukoplakic surface changes were given to the pathologist for examination. There was a single area of epidermal squamous cell anaplasia with a poorly defined basal cell layer and dermal invasion in one of the specimens (Figure 2). The exact site of cancer origin could not be determined. A consultation was held and examination of the fresh wound sites showed normal postoperative tissue reaction without any gross evidence of malignant change. Inguinal nodes were palpable in both groins. The original anal wounds were then widely extended, leaving only narrow anterior and posterior anorectal bridges. The palpable inguinal nodes were removed for examination and all excised tissues were tagged for identification. One of the excised lateral segments showed residual cancer *in situ.* The lymph nodes showed only nonspecific hyperplasia without cancer. The patient was examined regularly thereafter and there was no evidence of recurrence. (Last examined in December 1966.)

Case 3.—A 54-year-old woman had a St. Mark's ligature and excision hemorrhoidectomy in May 1964. The preliminary proctologic examination had been reported to be negative except for advanced hemorrhoidal disease. Postoperative examination of the tissues showed an area of intraepidermal junctional cloacogenic cancer of transitional cell type in one of the three surgical specimens (Figure 3). The site of origin of the cancer could not be determined because the specimens had not been tagged. After consultation it was decided to keep the patient under observation rather than to revise all of the wounds. There has been no evidence of recurrence of cancer up to this time (December 1966).

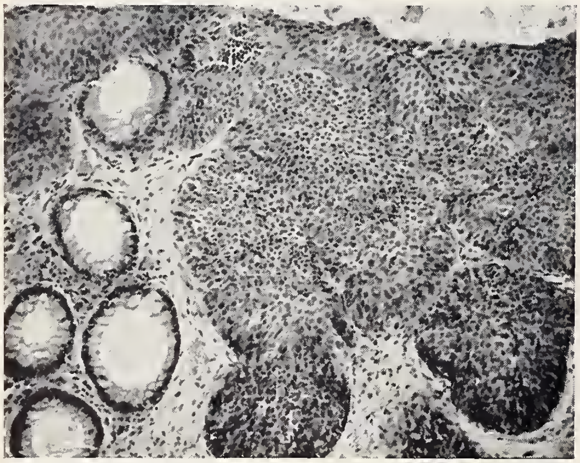


Figure 3.—(Case 3) Early intraepidermal junction cloacogenic transitional cell cancer discovered after hemorrhoidectomy (×125).

Case 4.—An 83-year-old woman was admitted to hospital in January 1965 because of severe recurrent anorectal bleeding and pronounced anemia. The proctologic diagnosis was chronic advanced hemorrhoidal disease and rectal mucosal prolapse. Previous sclerotherapy had been ineffective in controlling the bleeding. Radical hemorrhoidectomy was decided against because of the patient's age, and the pronounced deficiency of the perineal body. The more active bleeding points were treated by transfixation ligatures, and the prolapsing rectum was put back into place. A redundant segment of anal skin that had a superficial ulcerated area 0.3 cm in diameter was then excised. The pathologic diagnosis was basal cell cancer of the anal skin (Figure 4). The excised skin margins were free from tumor cells. The patient has

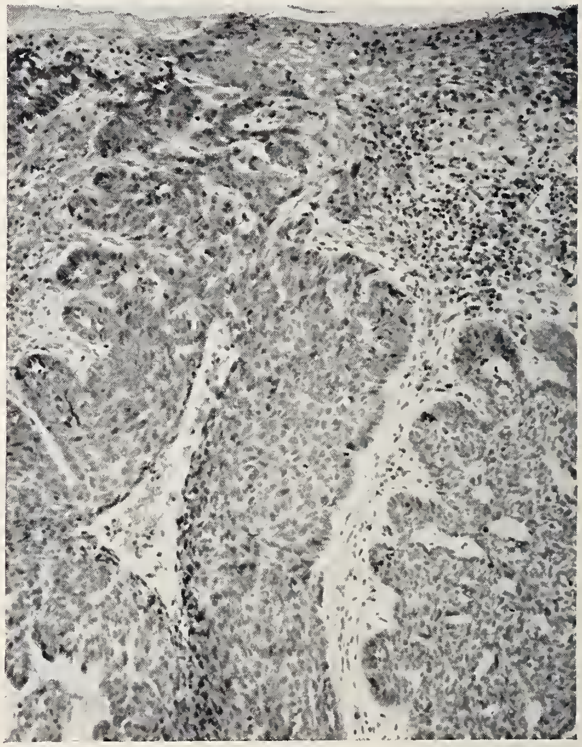


Figure 4.—(Case 4) Excised perianal skin segment showing ulceration and basal cell cancer (×300).

remained symptom-free. (Last examined in December 1966.)

CASE 5.—A 40-year-old white man was seen by a proctologist in December 1964 because of diffuse multiple condyloma accuminata involving the posterior third of the anal orifice. He had been treated surgically by another proctologist six months previously for anal warts in the same area. The warts had recurred quickly. Oral treatment with bismuth was poorly tolerated and did not control the condylomata. There was no mention of malignant changes in the report of the pathologic examination of the original surgical specimens.

The second procedure consisted of excision of the entire posterior warty anal mass. The pathologic diagnosis of the specimen was condyloma accuminata with an area of focal intraepithelial squamous cell cancer (Figure 5). Regular observation of the wound site is being continued for both condylomas and possible recurrence of cancer. (Last examined in December 1966.)

CASE 6.—A 47-year-old woman had recurrent anal fistula formations for more than five years and an anal fissure eventually developed. An excisional fistula-fissure operation was done in February 1960. A 4×7 cm segment of tissue was submitted for pathologic examination. The diagnosis was squamous cell cancer with focal invasion and extension into the lymph nodes. The patient was referred to the University of California Medical Center, where the original pathologic diagnosis was confirmed. In April 1960 an abdominoperineal resection was done. No residual squamous cell anal cancer or lymph node invasion was demonstrated. The patient has remained cancer free. (Last examined in December 1966.)

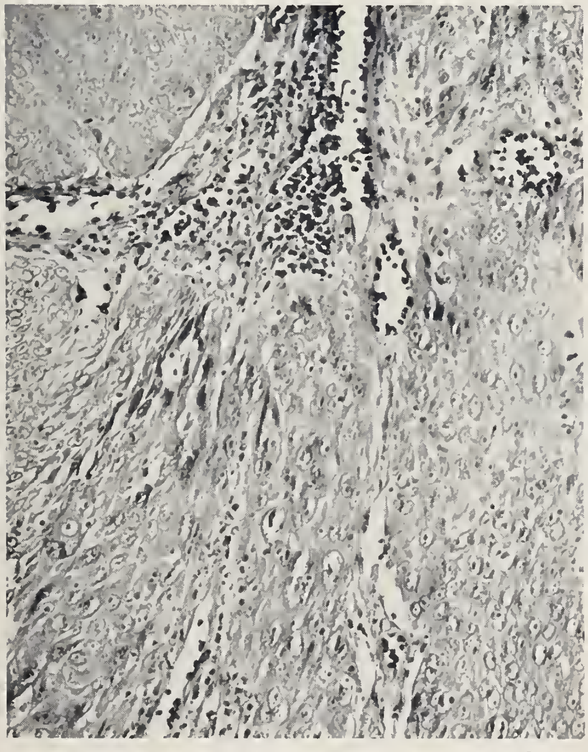


Figure 5.—(Case 5) Intraepidermal squamous cell cancer in anal condylomata (×300).

CASE 7.—A 48-year-old white man was seen in August 1961, 10 days after a posterior anal fissure had been excised. Routine pathologic examination of the excised tissue showed cloacogenic transitional cell cancer. Subsequent proctologic examination showed a normal appearing postoperative anorectal wound site. After consultation an abdominoperineal resection was done. Pathologic examination of the entire surgical specimen was negative for residual cancer except for a metastatic cloacogenic transitional cell cancer in a single mesorectal lymph node. There had been no recurrence to the time of most recent examination (December 1966).

Discussion

The possibility of malignant changes developing during the course of chronic benign anal disease, or of the presence of an early cancer being masked by the more conspicuous benign anal disease, should never be overlooked. Kuehn and coworkers[11] reviewed 157 cases of epidermoid cancer occurring in the anal canal or perianal region. The anal cancer had remained undetected in 37 cases for over six months. In 10 of the patients the diagnosis of anal cancer was not established until minor anorectal operations were done. Gordon[3] reviewed 1,890 surgical procedures done for hemorrhoids, fistula in ano, fissure in ano and other minor anorectal diseases. Microscopic examination of the postoperative tissues revealed previously unrecognized cancers in 1.1 per cent.[3] Tucker and Hellwig, in an analysis of 951 proctologic cases found clinically unrecognized cancers in 1.9 per cent.[18] Other observers[2,5-17] have also reported unsuspected anal cancers found by the pathologist after minor anorectal operations.

In a current ten-year survey of a total of 653 minor anorectal surgical cases at the University of California Medical Center, it was noted that 13 patients (2 per cent) had clinically unsuspected anal cancer. These lesions were discovered on pathologic examination after operation for a common, seemingly benign anal disease. In the representative cases reported herein, postoperative microscopic examination also showed previously undiagnosed anal cancers in supposedly benign anorectal diseases. Cases 1, 2 and 6 illustrate the need for preliminary biopsy studies of all suspicious areas before any anal operation is performed. Cases 2 and 3 point up the importance of tagging postoperative tissue specimens individually, as an aid to current treatment and also for adequate follow-up care.

The relationship between chronic anorectal disease and cancer has not been completely documented. Buckwalter and Jurayj,[1] who made a statistical study of the subject, concluded that chronic benign long-standing anorectal disease, when associated with chronic infection, plays a definite role in the genesis of epidermoid cancer in some cases. Other investigators have also emphasized the possibility of cancer developing in long-standing untreated fistula in ano, in the chronic strictures of lymphopathia venereum, in condylomata and in other chronic anal diseases.[2,10,12,13,15-17] Early, complete extirpation of any chronic irritative anorectal disease would appear to be the most effective means of preventing later malignant changes. Anorectal fistulectomy is therefore a more complete and safer procedure than simple fistulotomy.

REFERENCES

1. Buckwalter, J. A., and Jurayj, M. N.: Relationship of chronic anorectal disease to carcinoma, Arch. Surg., 75:352-361. 1957.

2. Friedberg, M. J., and Serbin, O.: Condyloma acuminatum: Its association with malignancy, Dis. Colon Rectum, 6:352-355, 1963.

3. Gordon, B. S.: Unsuspected lesions in anal tissue removed for minor conditions, Arch. Surg., 73:741-746, 1956.

4. Grinnell, R. S.: An analysis of 49 cases of squamous carcinoma of the anus, Surg. Gynec. Obst., 98:29-30, 1954.

5. Grodsky, L.: Bowen's disease of the anal region (squamous cell carcinoma in situ). Report of three cases, Amer. J. Surg., 88:710-713, 1954.

6. Grodsky, L.: Intraepidermal cancer of the anus; evolution to invasive growth, Calif. Med., 87:412-415, 1957.

7. Grodsky, L.: Extramammary Paget's disease of the perianal region, Dis. Colon Rectum, 3:502-509, 1960.

8. Grodsky, L.: Cloacogenic cancer of the anorectal junction. Report of seven cases, Dis. Colon Rectum, 6:37-44, 1963.

9. Grodsky, L.: Uncommon nonkeratinizing cancers of the anal canal and perianal region, New York State J. Med., 65:894-901, 1965.

10. Kline, R. J., Spencer, R. J., and Harrison, E. G.: Carcinoma associated with fistula in ano, Arch. Surg., 89: 989-994, 1964.

11. Kuehn, P., Beckett, R., Eisenberg, E., and Ree, J.: Epidermoid carcinoma of the perianal skin and anal canal, New Engl. J. Med., 270:614-617, 1964.

12. Levin, I., Romano, S., Steinberg, M., and Welsh, R. A.: Lymphogranuloma venereum: Recital stricture and carcinoma, Dis. Colon Rectum, 7:129-134, 1964.

13. McNally, A. R., and Dockerty, M. B.: Carcinoma developing in chronic draining cutaneous sinuses and fistulas, Surg. Gyn. Obstet., 88:87-96, 1949.

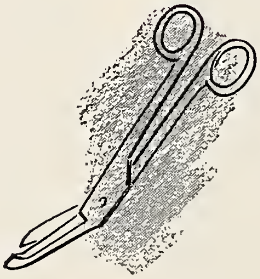
14. Schilla, F. W.: Basal cell carcinoma of the anal area, Dis. Colon Rectum, 2:210-215, 1959.

15. Schwartz, A.: Anorectal fistula and cancer, Dis. Colon Rectum, 6:305-307, 1963.

16. Skir, I.: Mucinous carcinoma associated with fistulas of long-standing, Amer. J. Surg., 75:285-289, 1948.

17. Stockman, J. M., and Young, V. T.: Carcinoma associated with anorectal fistula, Amer. J. Surg., 86:560-561, 1953.

18. Tucker, C. C., and Hellwig, C. A.: Proctologic tumors, J.A.M.A., 111:1270-1273, 1 October 1938.



The Problems of LSD^{25} and Emotional Disorder

J. THOMAS UNGERLEIDER, M.D., AND DUKE D. FISHER, M.D., *Los Angeles*

OVER THE PAST YEAR in the United States a new problem has arisen—that of abuse of LSD (lysergic acid diethylamide). The problem seemed to develop rather suddenly in the late summer of 1966 as increasing numbers of persons began to arrive at psychiatric clinics and medical emergency rooms throughout the country with symptoms following LSD ingestion. This occurred at about the same time that the mass media were publicizing LSD—unfortunately often in a seductive, alluring way—as a panacea for man's problems. Most of the major magazines, newspapers and television networks have featured one or another aspect of LSD, including, for example, its alleged use in helping architects build better buildings and enhancing creativity in art and music. It has been publicized as an answer to a variety of sexual problems and problems of living in general, as well as a revolutionizer in the treatment of mental illness.

Then, as the dangers of this drug became recognized, first in Southern California and in New York City, and then throughout the country, particularly on college campuses, a wave of hysteria began to sweep the nation. Federal laws passed early in 1965 making possession of LSD for sale or manufacture illegal (The Drug Abuse Control Amendments of 1965) did not stem the tide. The one legal manufacturer of the drug, Sandoz Laboratories, stopped the production of LSD in May of 1966, and all Sandoz-sponsored grants for research on this drug were cancelled.

This atmosphere of hysteria continues to pervade the entire LSD problem. Literature has appeared which blames LSD on the Communists and everyone seems to have his own theory on what should be done. In October of 1966, California (along with two other states) made even the possession of this drug illegal. In the meantime, much research into the LSD problems and potentials is being discontinued. Little is really known yet about LSD. This article will attempt to present some of the things that are known, and point up the many areas of uncertainty with particular emphasis on the psychological ramifications connected with the use of LSD.

Historical Factors

Lysergic acid diethylamide tartrate (LSD^{25}*) was synthesized in 1938 by a Swiss scientist named Hoffman. He recognized its perception-altering properties in 1943. Lysergic acid, the precursor of LSD is a constituent of ergot, a fungus that grows on rye. The drug is related to psilocybin, the active alkaloid of the Mexican mushroom, and to mescaline which is found in the peyote cactus buttons, except that it is many times stronger than these "hallucinogens." Morning Glory seeds also contain LSD-like compounds, although in milder form.

Man has used drugs which change the state of consciousness for thousands of years. Cannabis (Indian hemp) was brought to Europe in 1500 B.C. from Asia, and to the United States in 1920 as marijuana. Psilocybin and peyote were used by the Aztec Indians in Mexico centuries ago. Now we live in a drug age; it is an age of flagrant drug abuse. Thus perhaps it was only natural, once the word spread about LSD bringing the fountain of youth, for our culture to embrace this drug. Aldous Huxley first described its joys in his *Doors of Perception* in 1954.

From the Department of Psychiatry, UCLA Center for the Health Sciences, 760 Westwood Plaza, Los Angeles.

Assistant Professor, Department of Psychiatry, UCLA Medical Center (Ungerleider); Psychiatric Resident, Department of Psychiatry, UCLA Medical Center (Fisher).

Reprint requests to: Department of Psychiatry, UCLA Center for the Health Sciences, 760 Westwood Plaza, Los Angeles (Dr. Ungerleider).

*Commonly known as LSD, or by users as acid or L.

The use of LSD was first mainly limited to the upper middle classes, professionals and intellectuals, but gradually its usage has spread from colleges to high schools to junior high schools, and now to lower income groups. This should not surprise us when we have seventy million users of alcohol with five million alcoholics, and when thirteen billion doses of amphetamines and barbiturates are manufactured per year in the United States. The LSD problem has now also spread to England, Holland and France.

The term *psychedelic,* or "mind-manifesting," was coined by Osmond in 1957. To describe LSD as hallucinogenic is not always accurate, although widespread, because persons who see or hear things following LSD ingestion usually perceive *actual* objects or sounds as accentuated and/or distorted. These are then illusions, like a mirage, rather than true hallucinations, although sometimes true hallucinations do occur.

At first these perceptual changes caused by LSD were hailed as offering an experimental way to mimic schizophrenia and thus to study it. The "model psychosis" theory of LSD was thus conceived.[16,17] This theory died gradually as it was realized that LSD psychosis did not indeed mimic that of schizophrenia. Not only were there such dissimilar factors as the absence of so cardinal a symptom of schizophrenia as autism, but even the nature of the "hallucinations" was fundamentally different.

Physiologic Effects

Physiologically, LSD has few effects. It is readily absorbed from the intestinal tract. Thus there is little advantage in intravenous injection of the drug, although occasionally a user will try injecting it for a change.

There is usually a loss of appetite, although some studies have reported an increase in appetite following ingestion. Nausea, dizziness, headache and palpitations are often experienced, and periods of shivering alternate with heat flushes. The pupils are dilated and LSD users often wear sun-glasses, even at night, to combat photophobia. Heart rate and both systolic and diastolic blood pressures rise moderately, as does blood sugar. A fine tremor of the fingers and hands may be present. Recently grand mal seizures were observed in a previously non-epileptic person following ingestion of LSD.[6]

The drug is not physiologically addicting in that there are no withdrawal symptoms following discontinuation. However, it is psychically addicting in that after taking it the user frequently becomes convinced that he wants to keep taking it. In fact, LSD users, in contrast to hard narcotics addicts, develop a remarkable missionary or proselyting quality.

Laboratory techniques are not sensitive enough to determine the LSD concentration in body fluids and tissues, so C_{14}-labeled LSD has been used in animals. The highest concentration is found in the liver and the kidneys. The highest concentration in the brain is found in the hippocampus, basal ganglia, thalamic nuclei and cerebral cortex.[8] Behavior changes occur after the brain concentration of LSD is decidedly decreased. Most of LSD's metabolites are excreted in the feces.[12]

Tolerance to LSD develops rapidly; thus users rarely regularly take LSD more than twice weekly. There is no lethal dosage known in humans. One direct fatality was an elephant that died from an overdose when he was given 100 *milligrams* of LSD per kilogram of body weight in an experimental situation.*[5] Monkeys die after receiving 5 mg per kilogram of body weight.

Psychologic Effects

Sensations become intensified and perceptions are distorted under LSD. One man slept on the floor the night he took LSD because he was sure his bed was only two inches long. Illusory phenomena are common. Another man was restrained from diving off a cliff onto the rocks and the ocean below. Later he explained that he felt the breaking waves were a silk scarf and he wanted to dive into it. Faces often appear to be melting. One high school student cut all the flexor tendons in her wrist when she looked in the mirror and saw her face begin to dissolve. Time sense is especially distorted. We have seen persons under the influence of LSD stare at their fingers or at a leaf for hours. True hallucinations, predominantly visual, also occur.

Delusions are not infrequent. We treated, in crisis intervention, a young man who became convinced, a few hours after ingesting LSD for the first time, that he had to offer a human sacrifice, that is, kill someone, or die himself. He was prevented from throwing his girl friend off the roof of a Hollywood hotel. Additional comments on psychologic effects will be made later in a discussion of side effects.

---

*Also reported in Cashman, John: *The LSD Story,* Fawcett Publications, Greenwich, Conn., 1966, pp. 50-51.

Mode and Site of Action

Neither the mode nor the site of action of LSD is definitely known. It is even unknown whether the various perceptual distortions—"hallucinations" and the like—can be attributed to a primary action of LSD or whether these different effects involve different mechanisms and sites of action. The perceptual dysfunction itself, unassociated with impairment of skilled motor performance, presumably originates at a high level in the nervous system. The hallucinations, intensified with sensory deprivation, also seem to be of central origin, and not an enhancement of responses to peripheral stimuli.

Frontal lobe ablation in chimpanzees does not abolish a previously induced panic reaction in these animals.[1] Implanted cortical electrodes in humans following LSD have revealed paroxysmal electrical activity in the hippocampal gyri, amygdaloid nuclei and septum which parallel the perceptual changes.[11]

LSD is a strong antagonist to serotonin both in vitro and in vivo. One hypothesis for the action of LSD is that it antagonizes serotonin in the central nervous system, in which it is found especially in the basal ganglia. Another theory, based on the observation that most of the LSD has left the brain before the mental effects are seen, is that LSD acts as a trigger or is changed into a metabolite which in turn causes the mental changes. LSD also affects various enzyme systems within the body. Inhibition of cholinesterase and monamine oxidase in spinal cord neurons has been demonstrated in animals following LSD administration.

Animal Experiments

Dogs and cats exhibit behavioral changes, like pawing the air, which suggest they may be hallucinating. They become confused in the performance of various tests. Pigeons become catatonic. Ants attack their nestmates. Spiders spin disorganized webs. Body tempterature increases, particularly in rabbits. Large doses of LSD cause complete blindness in cats and monkeys. As was mentioned previously, very large doses kill monkeys.

Three Areas of Research

There have been three main approaches in the work on LSD to date. The first has to do with the controlled administration of a known amount of LSD experimentally, following the screening of subjects by psychiatric interviews and/or psychological testing. Resultant subjective and/or objective effects of the drug are then recorded in this "experimental" study of LSD.[5,7] There are a number of experimental studies about personality and performance changes under LSD and, particularly with work on LSD, the "set" of the researcher (his attitude toward LSD) may become apparent. For example, in one experiment raters were used who worked gratis, who may have taken LSD themselves, and who were instructed to rate patients as improved if they were "more flexible" and demonstrated less "unrealistic rigidity."[13] One must be careful and critical in evaluating such studies.

The evaluation of creativity under LSD is extremely difficult. If an artist paints a picture while on LSD it may be rejected by the public and also by art critics and yet it could conceivably be a creative work. Perhaps this reservation also applies when the artist himself (as was once our experience) later terms his artistic effort under LSD "just chicken scratches." But certainly many persons do *feel* that they are more creative under LSD. Subjective feelings of improvement concomitant with objective decrease in performance will be dealt with further under a discussion of side effects later in this article.

The second type of approach with LSD has to do with its therapeutic effectiveness. It has been used for alcoholics to help them stop drinking.[3] It has been used in patients dying of cancer to alleviate the pain and help them die a more "dignified" death.[9] It has been used as a psychotherapeutic aid in general to increase insight and lift repressions,[10] and with autistic children to increase socialization.[14] More extensive claims have been made for LSD, and it has been cited as a specific cure for frigidity, impotence and homosexuality.

It is difficult, but essential, to evaluate just what therapeutic efficacy LSD does possess. Extravagant claims and total disclaimers now vie in an atmosphere of hysteria. Several points are noteworthy. For an experiment to be valid, it should be possible to reproduce the results. This has not yet been convincingly done in the therapeutic approach to LSD, in our opinion.

In addition, the attitude of the researcher is vital. We have spoken to researchers, themselves LSD users, who become so enthusiastic that they even refused to consider psychosis and suicide as bad results. Particularly with LSD work, as with hypnosis, one's attitude and expectations are vital factors in the results one achieves.

And finally, it is essential that there be only one variable at a time. In one experiment for example, to find that the recidivism rate among psilocybin-treated prisoners was significantly less than among those not treated sounded encouraging until further scrutiny revealed that the treatment group received many other advantages. These included close relationships with the investigators, a special pre-parole course of instruction, special assistance in obtaining housing and employment and follow-up contact from the researchers. It would be hard to say that it was the LSD alone that helped.

The third approach to LSD has to do with observation of the side effects which occur following the ingestion of the drug. There are both acute and chronic side effects, and their occurrence *cannot* be predicted. Psychiatric interviews and psychological testing do not screen out adverse reactors. Some of the worst reactions have been in persons, often physicians and other professionals, who appeared stable by every indicator. Conversely, others who have had past histories of severe psychiatric problems and have been leading marginal existences have seemed to tolerate LSD without ill effect.

There is some work to show that persons who place a premium on self control, planning, caution and impulse restriction, and who sacrifice spontaneity, do particularly poorly on LSD.[2]

Acute Side Effects

Four major types of acute symptoms have been seen. These include, in decreasing frequency, hallucinations (both auditory and visual), anxiety to the point of panic, severe depression with suicidal thoughts or attempts and confusion. These symptoms may occur in patients who have taken the drug once or 60 times. They occur in persons who have only taken LSD as well as in persons who are chronic multiple drug abusers.

At the Psychiatric Emergency Room and Admitting Office of the Neuropsychiatric Institute at the University of California, Los Angeles (UCLA), Medical Center before September of 1965, approximately one problem case associated with LSD ingestion was seen every other month. In the seven months from September 1965 to 1 April 1966, the incidence of "LSD cases" increased to between five and 15 a month and made up 12 per cent of all cases dealt with by the psychiatric emergency service.[18] The 70 "LSD patients" seen in the seven-month period (and in increasing numbers despite the Federal Drug Abuse Control Amendments, effective 1 February 1966) had several characteristics. The group was predominantly single, white, male and young (average age, 21). The members came from throughout the Los Angeles area and most were either unemployed or students. Twenty-eight (40 per cent) of the group had taken drugs other than LSD within six weeks of being seen in the emergency room. Twenty-five (36 per cent) had a history of chronic marijuana use, but 28 (40 per cent) had never taken drugs other than LSD. Our sample contained no professional persons, in contrast to the findings of others that "LSD seems mainly to be used by professionals, intellectuals or middle-class people."[2]

Treatment of the acute symptoms first must be directed toward preventing the patient from physically harming himself or others. Thus the frequent indication for psychiatric hospitalization. Twenty-five of the 70 patients in our study were admitted to hospital. It is important for the therapist not to increase the patient's anxiety by being anxious himself. One patient panicked when he called a center for crisis intervention and was told by the therapist on call that he had "just caused permanent irreversible brain damage" to himself by taking LSD.

Chemical agents are also a vital part of the treatment regime. Chlorpromazine (Thorazine®) is the most effective antagonist to LSD's effects.[4] This is seen clinically by reversal of the hallucinations and can also be demonstrated by reversibility of the electroencephalographic changes.[11] This antagonistic effect of chlorpromazine can also be demonstrated in animal experiments.[15] In contrast, reserpine may actually enhance the effect of LSD.[11] Barbiturates may have some antagonistic effects to LSD, as may azacyclonal (Frenquel®). It should be emphasized however that chlorpromazine is not always effective in treatment of LSD complications. For example, a 22-year-old, single, white man became psychotic approximately 24 hours after the ingestion of LSD. He had both auditory and visual hallucinations. He had used LSD once before without difficulty. Several parenteral doses of 50 mg of chlorpromazine, plus orally administered chlorpromazine up to 2,000 mg per day, and chlorpromazine in conjunction with trifluoperazine hydrochloride (Stelazine®), resulted in no reduction or improvement of the psychosis. The patient improved slowly after a period of six weeks; the improvement was seemingly unrelated to the phenothiazine medication. It is important to note that recurrences of the acute side effects from LSD in all their original intensity often appear up to a year after the ingestion, without further ingestion of the drug. This is regardless of set or setting, which we will elaborate upon further. The efficacy of inducing these side effects in chemical warfare, via contamination of the water supply or as inhalants in the air, is, as far as we know, unproven to date.

Chronic Side Effects

Certain chronic changes have been noted among LSD users. While the authors' initial observations were drawn from patients who had been put in hospital, we have also had the opportunity to observe large numbers of persons in the community who have taken LSD.[19] Many had had bad experiences but had not seen a physician or gone to a hospital. These persons in the community who had the chronic LSD reactions in varying degrees, varied from white collar workers and professional people to unemployed "beatniks," construction workers and longshoremen.

We have observed the ingestion of LSD by individuals and by groups of from two to three to 50 or 60 persons. We have seen it taken indoors and also in scenic surroundings, at "kick-type" Hollywood parties and at quiet religious gatherings, and from Orange County to San Francisco.

An effect of LSD that has been noticed quite frequently and is particularly striking to us is a dramatic shift in one's value systems. Many persons after using LSD are no longer interested in working or playing what they call the "ego games" of society.* LSD users often leave their families and become quite withdrawn, devoting most of their time to thinking, writing and talking about LSD. They take quite literally Dr. Timothy Leary's admonition to "turn on, tune in, and drop out." We met one man at an LSD party who had spent the previous two years wandering around the desert with a pack on his back, contemplating the experiences of LSD. Three years before, he had been an international lawyer in New York City. We have talked to students who, after taking LSD, became much less interested in their academic work and preferred to spend their time "thinking kind thoughts." We have seen formerly productive persons who have adopted the attitude that one should live merely for subjective introspective experiences and not play the various "games," like work, that society demands. Since many people who experience this change of attitude are never seen by psychiatrists, one can only speculate as to their frequency.

Another chronic difficulty with LSD is what we would term "perceptual distortion." This refers to a subjective feeling of improvement concomitant with an objective loss of functioning. For example, a band leader phoned us because his drummer was producing such terrible music. He was so out of tune and rhythm that patrons were unable to dance to the band's music. Nevertheless when we interviewed the drummer, he told us that he felt he was playing "like Gene Krupa," and was more than satisfied with his music. A law student told us that LSD had opened such horizons that he felt his legal studies were dull and boring by comparison. He speculated at length (while lying in his room during and between LSD trips) about the advisability of giving the world's leaders LSD so that they would love, not hate or make war. A group of LSD users whom we studied fairly intensively over a period of several months were convinced that they could "pick up vibrations" from other people, and that they could determine if someone else had used LSD merely by casual inspection. When these convictions were put to a test, these persons were completely unable to discriminate as to who had used LSD or what someone else was thinking. In fact we found that they appeared to have actually suffered a loss of their ability to discriminate and to observe.

Unfortunately, the idea of LSD seems to have particular attraction for the adolescent. Most adolescents are struggling with feelings of aggression and sexuality, along with the need to establish an identity, and many of them see LSD as offering a "magic solution" for these struggles. LSD provides an intense introspective experience to the adolescent, and enables him to deny feelings of aggression and sexuality at the same time that it gives him "membership" in the group of LSD users with a common language and dress. But many times the adolescent is so overwhelmed by the LSD experience that his search for identity becomes a florid, psychotic nightmare. Similarly, the attempt to use LSD is a way of denying sexuality and aggression, which robs the adolescent of his chance to work out his conflicts in regard to these instinctual drives in a healthy way. There is probably no other period in our lives more loaded with conflict than adolescence. Yet, it is persons of this very age group that, by virtue of their struggles, are the most attracted to the magic promises of LSD. This is one of the truly great dangers of the drug.

---

*Dr. Timothy Leary called ego games "The tribal game . . . mechanized, computerized, socialized, intellectualized, televised, sanforized" [Playboy Magazine, September 1966, p. 106]. In other words, conformity, work and organization as well as materialism, are all called "ego games" by LSD users.

The criteria for mental health, according to Freud, are the ability to work and to love, and LSD users seem to have special difficulties in both of these areas. We have already mentioned the numerous persons we have observed who have lost their incentive to work after using LSD. The ability to love, that is to have psychic intimacy with another person, seems also to be decreased by LSD. In contradiction to the claims that the drug helps one to get closer to other people, we have noticed that users become more introspective and invested in themselves. Several LSD sessions that we have attended were filled with excited individuals proclaiming their feeling of being especially close to various other people in the room. Nevertheless, we were impressed with the number of monologues that were taking place at the sessions. Very few of the participants were at all interested in or relating to others. On the other hand, they seemed to be enjoying their highly introspective experience, the extreme results of which are autism and psychosis.

LSD also seems to provide more primitive defenses against the normal anxiety and depression that most of us face. We have noticed many LSD users who develop a more primitive way of handling their feelings. While many of us get angry, anxious, depressed or even withdrawn when we encounter periods of stress, the LSD user often hallucinates, becomes paranoid or perceives people as caricatures. As was previously mentioned, users frequently experience their psychotic or other symptoms in their original intensity as much as a year after using the drug and without taking the drug again. Thus, many people who have a great deal of difficulty tolerating the anxiety and stress of everyday living anyway are provided, in a sense, with a psychotic defense by LSD. They experience an estrangement from reality with the drug.

Another disturbing aspect of the use of LSD is the missionary quality that develops in many of its users. Many LSD users are so affected by the drug that it becomes impossible for them to be objective when discussing its effects.[20] Unfortunately this lack of objectivity has extended to some researchers in the field, researchers who also are LSD users. There is a great deal of proselyting and insistence on the part of users that other persons must share the same kind of subjective awareness that they experience. We have no doubt that this is a sincere conviction on the part of many LSD users, since in our research we have seen mothers who have given LSD to their infants, brothers who encourage their sisters to take LSD, and individuals who have taken their life's savings and purchased LSD in order to distribute it gratis to complete strangers. Despite all these chronic personality and behavioral changes, organic brain damage has not yet been demonstrated in humans.

The "Trip" Itself

Users of LSD call themselves "acid heads," and the LSD experience itself is a "trip." (The signs on cars which read "CONSULT YOUR LOCAL TRAVEL AGENT" refer to the LSD trip.) Many users prefer to have their experience out of doors, particularly by the ocean or a lake, or in the mountains or woods, or in the desert. They usually ingest what has been sold to them as anywhere from 100 to 250 micrograms of LSD, although the amount and purity of the substance is questionable because of its black-market source. It is available at anywhere from 50 cents to 10 dollars. Atropine-like compounds have been substituted for LSD. We recently had one capsule, purchased by a user who paid for 250 micrograms, analyzed. It was found to contain approximately 100 micrograms of LSD.

Users usually like to hear music (particularly played by performers like Ravi Shankar) and to see intense colors (witness the rash of "light shows" and the use of strobe lights) while they have their LSD experiencc. They often read, especially from the Tibetan Book of the Dead, although often silently. Many chronic users have developed "aids" to help them reverse any bad effects—often reassuring phrases from various psychedelic books. (We have seen a number of users whose aids did not help.) One hundred to 250 micrograms is the usual dosage for an LSD experience although a number of users claim to have ingested 2,000 micrograms daily for a period of weeks.

A great deal has been written about "set" and setting. The set or attitude with which one approaches the LSD experience is extremely important, as with hypnosis. In fact, people are now taking trips *without* drugs.[21] However, set does not completely determine the type of "trip." For, other experiments have recorded subjects who expected psychotic reactions from LSD but experienced only pleasant feelings of relaxation.[2] And we have seen persons who appeared to take the drug under what they felt were ideal conditions, subsequently have a very severe adverse experience.

The setting in which one takes LSD is likewise a very important variable. Persons given LSD without their knowledge are extremely liable to become panicky and have a severe reaction. There are several such incidents recorded and one alleged suicide following a party where the punch had been "spiked with LSD. However, it is not usual for LSD to be secretly placed in punch or any other liquid. The user wants people to take LSD, but to know that they are taking it. We have seen persons who have taken LSD in what they consider an ideal setting (with one or two good friends, with a guide or "sitter," with soft music playing, and in a relaxed environment) who still had bad experiences.

Epilogue

Until more is known about the short- and long-term effects of LSD as well as how to predict who will have a bad experience, it must be considered a very dangerous drug. Unfortunately, the ready market for and the easy manufacture of the drug, have resulted in an almost unlimited black market supply. This colorless, odorless, tasteless liquid will be practically impossible to regulate by law alone. There is no test yet developed to detect the drug inside the body. It can be concealed on the back of an envelope or soaked into a coat, and one can take a trip merely by licking the envelope flap or sucking on the coat lapel. The LSD users are often the very people who have the most to lose from its use. We certainly hope that LSD research will resume, but we appeal to those who loudly proclaim that "everyone" should take LSD to consider the highly subjective and often untoward response to the drug. We have frequently seen the most ardent enthusiast become the most eloquent opponent after just one bad trip.

Man's search for Eldorado continues. LSD will surely become obsolete as other more potent "psychedelics" are developed. Already a compound "DMT" (dimethyltryptamine) is available in limited amount in the black market, and there are now "sophisticated" users who scoff at something as weak and unappealing as LSD.

Generic and Trade Names of Drugs

Chlorpromazine—*Thorazine.*
Azacyclonal—*Frenquel.*
Trifluoperazine hydrochloride—*Stelazine.*

REFERENCES

1. Baldwin, M., Lewis, S. A., and Bach, S. A.: The effects of lysergic acid diethylamide after cerebral ablation, Neurology, 9:469-474, 1959.

2. Blum, R., (Ed): Utopiates: The Use and Users of LSD,^{25} Atherton Press, New York, 1964.

3. Chwelos, N., Blewett, D. B., Smish, C. W., and Hoffer, A.: Use of LSD in the treatment of alcoholism, Quart. J. Stud. Alc., 20:577-590, 1959.

4. Clark, L. D., and Bliss, E. L.: Psychopharmacological studies of lysergic acid diethylamide (LSD^{25}) intoxication, A.M.A. Arch. Neurol. and Psychiat., 78: 653-655, 1957.

5. Cohen, S.: The Beyond Within, Atherton Press, New York, 1964.

6. Fisher, D., and Ungerleider, J. T., Grand Mal seizures after LSD ingestion, in preparation.

7. Hollister, L. E., and Hartman, A. M.: Mescaline, LSD and Psilocybin: Comparison of clinical syndromes, effects on color perception and biochemical measures, Comp. Psychiat., 3:235-241, August 1962.

8. Jacobsen, Erik: The clinical pharmacology of the hallucinogens, Clin. Pharmacol. Ther., 4:480-503, July-August 1963.

9. Kast, E.: Pain and LSD^{25}: A theory of attenuation of anticipation, from LSD, the Consciousness-Expanding Drug, Edited by D. Solomon, Putnam Sons, N.Y., 1964, p. 241.

10. Lewis, D. J., and Sloane, R. B.: Therapy with LSD, J. Clin. Exp. Psychopath., 19:19-27, 1958.

11. Monroe, R. R., Heath, R. C., Mickle, W. A., and Llewellyn, R. C.: Correlation of rhinencephalic electrograms with behavior: A study on humans under the influence of LSD and Mescaline, Electroencephalog. Clin. Neurophysiol., 9:623-642, 1957.

12. Rinkel, M.: Pharmacodynamics of LSD and Mescaline, J. Nerv. and Ment. Dis., 125:424-427, July 1957.

13. Savage, C., Fadiman, J., Mogar, R., and Allen, Mary H.: The effects of LSD therapy on values, personality and behavior, Int. J. of Neuropsychiat., 2:241-254, June 1966.

14. Simmons, J. Q. III, Leiken, S. J., Lovas, O. S., Shaeffer, B., and Perloff, B.: Modification of autistic behavior with LSD, A. J. Psychiat., 122:1202-1211, May 1966.

15. Sturtevant, F. M., and Drill, V. A.: Effects of mescaline in laboratory animals and influence of ataraxics on mescaline response, Proc. Soc. Exper. Biol. and Med., 92:383-387, 1956.

16. Tolan, E. J., and Lingl, F. A.: Model Psychosis produced by inhalation of gasoline fumes, Amer. J. Psychiat., 120:757-761, February 1964.

17. Unger, S. W.: Mescaline, LSD, Psylocybin and personality change, Psychiat., 26:111-125, May 1963.

18. Ungerleider, J. T., Fisher, D., and Fuller, M.: The dangers of LSD, J.A.M.A., 197:109-112, August 1966.

19. Ungerleider, J. T., and Fisher, D: LSD: Research and Joyride, The Nation, 16 May 1966.

20. Ungerleider, J. T., and Fisher, D.: LSD: Fact and fantasy, Arts & Architecture, 83:18-20, December 1966.

21. The Psychedelic Game: Mademoiselle, March 1966, p.179.

CASE REPORTS

Facial Paralysis Occurring With Hypothyroidism

A Report of Two Cases

JERRY M. EARLL, M.D., AND
FELIX O. KOLB, M.D., *San Francisco*

Neurologic complications of myxedema range from simple headache to convulsion, coma and death. This subject was recently reviewed by Sanders,[6] who noted that almost 80 per cent of myxedematous patients complain of paresthesias. The most frequently impaired cranial nerve is the auditory component of the eighth nerve, and 15 to 31 per cent of myxedematous patients have diminished auditory acuity.[2,4] The vestibular component of the eighth nerve has been known to be involved,[1] and vertigo[4] and tinnitus[2-4] may occur. Although hoarseness and dysarthria are commonly noted, it is postulated that local myxedematous infiltration of the vocal cords and tongue, rather than recurrent laryngeal nerve paralysis, is the mechanism. Familial nerve deafness and goiter have been described, and trigeminal neuralgia has been observed in hypothyroid patients.[4,6,8] Bell's palsy, associated with hypothyroidism, was mentioned in one patient and no similar association had been previously noted.[6] Two patients whose acquired hypothyroidism was complicated by facial paralysis are described in this report.

Reports of Cases

Case 1.—A 59-year-old Caucasian man noted sudden paralysis of the left side of the face in August 1965. Earlier employment had resulted in a high degree of acoustic trauma, and audiograms in July 1965 confirmed a bilateral high tone loss of 60 per cent. There was pronounced impairment of speech discrimination. Vestibular function was intact. When the paralysis did not improve, the patient was admitted to hospital for decompression of the facial nerve in October 1965.

The patient had classic myxedematous facies, low voice, dull wit, a pulse rate of 60 beats per minute and blood pressure of 130/88 mm of mercury. The skin was dry and facial features were coarse enough to suggest acromegaly. There was pitting edema in the lower extremities, and deep tendon reflexes could not be obtained. Complete left facial nerve paralysis of peripheral type was present. Electrical stimulation brought no muscular response. The remainder of the examination was within normal limits. Ability to taste was not tested.

Results of laboratory studies included hemoglobin 12.6 gm per 100 ml, hematocrit 33 per cent and leukocytes 8,950 per cu mm—44 per cent neutrophils, 44 per cent lymphocytes, 4 per cent eosinophils, 2 per cent basophils, and 6 per cent monocytes. Urinalysis showed specific gravity of 1.019 and a 2 plus protein content. Serum electrolytes were as follows: Sodium 137, chloride 93.5, carbon dioxide 27.3 and calcium 9.2 mEq per liter; phosphorus 3.5 mg per 100 ml. Alkaline phosphatase was 5.2 Shinowara-Jones-Rinehart (SJR) units, and creatinine 1.41 mg per 100 ml. The serum proteins were within normal range, cholesterol 401 mg per 100 ml and triglycerides 2,300 mg per 100 ml (normal 50 to 150). Butanol extractable iodine was 0.9 mcg per 100 ml (normal 2.3 to 5.2). Twenty-four-hour radioactive

---

From the Metabolic Research Unit, University of California Medical Center, San Francisco.

Submitted 30 August 1966.

Reprint requests to: Metabolic Research Unit, University of California School of Medicine, San Francisco 94122 (Dr. Earll).

iodine uptake was 4 per cent and no increase occurred after two days of thyroid stimulating hormone, 10 units a day. A Burdick photomotogram T ½ = 600 milliseconds at the ankle (normal 250 to 380). X-ray films showed borderline enlargement of the cardiac silhouette. The sella was normal in size, but thinning of the bone at the posterior inferior aspect of the sella with minor shaping of the clinoids was suggestive of increased intrasellar pressure. Views of the hands showed large bone structure with prominent tendon insertions. Heel pads measured 22 and 23 mm. Baseline 24-hour urinary steroids were low (17-hydroxycorticosteroids 3.3 mg and 17-ketosteroids 2.5 mg). These levels increased to 7.4 and 4.7 mg respectively with intravenous infusion of 25 units of corticotropin. Total gonadotropins were negative at 5 mouse units.

The patient was treated with 12.5 mcg of L-triiodothyronine daily and the amount was increased to 50 mcg daily within six days. Because the exact status of the pituitary-adrenal axis was not established, supplementary cortisol was administered for the facial nerve decompression, which was performed in November 1965. The proximal portion of the vertical part of the nerve was somewhat atrophic, but there appeared to be moderate ballooning of the nerve from its sheath at the stylomastoid foramen, indicating increased pressure. The manifestations of myxedema steadily regressed and when last observed the patient was euthyroid while taking 300 mg of thyroxin daily. There was definite improvement in the paralysis, with return of about 50 per cent function in six months. Auditory function improved only a little. The serum lipids decreased significantly, cholesterol to 230 mg and the triglycerides to 985 mg per 100 ml. Gonadotropins were positive at 5 and 80 mouse units, and the serum growth hormone was normal at less than 1 mμg per ml when measured by radioactive immunoassay.

Case 2.—A 23-year-old Caucasian woman noted the first symptoms of thyrotoxicosis during her first pregnancy. These symptoms progressed after delivery in February 1964, and when she consulted a private physician in April 1964, a diffuse toxic goiter was confirmed by laboratory studies. She was treated at first with 10 mg of methimazole every 8 hours, and later with 100 mg of propylthiouracil every 8 hours. The hypermetabolic symptoms rapidly subsided over a period of six weeks and she was referred to the thyroid clinic for further evaluation in July 1964. Right facial paralysis developed suddenly before she entered the clinic. The goiter had been enlarging slightly, and she had been feeling cold, depressed and fatigued for several weeks. Physical examination revealed complete right facial nerve paralysis of peripheral type. The thyroid gland was twice normal size, the pulse rate was 60 beats per minute, the skin was dry, the hands were cold and reflexes were definitely delayed. Taste acuity was not tested. The protein-bound iodine was 2.9 mcg per 100 ml (normal 3.5 to 8), Lawson kinemometry value was 310 milliseconds (normal 200 to 280) and radioactive iodine uptake was 11 per cent in 24 hours. The dose of propylthiouracil was reduced to 50 mg every 8 hours, and there was prompt improvement in symptoms of hypothyroidism, the goiter diminished in size, and the facial paralysis rapidly improved. There was no residual facial paralysis in October 1964 when the patient was last seen. She was being maintained on propylthiouracil. The pulse rate was 80 beats per minute, she appeared euthyroid and kinemometry revealed a normal value of 240 milliseconds.

Discussion

In these two patients classic unilateral Bell's palsy developed in association with acquired hypothyroidism. Both experienced the usual rather sudden onset of paralysis, although in Case 1 there was rather chronic and severe primary myxedema and in Case 2 a relatively acute iatrogenically-produced hypothyroid state. Prompt recognition and correction of the hypothyroid state in Case 2 was associated with rapid and complete recovery from the paralysis. In Case 1, the hypothyroid state was not recognized at first and the facial paralysis did not completely abate in spite of thyroid replacement and surgical decompression. It is unlikely that the therapeutic levels of cortisone briefly used in Case 1 played a significant role in the patient's improvement.

The anatomical course and confinement of the facial nerve, which make it subject to the usual Bell's palsy, could account for its vulnerability to myxedematous infiltration and swelling of soft-tissues (Figure 1). Increases in soft-tissue mass occur in both myxedema and acromegaly, and in both conditions paresthesias and neuropathic changes are frequently noted. The carpal tunnel syndrome has been reported in association with myxedema,[7] and in two cases reported by Purnell[5]

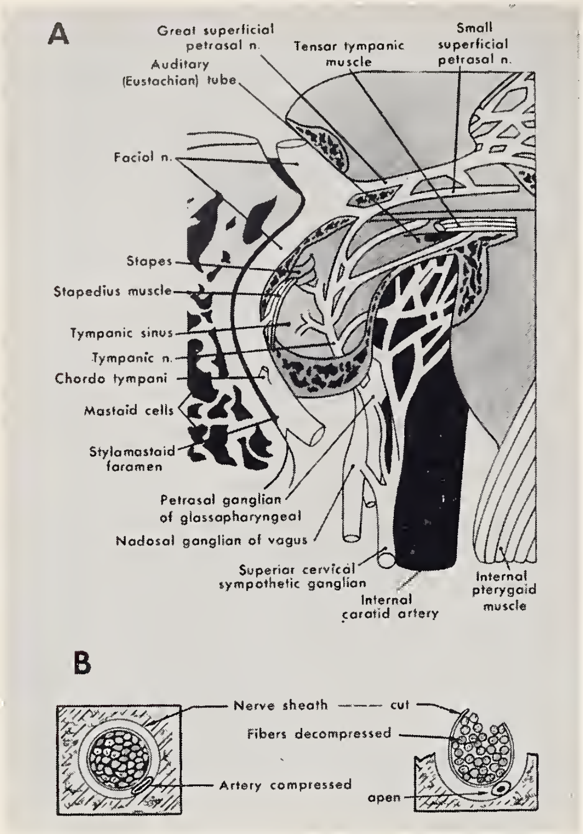

A
Great superficial petrosal n.
Tensor tympanic muscle
Small superficial petrosal n.
Auditory (Eustachian) tube
Facial n.
Stapes
Stapedius muscle
Tympanic sinus
Tympanic n.
Chorda tympani
Mastoid cells
Stylomastoid foramen
Petrosal ganglion of glossopharyngeal
Nodosal ganglion of vagus
Superior cervical sympathetic ganglion
Internal carotid artery
Internal pterygoid muscle
B
Nerve sheath
cut
Fibers decompressed
Artery compressed
open

Figure 1.—*A*, Right facial nerve within the skull. From Morris' Human Anatomy, B. J. Anson, copyright 1966, Blakiston Division, McGraw-Hill Book Co. Used by permission. Modified from the original. *B*, Schematic illustration of facial nerve before and after decompression (Case 1).

incision of the transverse carpal ligaments revealed an edematous, noninflammatory thickening of the flexor synovalis about the nerve. In Case 1 herein reported, the ballooning of the facial nerve from its sheath during surgical operations suggests a similar problem with edema and swelling.

Summary

Two hypothyroid patients in whom reversible facial paralysis developed are reported. This association has not been previously emphasized. The anatomical course and confinement of the facial nerve make its situation similar to the median nerve's confinement in the wrist; both may be vulnerable to the profound metabolic and soft-tissue changes of hypothyroidism. Prompt recognition and treatment of the associated hypothyroidism should offer the best chance for complete recovery.

REFERENCES

1. Barlow, R. A.: The study of vestibular nerve function in myxedema, Amer. J. Med. Sci., 164:401, 1922.

2. Bloomer, H. A., and Kyle, L. H.: Myxedema: A re-evaluation of clinical diagnosis based on eighty cases, Arch. Intern. Med., 104:234, 1959.

3. Hollender, A. R.: Hypometabolism in relation to ear, nose, and throat disorders, Arch. Otol., 63:135, 1956.

4. Nickel, S., and Frame, B.: Neurologic manifestations of myxedema, Neurology, 8:511, 1958.

5. Purnell, D. C., Daly, D. D., and Libscomb, P. R.: Carpal tunnel syndrome associated with myxedema, Arch. Intern. Med., 108:751, 1961.

6. Sanders, V.: Neurologic manifestations of myxedema, New Engl. J. Med., 266:547, 599, 1963.

7. Schiller, F., and Kolb, F. O.: Carpal tunnel syndrome in acromegaly, Neurology, 4:271, 1954.

8. Watts, F. B.: Atypical facial neuralgia in hypothyroid state, Ann. Intern. Med., 35:186, 1951.

Subcutaneous Fat Necrosis With Acute Hemorrhagic Pancreatitis

A Case in a Child with Steroid-Resistant Nephrosis Treated with 6-Mercaptopurine

EVA HEUSER, M.D., ELLIN LIEBERMAN, M.D.
GEORGE N. DONNELL, M.D., AND
BENJAMIN H. LANDING, M.D., *Los Angeles*

PANCREATITIS has frequently been described in steroid-treated patients who come to autopsy.[*] The histologic process is frequently mild and is an incidental finding, pancreatitis not having been manifested clinically.[2,4,12,15,22] The following case report illustrates the occurrence of clinically inapparent acute hemorrhagic pancreatitis in a six-year-old Negro boy with steroid-resistant nephrosis. The pancreatitis was associated with massive disseminated fat necrosis which extensively involved subcutaneous tissues. A final complication in this patient was generalized fungal infection.

Report of a Case

The patient, a six-year-old Negro boy, was admitted to Childrens Hospital of Los Angeles with

---

From the Departments of Pathology and Pediatrics, University of Southern California School of Medicine, and the Department of Pathology and Division of Renal-Metabolic Disease, Childrens Hospital of Los Angeles.

Submitted 16 August 1966.

Reprint requests to: Childrens Hospital of Los Angeles, 4614 Sunset Boulevard, Los Angeles 90027 (Dr. Heuser).

[*] Reference Nos. 2, 4, 12, 15, 22.

a diagnosis of nephrosis. He had been in good health until chicken pox developed three and a half weeks before admission. A few days later periorbital edema developed, followed by generalized swelling which disappeared without specific therapy. The patient then remained asymptomatic for one week but peripheral edema recurred and he was referred to Childrens Hospital.

On physical examination mild periorbital and pretibial edema were noted. No other abnormalities were observed. Blood pressure was 100/70 mm of mercury.

A hemogram was within normal range. The erythrocyte sedimentation rate was 117 mm in one hour (Westergren method). The specific gravity of the urine was 1.033 and there was a 4-plus reaction for protein. On microscopic examination there were 3 to 7 leukocytes, 5 to 10 erythrocytes and rare hyaline cases per high power field. Blood urea nitrogen was 10 mg per 100 ml, albumin 1.7 gm, globulin 2.9 gm and cholesterol 462 mg per 100 ml.

Treatment consisted of prednisone, 75 mg per square meter of body surface per day. Diuresis occurred and by the eleventh hospital day proteinuria had disappeared. The same dose of prednisone was then given four days a week but relapse occurred six weeks later. Treatment consisting of daily prednisone, 75 mg per square meter a day, spironolactone and chlorothiazide resulted in a clinical remission of five days' duration. The patient then gained 10 pounds in two days and he was thereafter in the hospital for eight and a half months. Daily administration of chlorothiazide, spironolactone and prednisone was continued and led to transient weight loss. Subsequently the patient had no response to corticosteroids or diuretics. By the end of the second month there was pronounced ascites and the patient complained of vague abdominal discomfort. A culture of fluid drawn from the abdominal cavity grew no organisms. Once during the fourth hospital month the child fainted and vomited while being photographed. The drugs being given at the time were prednisone and ACTH. Blood pressure was 100/30 mm of mercury and serum chemical values were: Sodium 107 mEq and potassium 4.9 mEq per liter, calcium 9 mg and phosphorus 5.1 mg per 100 ml. Subjective improvement followed oral sodium chloride supplementation. Later that month the blood pressure rose to 150/100 mm of mercury and the patient complained of diffuse abdominal pain. At that time serum calcium was 7.5 mg and phosphorus was 5.3 mg per 100 ml. The child also spoke of transient hyperalgesia and pruritis of the shoulders, but no abnormalities were evident on inspection. During the sixth month, while the patient was still receiving spironolactone, chlorothiazide and prednisone (5 mg three times a day) a 40-day course of 6-mercaptopurine was begun. There was no diuretic response. The number of leukocytes in peripheral blood fell moderately, erythroid activity of the bone marrow became slightly depressed and the drug was discontinued.

Coincidental with the last few days of mercaptopurine therapy, exquisitely tender, indurated pruritic erythematous lesions appeared over the thorax, and spread rapidly to the buttocks, back and extremities. Cellulitis was suspected and the patient was treated with penicillin and streptomycin. Leukocytes numbered 11,900 per cu mm with 79 per cent neutrophils. Blood cultures were ster-

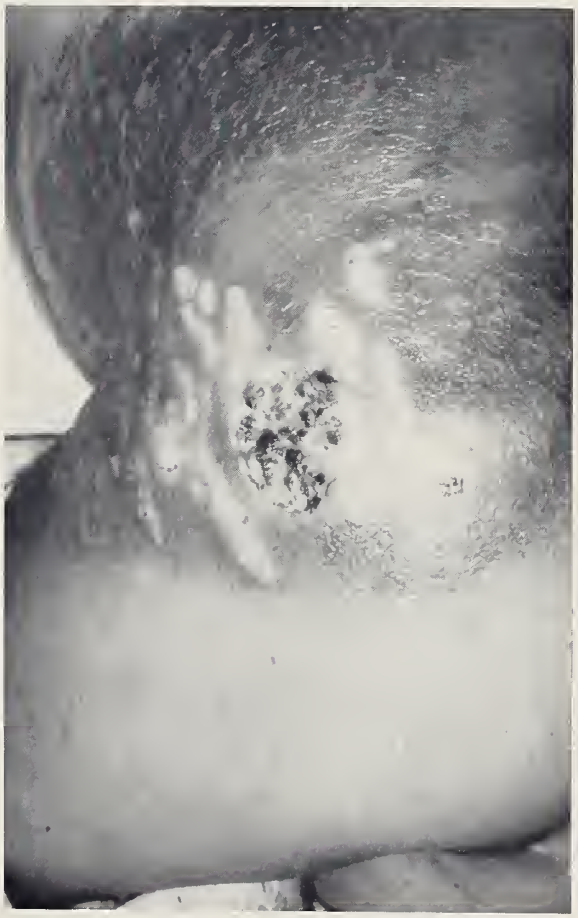


Figure 1.—Subcutaneous fat necrosis of the thigh and back with ulceration surrounded by increased pigmentation.

ile. Within 10 days the painful skin lesions had become vesiculated along the crease lines and striae. Subsequently the vesicles coalesced and ulcerated; thick yellow material oozed from these areas (Figure 1). Pigmentation and superficial desquamation later took place over the back. Exquisite and tender pruritis continued. Numerous cultures of the skin, oral cavity and feces grew candida albicans, but blood cultures remained sterile. Gradually the patient became febrile and leukocytosis appeared.

Two weeks before he died, a grand mal seizure associated with serum calcium of 5.6 mg per 100 ml occurred. Subsequently, oliguria, Cheyne-Stokes respirations, recurrent tetany and prolonged hypotension developed and terminated fatally.

Pathologic Findings

At autopsy, anasarca, a Cushingoid configuration and poor nutritional state were noted. The skin lesions involved thorax, abdomen, back and extremities. The largest ulcers on the back were 40 cm across. The skin along folds appeared elephantiatic, with deeply indurated subcutaneous tissues. The lesions were massively ulcerated and had dirty necrotic bases. In all the appearance was that of a third degree burn which had become badly infected. Hydrogen peroxide application to ulcer bases resulted in bubbling. Bullae 3 to 5 centimeters in diameter were present on the forearms and legs. Nonulcerated skin was indurated and edematous; areas with dried, scaling surfaces were also present. There was 200 ml of clear yellow ascitic fluid in the peritoneal cavity and 20 to 30 ml in each hemithorax.

The pancreas was enlarged and was surrounded and infiltrated by a large zone of hemorrhagic necrosis. The parenchyma, where visible, appeared dark red and soft. The pancreatic duct was dilated but contained no stones. The extrahepatic biliary tree was normal.

Almost all body fat showed necrosis, which was in general nonhemorrhagic. Cut section of the thick subcutaneous panniculus over the upper anterior thorax had the classical indurated marbled white opaque appearance of necrotic fat. The right kidney weighed 126 gm and the left 149 gm (the expected weights are 73 and 74 gm, respectively). The cortex on cut surface appeared pale and smooth with normal thickness but poor corticomedullary demarcation. The liver was pale and large, weighing 1,158 gm (expected weight 712 gm) and the spleen appeared atrophic, its weight 36 gm against an expected 68 gm.

Microscopic Findings

The renal process found was consistent with that of "lipoid nephrosis." The glomeruli showed minimal nonspecific glomerulitis characterized by mesangial thickening. They were normal in number and none were hyalinized. Tubules showed generalized slight luminal dilatation without specific epithelial changes. Occasional hemorrhagic microabscesses were present and they contained fungi with morphologic characteristics consistent with cryptococcus neoformans and candida albicans.

There was hemorrhagic pancreatitis with occasional inflammatory infiltrate in the edematous and hemorrhagic septa. Fat necrosis without calcification was prominent in peripancreatic fat deposits and the islets appeared normal. There was no vasculitis (Figure 2). The liver showed fatty metamorphosis while the appearance of the adrenal cortex was that of stress response with pseudotubular degeneration.

The fat necrosis found was subacute, without calcification, cyst formation or hemorrhagic areas.

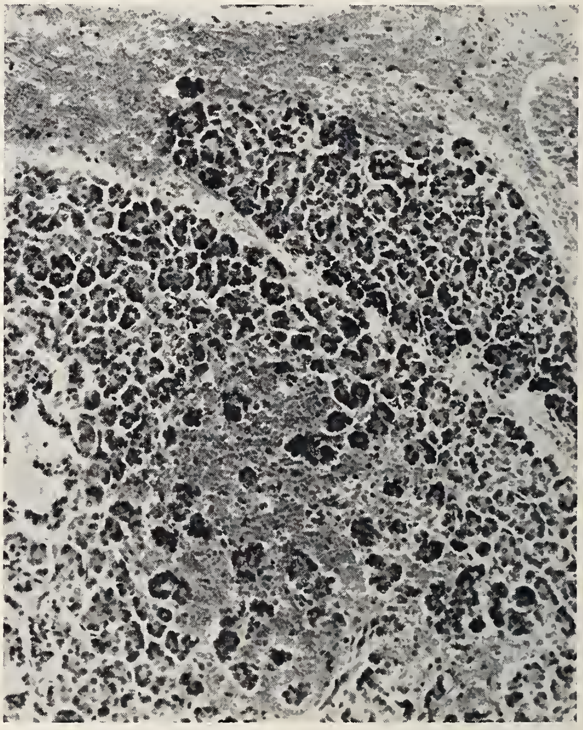


Figure 2.—Pancreas showing acute hemorrhagic necrosis of parenchyma (Hematoxylin and eosin stain, ×125).

Epidermal ulceration was demonstrated. The dermis was atrophic, with loss of adnexa. Necrotic subcutaneous fat with neutrophilic infiltrates formed the base of the ulcers.

The subcutaneous fat showed, in addition to necrosis, infection by yeasts, morphologically consistent with candida albicans and cryptococcus neoformans (Figures 3 and 4).

Similar yeasts were seen in the small foci of renal infection, and candida albicans alone was seen in foci of enteric and colonic ulceration, as well as in myocardial abscesses. There was no evidence of meningitis. Fat necrosis was present in peritoneal fat, colonic submucosal fat and epidural fat.

Additional findings included atheromatosis of the aorta, agonal thrombosis of right and left ventricles of the heart, pulmonary edema with foci of reactionless bacterial growth, lymphoid atrophy, osteoporosis, conversion arrest of rib and vertebra, colloid depletion of the thyroid gland and hypertrophy of the left ventricle. A parathyroid gland was normal. The bone marrow revealed moderate myeloid and erythroid hypoplasia.

Postmortem cultures of heart blood yielded candida albicans. Heart blood and lung cultures grew betaenterococcus. The cryptococcus-like yeast was not isolated.

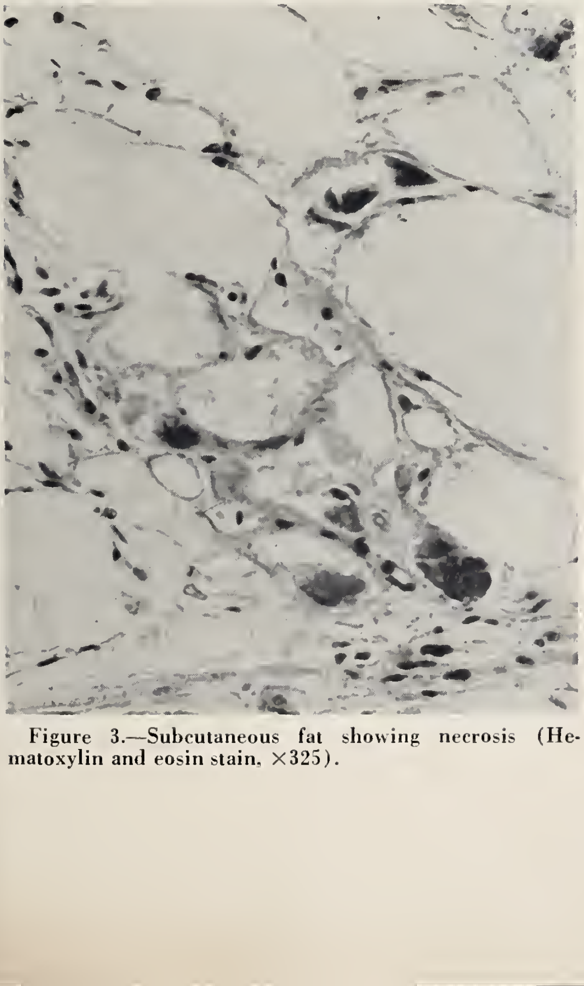


Figure 3.—Subcutaneous fat showing necrosis (Hematoxylin and eosin stain, ×325).

Discussion

Etiologic concepts and data on the incidence of "acute pancreatitis" have been confused by use of the same term for the classical clinical disease, with or without autopsy confirmation, as for incidental autopsy findings. Reported pancreatic lesions described as "acute pancreatitis" may vary from acute hemorrhage and necrosis[2] to variable foci of interstitial edema, ductular and acinar dilatation, degeneration and inspissation of secretions.[4,15] Often the findings are similar to those of "pancreatic autolysis," frequently interpreted in autopsy of adults as a meaningless phenomenon.[17,21] The significance of this appearance should not be minimized, however, because in many instances peripancreatic fat necrosis has been coexistent with it.[17,21] Variations in the microscopic appearance of "pancreatitis" may have etiologic or pathogenetic significance when the many agents or factors which have been implicated in its development are analyzed.

The incidence of pancreatitis diagnosed at autopsy is apparently reliably higher in children who were receiving steroids than in those who were not.[15] These children usually have not had classical clinical findings. Early reports described steroid-associated pancreatitis as most often mild, patchy and interstitial rather than diffuse and hemorrhagic.[4,15,17] Therefore, steroids may not be so directly related to acute hemorrhagic pancreatitis as has been implied. The majority of patients with steroid-associated acute hemorrhagic pancreatitis have been treated for "collagen diseases." Similar pancreatic involvement has also been encountered in patients receiving steroids who have chronic renal disease and hypertension. Pancreatic lesions,

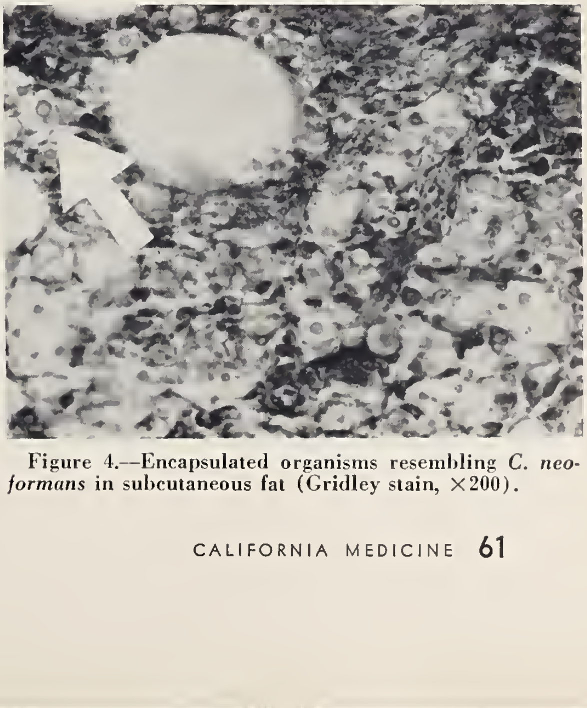


Figure 4.—Encapsulated organisms resembling *C. neoformans* in subcutaneous fat (Gridley stain, ×200).

however, occur in these patients in the absence of steroids, although with a lesser incidence.[15]

Since the pancreatitis noted in most patients with asthma or leukemia treated with steroids is not acute and hemorrhagic but patchy and milder,[15,21] vascular lesions common to both collagen disease and to chronic renal disease with hypertension may be a critical factor, resulting in progressive hemorrhagic disease.

McKay,[13] on the other hand, suggested that the common denominator in the genesis of pancreatitis may be intravascular coagulation. The effect of lipids upon platelet aggregation[7] may provide a unified concept for the diverse clinical circumstances in which hyperlipemia, vasculitis and pancreatitis coexist (for example, familial hyperlipemia, diabetes mellitus, collagen diseases and steroid-resistant nephrosis).

Recently, pancreatitis associated with immunosuppressive therapy has been reported by Tilney and coworkers,[19] who discussed the role of the drugs prescribed in conjunction with the underlying diseases, and their consequences. These investigators reported two cases in which acute hemorrhagic pancreatitis developed following renal transplantation, while the patients were receiving azathioprine (Imuran®) and corticosteroids. They postulated that patients receiving immunosuppressive therapy might fail to form antibodies to products of limited pancreatic necrosis. In the absence of these protective antibodies, subsequent necrotic foci may not be contained and thus pan-pancreatic hemorrhagic and necrotizing lesions may result.[1] Both of the patients had significant chronic and acute pancreatic vascular lesions. Despite clinical evidence compatible with "malignant hypertension," extrapancreatic vascular beds were not described and the vascular lesions were designated as "vasculitis of rejection."

Clinically, pancreatitis may not be suspected unless abdominal pain with peritoneal tenderness and elevation of serum amylase are present. In the case herein reported, extensive disseminated fat necrosis provided a diagnostic clue even though the patient at no time complained of severe abdominal pain. Both disseminated and subcutaneous fat necrosis are well recognized complications of pancreatitis and descriptions of these lesions are included in discussions of pancreatitis.* One observer proposed that fatal chronic relapsing nonsuppurative panniculitis might be, in fact, chronic pancreatitis associated with subcutaneous fat necrosis.[14] This proposal implies that pancreatitis may be relatively painless even when associated with disseminated fat necrosis. Observation of the patient in the present case lent support to this thesis. The severity of the skin lesions may be explained in part by secondary fungal infection. Candidiasis and cryptococcal infections either singly or in combination are known to occur during steroid therapy.[6,20] Candidiasis has also been reported in association with pancreatitis in patients who have not received steroids.[3,16]

Summary

Disseminated fat necrosis with severe skin involvement developed in a six-year-old Negro boy with nephrosis resistant to prolonged steroid therapy who also received a 40-day course of 6-mercaptopurine. At autopsy acute hemorrhagic pancreatitis was found with subcutaneous fat necrosis and secondary fungal infection. Some of the factors implicated in acute pancreatitis, for example, steroid and immunosuppressive therapy, vasculitis, hyperlipemia and intravascular coagulation are discussed. Although the causative roles of individual factors presumed to operate at different points in the pathogenesis of pancreatitis cannot be completely established, the possibility of relatively silent pancreatitis should be borne in mind when necrotizing skin lesions develop in patients with abnormal immune processes or who are receiving immunosuppressive drugs.

GENERIC AND TRADE NAME OF DRUG

Azathioprine—*Imuran.*

REFERENCES

1. Apostolou, K., Nasbeth, D. C., Shapiro, D. H., and Ryak, P.: Immunity to lethal effects of acute pancreatitis, S. Forum, 26:382-384, 1965.

2. Baar, H. S., and Wolff, O. H.: Pancreatic necrosis in cortisone-treated children, Lancet, I:812:815, 1957.

3. Barrett, B., Volwiler, W., Kirby, Wm. M., and Jensen, C. R.: Fatal systemic moliliasis following pancreatitis, Arch. Int. Med., 99:209-123, 1957.

4. Carone, F. A., and Liebow, A. A.: Acute pancreatic lesions in patients treated with ACTH and adrenal corticoids, New Engl. J. Med., 257:690-697, 1957.

5. Cohen, D., Schoop, R., and Berman, M.: Subcutaneous fat necrosis and polyarthritis in a patient with pancreatitis, Arch. Derm. & Syph., 80:602-603, 1959.

6. Goldstein, E., Rambo, O. N.: Cryptococcal infection following steroid therapy, Ann. Int. Med., 56:114-120, 1960.

7. Hoak, J. C., Connor, W. E., Warner, E. D.: Thrombogenic effects of albumin-bound fatty acids, Arch. Path., 81:136-139, 1966.

8. Immelman, E. J., Bank, S., Krige, H., and Marks, I. N.: Roentgenologic and clinical features of intra-

---

*Reference Nos. 5, 8, 9, 10, 11, 18.

medullary fat necrosis in bones in acute and chronic pancreatitis, Amer. J. Med., 36:96-105, 1964.

9. Jackson, S. H., Savidge, R. S., Stein, L., and Varley, M.: Carcinoma of the pancreas associated with fat necrosis, Lancet, 2:962-967, 1952.

10. Kushner, D. S., and Szanto, P. B.: Fulminant polyarthritis, fever and cutaneous nodules in an alcoholic patient, J.A.M.A., 167:1625-1632, 1958.

11. Lucas, P. F., and Owen, T. K.: Subcutaneous fat necrosis, polyarthritis and pancreatic disease, Gut, 3:146-148, 1962.

12. Marczynska-Robowska, M.: Pancreatic necrosis in a case of Still's disease, Lancet, I:815-816, 1957.

13. McKay, D. G.: Disseminated Intravascular Coagulation, Harper and Row, New York, 1965, pp. 493.

14. Moore, S.: The relation of pancreatic disease to Weber-Christian disease, Canad. Med. Assn. J., 88: 1238-1241, 1963.

15. Oppenheimer, E. H., and Boitnott, J. K.: Pancreatitis in children following adrenal cortico-steroid therapy, Bull. J. Hopkins Hosp., 107:297-306, 1960.

16. Pancreatitis and moniliasis. Case report from the Massachusetts General Hospital. Case No. 57-1961, New Engl. J. Med., 265:287-293, 1961.

17. Stumpf, H. H., Wilens, S. L., and Somoza, C.: Pancreatitic lesions and peripancreatic fat necrosis in cortisone-treated rabbits, Lab. Invest., 5:224-235, 1956.

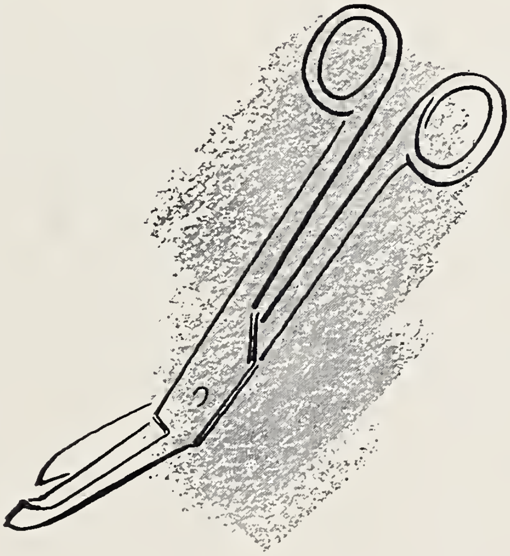
18. Swerdlow, A. B., Berman, M. E., Gibbel, M. I., Valaitis, J.: Subcutaneous fat necrosis associated with acute pancreatitis, J.A.M.A., 173:765-769, 1960.

19. Tilney, N. L., Collins, T. T., Jr., and Wilson, R. E.: Hemorrhagic pancreatitis, New Engl. J. Med., 274:1051-1057, 1966.

20. Walz, D. V., Hasenclever, H. F., McKeen, A. P.: A dual human infection with candida albicans and cryptococcus neoformans, Amer. J. Clin. Path., 26:794-798, 1956.

21. Williams, W., and Patrick, J. R.: Death from asthma in children, Amer. J. Dis. Child., 110:4-23, 1965.

22. Zion, M. M., Goldberg, B., Suzman, M. M.: Corticotrophin and cortisone in treatment of scleroderma, Quart. J. Med., 24:215-227, 1955.



MEDICAL STAFF CONFERENCE

The Spectrum Produced by Malignant Carcinoid Tumor

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California Medical Center, San Francisco. Taken from transcriptions, they are prepared by Drs. Martin J. Cline and Hibbard E. Williams, Assistant Professors of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine.

Dr. Anthony Sebastian*: The patient, a 35-year-old Caucasian mother of two, entered Herbert C. Moffitt Hospital with chief complaint of severe flushing episodes. The present illness began 13 months ago with the first of recurring episodes of diarrhea and flushing of the face. These episodes increased in severity. Two months after onset they were accompanied by severe, constant epigastric pain, and after another two months by severe crushing substernal chest pain. Eight months before admission, the patient became anorectic and began to lose weight. She was admitted to the San Francisco General Hospital where the diagnosis of a malignant carcinoid tumor was made.

On liver scan, she was found to have metastatic disease to the left lobe of the liver. Laboratory studies showed elevation of urinary 5-hydroxyindolacetic acid and of plasma serotonin. A provocative test with intravenous injection of epinephrine precipitated a severe flushing episode. Left lobar hepatectomy was carried out and an extensive search of the bowel at this time failed to reveal a primary source. Microscopic examination of the liver confirmed the diagnosis. Postoperatively, the patient had an acute transitory episode of acute psychosis. Thereafter, she did quite well and for several months was completely free of symptoms until two months ago when anorexia and flushing episodes recurred.

On physical examination, the patient was noted to be thin and apprehensive. Vital signs were normal except for intermittent tachycardia. The skin was clear, without telangiectasia. No abnormality was heard on auscultation of the lungs. There was a grade III systolic ejection murmur, best heard in the third intercostal space along the left sternal border, associated with an ejection click. The liver was enlarged below the level of the umbilicus and was smooth and slightly tender. The spleen was felt 3 cm below the left costal margin. The rest of the physical examination was within normal limits.

Laboratory data included increased serum alkaline phosphatase, depressed prothrombin time, slightly elevated butanol-extractable iodine, and elevated urinary 5-hydroxyindolacetic acid both before and after flushing attacks. During her stay in hospital the patient had several episodes of flushing, lasting as long as 45 minutes, accompanied by apprehension, tremulousness, hyperventilation, intense erythema of the face, arms, chest and neck, tachycardia, slightly decreased blood pressure and pronounced conjunctival injection. She complained of severe abdominal cramping pain, nausea and stabbing pain in the chest. An electrocardiogram taken during one of the episodes was normal, and in none of them were diaphoresis, dyspnea, wheezing, facial edema, lacrimation or pupillary changes observed.

Dr. Warren M. Russell*[1]: A roentgenogram of the chest showed that a thoracotomy had been performed and there was some regrowth of bone along the rib tract. The right diaphragm was con-

---

This work was supported in part, by U.S.P.H.S. Grant No. HE 09964 and in part, by U.S.P.H.S. Grant No. HE 06286, through the C.V.R.I.

*Intern in the Department of Medicine.

*[1]Assistant Professor of Radiology.

siderably elevated and the liver was large. A gastrointestinal series demonstrated displacement of the stomach to the right and anteriorly by a very large mass in the region of the liver, presumably a greatly enlarged liver. A late film, with most of the contrast medium in the colon, demonstrated a large liver and abnormally large bowel. Much of the colon, particularly the transverse portion, was moderately dilated with some areas of extrinsic indentation. On fluoroscopic examination the transverse colon appeared relatively fixed. Many portions of the large and small bowel had edematous folds suggesting vascular or lymphatic obstruction, although a similar appearance has been seen in the carcinoid syndrome, without metastatic lesions. The terminal ileum was only faintly visualized but it also appeared edematous. The overall appearance was nonspecific; it would be consistent with a diffuse peritoneal carcinomatosis from any source.

DR. KENNETH L. MELMON*[2]: This disease is a fascinating one which has stimulated a great deal of research in the 10 to 12 years since its recognition. The syndrome was initially described by Thorson and Waldenstrom.[1] Dr. Waldenstrom is a very astute clinician. Several years ago he had a young patient who had relatively few symptoms but complained of flushes. On examination the murmur of pulmonic stenosis was heard. Dr. Waldenstrom was unable to explain any relationship between the flushes and the interrupted pulmonic blood flow. On a visit to a hospital in Europe, the house staff presented another patient who was flushing, and who had a carcinoid tumor of the liver. It later became apparent that the patient whom he had seen with pulmonic stenosis and flushing also had a carcinoid tumor which had spread to the liver. The classical paper and the description of the patient with a typical ileal carcinoid tumor was conceived, and the disease entered the medical canon.

Almost simultaneously, Lembeck isolated from carcinoid tumors two biologically active substances. One, which he called serotonin, was subsequently chemically identified as 5-hydroxytryptamine.[2] Lembeck stated that he could not reproduce all the symptoms of the syndrome when he gave serotonin to patients with carcinoid tumors. Massive amounts of serotonin produced symptoms, but the patients objected violently and stated that these were not the symptoms that they spontaneously experienced. The second substance has not been identified, but may be the peptide bradykinin. The hallmark of the disease became the excessive production of serotonin and, despite Lembeck's warning, by 1952 all the symptoms of the syndrome were attributed to serotonin. In fact, the current medical writer, William Bennett Bean, wrote a poem summarizing the disease[3]:

This man was addicted to moanin',
Confusion, edema, and groanin',
Intestinal rushes,
Great tri-colored blushes,
And died of too much serotonin.

Thus, the disease was thought to be completely understood and in textbooks the chapters on the carcinoid syndrome have become briefer and briefer. They are likely to become longer in the future, however, for I hope to show this morning that these patients do not "die of too much serotonin," and that the disease has a spectrum of clinical as well as biochemical abnormalities. The arguments that serotonin does not cause all symptoms of carcinoid syndrome are based on the following observations: (1) Serotonin is not produced in excess by all patients who have the carcinoid syndrome; (2) the syndrome changes character depending on the site of the primary carcinoid tumor and not its serotonin production; and (3) the syndrome may be produced by other varieties of tumors, some of which have no ability to increase the production of serotonin.

I shall try to describe the spectrum of the carcinoid syndromes. The ileal carcinoid tumor with a syndrome is the most common. It may be identified at operation and does not produce symptoms (although it probably does produce excess serotonin) until metastasis to the liver occurs. The terminal ileum is the most commonly involved ileal segment. Metastasis occurs first in the mesenteric lymph nodes and then the liver. The huge liver observed in the patient presented this morning is not unusual in a patient with a metastatic carcinoid tumor which has been present for several years. The liver was functioning normally in this patient, as it frequently does, despite massive replacement by neoplastic growth. The left lobe of the liver receives the blood coming from the esophagus, the stomach and the upper small intestine. Tumors in the terminal ileum would be expected to drain into the right lobe of the liver since the venous blood of the ileum drains directly into that lobe. Thus, if one finds a patient with an ileal carcinoid tumor and a carcinoid nodule in the left lobe of the liver, the nodule is unlikely to be an isolated metastatic lesion. I think this was the case with the patient today. When patients with carcinoid tumor are operated upon, they frequently have unusually severe fibrotic reactions. These are severe enough to convince experienced surgeons to avoid laparotomy if possible. I think the upper gastrointestinal series in the present case demonstrated the matting and partial obstruction caused by the fibrosis subsequent to laparotomy.

---

*[2] Assistant Professor of Medicine and Pharmacology, Associate—Cardiovascular Research Institute.

The heart lesions in the classical carcinoid syndrome consist of diffuse deposits of collagen over the entire endocardium including the valve surface.[4] They may interfere with closure to such a degree that very severe insufficiency, stenosis or congestive heart failure occurs. These lesions are usually on the right side of the heart. Serotonin concentration has been presumed to be higher on this side than on the left because lung tissue has abundant monoamine oxidase activity. However, Robertson and his coworkers could not demonstrate consistent significant differences in the serotonin concentration between pulmonary artery and venous blood or a consistent rise in peripheral arterial serotonin during a flush.[5] The predominant lesions will occur on the left side of the heart in patients with hepatic carcinoid tumor when there is an associated atrial septal defect or in patients with bronchial carcinoid tumor, where venous blood from the tumor drains directly into the left atrium. Microscopic lesions of carcinoid heart disease are usually present throughout the endocardium of both sides.

If serotonin were responsible for all the symptoms of carcinoid syndrome, interference with its production or with its effect should abolish or ameliorate the symptoms. N-methylated lysergic acid diamide (methysergide) is a drug which appears to work primarily on the effects of serotonin and does not influence the production of 5-hydroxyindolacetic acid. Administration of this drug produces a predictable decrease in the number and weight of bowel movements. On occasion, when a patient is experiencing malabsorption, the fat content of the stool also decreases.[6] This drug does not, however, consistently alter the flushes or prevent the heart lesions or other symptoms of the carcinoid syndrome. Similarly, if serotonin synthesis is blocked by administration of adequate doses of a decarboxylase inhibitor (alphamethyldopa, for example) the symptoms of the classic syndrome do not change. Serotonin administration may produce a flush when given in huge amounts (3 mg or more), but the flush is usually associated with severe abdominal pain, headache, urinary and fecal incontinence, apnea, a *rise* in the blood pressure and bradycardia.

A retinal lesion, which has not yet been described in the literature, may be seen in the carcinoid patient, but probably is not produced by serotonin. It is found in the patient who consistently flushes. The lesions are very small punched-out areas of postischemic change in the peripheral retina. Usually they do not produce any symptoms, but in one of the patients whom I observed, this lesion was located on the macula and caused blindness during flushing. The peptide bradykinin seems to be able to produce the vascular changes seen in the carcinoid eye lesion.

It has become obvious in the course of observing several patients that the typical carcinoid syndrome which occurs with carcinoid tumors of the ileum is not seen consistently when tumors are located at other sites. In the ileal carcinoid syndrome, at least 99 per cent of the indoles are completely metabolized to 5-hydroxyindolacetic acid (5-HIAA); this is not the case with carcinoid tumors in a particular atypical location. Oates observed an atypical syndrome in a patient with gastric carcinoid.[7] The tumor lacked decarboxylase activity and therefore could not convert 5-hydroxytryptophan (5-HTP) to serotonin (5-hydroxytryptamine). The excessive 5-hydroxytryptophan was secreted into the blood stream and circulated as such until it was decarboxylated by the kidney. In this patient, in addition to 5-HIAA, there was a remarkable increase in the urinary concentrations of 5-HTP and 5-HT. A great deal of histamine was also produced by this patient and it may have been responsible for the unusual, diffuse, "blotchy" flushes and gastric ulcers. A relatively low 5-HIAA, high serotonin and high histamine concentration in the urine are characteristic of patients with primary gastric carcinoid tumor.

Another unusual patient with an atypical carcinoid syndrome was sent to us by an allergy clinic. The patient had a very responsible position with the government. He would frequently go home sick when an exceedingly important deadline was to be met. Between sick spells, he was asymptomatic. During an attack he was admitted to the National Institutes of Health and had a severe flush which did not resemble those we had seen before. In addition, puffiness of the face, periorbital edema, uncontrollable lacrimation and salivation were prominent features. The flush continued for several days and was associated with hypotension, peripheral edema, tremulousness, fever and oliguria.

Yet another patient came to us with mitral stenosis and similar complaints. She had been operated upon previously for a bronchial carcinoid tumor. When she became oliguric and retained fluid, severe, life-threatening pulmonary edema developed. Between flushes, she had no symptoms. The flushes occurred on a predictable schedule—two days of every nine over a two-year period. Since the patient did have hemodynamically significant mitral stenosis, we wondered if the carcinoid tumor had recurred; indeed it had. Because this patient resembled the previous unusual patient with a bronchial carcinoid tumor, and because there was some evidence that corticosteroids had modified the previous patient's severe flush, we administered prednisone during a flush. No effect was noted. We then gave a large amount of prednisone (40 mg per day) for longer periods and there were no further flushes. Within hours after placebo was substituted for prednisone, three flushes occurred. We resumed prednisone therapy and the patient remained symptom-free for more than two years.[8] During this entire period the patient's excretion of 5-HIAA was only modestly elevated. Steroids in other types of carcinoid syndrome have little effect or may ameliorate the flushing slightly.

The next patient with this variant of the classical syndrome was a piano player in a cabaret. She complained of ceaseless crying but the chief complaint was that she had to take Turkish towels to bed with her to keep her saliva from soaking through the pillow. This story sounded familiar, and we suspected a bronchial carcinoid despite the absence of typical flushes. It was subsequently found that she had a bronchial carcinoid tumor with widespread metastasis.

Interestingly, when we searched the literature we found a very high incidence of pluriglandular adenomatosis, Cushing's disease and acromegaly in the patients with bronchial carcinoid tumors. Typical features of the bronchial carcinoid tumor are summarized in Table 1.

Reports of carcinoid tumor began appearing which stressed the tumor's ability to produce histamine, 5-HTP and catechols. It became apparent that this class of tumors may be multi-potential and capable of producing several biologically active substances. The next question was: Which of the substances produced by the tumor was responsible for the flushes? It could not be serotonin because:

1. Anti-serotonin agents were ineffective in preventing flushes;

2. Serotonin is not made by many tumors associated with the syndrome;

3. Other tumors which produce the carcinoid syndrome may not have the histologic features of carcinoid or the ability to produce serotonin. The latter include pancreatic duct neoplasms, non-beta islet cell adenomas, neoplasms in the biliary tract, stomach and ovaries and oat cell carcinomas of the bronchus;

4. The amount of serotonin excess does not correlate with the severity or frequency of flushes. For example, the patient described today had only a modest elevation in 5-HIAA excretion but very severe symptoms.

TABLE 1.—*Distinctive Features of the Bronchial Carcinoid Syndrome*

1. Prolonged, severe flushing attacks.
   A. May include:
      Anxiety, tremulousness, disorientation.
      Periorbital and facial edema.
      Excessive lacrimation, salivary gland enlargement with excess salivation, diaphoresis.
      Nausea, vomiting, explosive diarrhea.
      Dyspnea, wheezing.
      Fever.
      Hypotension, tachycardia.
      Oliguria.
      Death during an attack.
   B. Possibility of unique and dramatic control by corticosteroids. Phenothiazine drugs also useful.
2. Widespread metastasis with osteoblastic bone lesions.
3. Left-sided heart lesions, with pulmonary edema during flushing.
4. Association with other endocrine disorders.

We reasoned that if the tissue was able to make many biologically active chemicals, then it could be making another yet undetected chemical responsible for the flush. Moreover, we were impressed that the autonomic nervous system was related to flushes. Patients who experienced nervousness or tension seemed to flush frequently, as did the patient discussed this morning. Was there any relationship between the autonomic nervous system and the production of the flush? Could catechols for some reason have a paradoxical activity and produce flushes and hypotension in carcinoid patients?

As we were thinking thus, so was Robertson, whom I referred to earlier. Dr. Robertson showed that epinephrine given intravenously to carcinoid patients produced flushes. The flush was associated with profound hypotension just as the patient had experienced in spontaneous flushes. By infusing catechols into the internal carotid artery of a patient with carcinoid tumor, the investigators showed that these flushes were not a direct effect of catechol. The local effect of the epinephrine produced blanching in half of the face, and the systemic effect (flushing) followed in about 60 to 90 seconds. Robertson and later Levine reasoned that catechols were releasing a vasodilating substance responsible for the flush.[9] The same effects were produced by the ingestion of cheese which contained tyramine. Tyramine's major pharmacologic effect is to release endogenous catechols which then indirectly produce the flush.

The relationship between the catecholamine and the flush has practical importance. As was illustrated this morning, subpharmacologic doses of catechols can be used as a diagnostic test in evaluating the carcinoid patient who has low 5-HIAA excretion. In addition, one must remember that agents which increase peripheral vascular resistance by the release of endogenous catechols cannot be used during operation because they will continue to provoke the flush and the hypotension associated with it. Such agents include epinephrine, norepinephrine, metaraminol and mephentermine. When a drug must be used to increase peripheral vascular resistance, it has to be one which has a direct effect on the small vessels independent of catechol release, for example, methoxamine or angiotensin. The latter two drugs can increase blood pressure without provoking a flush.

If catechols do not produce the flush, what does? We looked for a substance which could lower blood pressure, stimulate lacrimation and salivation, produce edema, increase cardiac output and be released by catecholamines. Among vasodilator peptides which are normally found in the gut, in the central nervous system and in the lacrimal and salivary glands is bradykinin. Intravenous administration of bradykinin produced a flush which was similar to the spontaneous and epinephrine-provoked flushes.[10] In addition, the peripheral vascular changes associated with a spontaneous flush were mimicked by the administration of bradykinin. Finally, we were able to look for the enzyme kallikrein, which produces bradykinin and the peptides themselves in several patients with the carcinoid syndrome. Our data are summarized by the following statements:

1. Kallikrein can be found in some carcinoid tumors. Dr. Nathan Back of Ohio has detected kallikrein in the primary tumor of the patient presented today;

2. This kallikrein is released into the systemic circulation during a flush;

3. The peptide itself can be isolated from the hepatic vein during an epinephrine-induced flush;

4. The peptide formed by the tumor kallikrein has been characterized and it is lysyl-bradykinin.[11]

Oates has identified bradykinin in hepatic venous blood.[12] The kinin concentration rises in the blood in association with the flushes, the drop in mean blood pressure and an increase in forearm blood flow.[13] (Chart 1.) These facts, I think, relate the kallikrein-kinin system as a pathogenetic one in the production of the flush (Chart 2).

We do not have time to discuss treatment, but one can treat symptomatically if one knows what the tumor is producing. There is no good evidence that antitumor agents are effective in decreasing symptoms or in changing the prognosis in this disease. These agents need further, controlled evaluation.

In summary, we must now view the carcinoid syndrome as a series of events which can be pro-

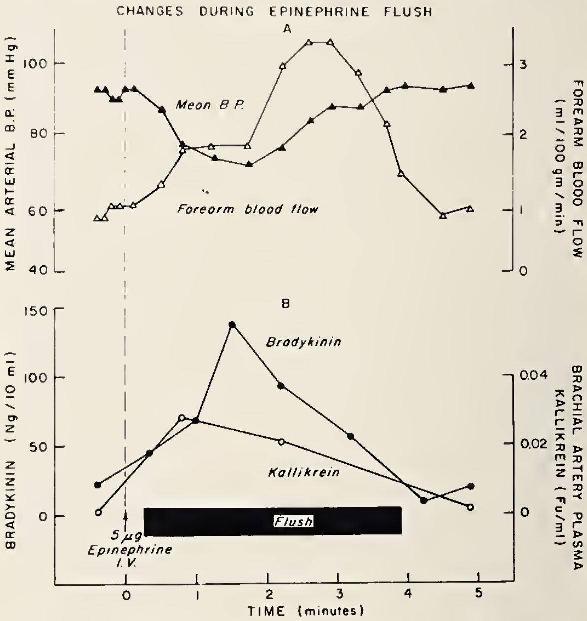


Chart 1.—Serial determination of mean arterial pressure (B.P., solid triangles), forearm blood flow (open triangle, top panel A), bradykinin (closed circles), and kallikrein (open circles, bottom panel B) before and after intravenous injection of epinephrine into a patient with carcinoid tumor. The lower than normal control values for forearm blood flow are compatible with the fact that the patient was in congestive heart failure.[13] Fu=Frey Unit; Ng=Nanogram (also called millimicrogram).

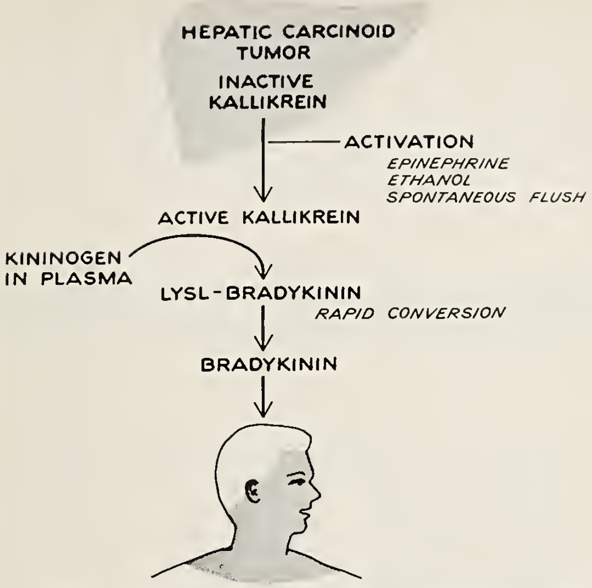


Chart 2.—Summary of the present concepts by which kallikrein and kinins may contribute to the carcinoid flush. The flush mechanism, however, is complex; in some patients, kinin levels are normal. (Chart reprinted by permission of *Physiology and Pharmacology for Physicians*, "Kinins in Medicine—Present and Future," Melmon, K. L., Vol. 1, No. 6, June 1966, p. 3.)

duced by a variety of tumors, including neoplasms that have no relationship to carcinoid tumor. When the tumor has the histological characteristics of carcinoid, the syndrome produced seems dependent upon the location of the primary growth. Any tumor may produce a variety of biologically active chemicals at any given time. These chemicals include serotonin, 5-hydroxytryptophan, histamine, catecholamines and vasodilator peptides-kinins. It may be possible that the tumors are capable of producing other vasoactive peptides or even more sophisticated proteins capable of inducing such changes as acromegaly, pluriglandular adenomatosis, Cushing's disease or pigmentation. It seems reasonable to view the syndrome in a particular patient as the momentary "algebraic sum" of these biologically active substances. It also appears mandatory to evaluate each case separately and thoroughly before reasonable therapy is offered or can be expected to be efficacious.

Dr. Lloyd H. Smith, Jr.[*3]: I think the applause reflects the outstanding job that Dr. Melmon has done pulling together this clinical syndrome and its background and the chemical and pharmacologic features.

Recent Summaries in the Literature

1. Thorson, A., Biörck, G., Björkman, G., and Waldenström, J.: Malignant carcinoid of the small intestine with metastasis to the liver, valvular disease of the right side of the heart (pulmonary stenosis and tricuspid regurgitation without septal defects), peripheral vasomotor symptoms, bronchoconstriction, and an unusual type of cyanosis. A clinical and pathologic syndrome, Am. Heart J., 47:795, 1954.

2. Lembeck, F.: 5-hydroxytryptamine in a carcinoid tumor, Nature, 172:910, 1953.

3. Bean, William B.: Vascular Spiders and Related Lesions of the Skin, Charles C Thomas, 1958, p. 117.

4. Roberts, W. C., and Sjoerdsma, A.: The cardiac disease associated with the carcinoid syndrome (carcinoid heart disease), Amer. J. Med., 36:5, 1964.

5. Robertson, J. I. S., Peart, W. S., and Andrews, T. M.: The mechanism of facial flushes in the carcinoid syndrome, Quart. J. Med., 31:103, 1962.

6. Melmon, K. L., Sjoerdsma, A., Oates, J. A., and Laster, L.: Treatment of malabsorption and diarrhea of the carcinoid syndrome with methysergide, Gastroenterology, 48:18, 1965.

7. Oates, J. A., and Sjoerdsma, A.: A unique syndrome associated with secretion of 5-hydroxytryptophan by metastatic gastric carcinoids, Amer. J. Med., 32:333, 1962.

8. Sjoerdsma, A., and Melmon, K. L.: Severe flushing reactions responsive to steroids in patients with bronchial carcinoid, Lancet, 2:791, 1964.

9. Levine, R. J., and Sjoerdsma, A.: Pressor amines and the carcinoid flush, Ann. It. Med., 58:818, 1963.

10. Oates, J. A., Melmon, K. L., Sjoerdsma, A., Gillespie, L., and Mason, D.: Release of a kinin peptide in the carcinoid syndrome, Lancet, 1:514, 1964.

11. Melmon, K. L., Lovenberg, W., and Sjoerdsma, A.: Identification of lysylbradykinin as the peptide formed in vitro by carcinoid tumor kallikrein, Clin. Chim. Acta, 12:292, 1965.

12. Oates, J. A., Pettinger, W. A., and Doctor, R. B.: Evidence for the release of bradykinin in carcinoid syndrome, J. Clin. Invest., 45:173, 1966.

13. Mason, D. T., and Melmon, K. L.: Abnormal forearm vascular responses in the carcinoid syndrome: The role of kinins and kinin generating systems, J. Clin. Invest., 48:1685, November 1966.

[*3] Professor and Chairman, Department of Medicine.

California
Medicine

Editor
DWIGHT L. WILBUR, M.D.

Assistant to the Editor
ROBERT F. EDWARDS
For information on preparation of manuscript, see advertising page 2

Policy Committee—Editorial Board
JAMES C. MacLAGGAN, M.D., San Diego
JOHN G. MORRISON, M.D., San Leandro
WILLIAM F. QUINN, M.D., Los Angeles
JOSEPH W. TELFORD, M.D., San Diego
CARL E. ANDERSON, M.D., Santa Rosa
HELEN B. WEYRAUCH, M.D.
San Francisco
DWIGHT L. WILBUR, M.D., San Francisco

EDITORIAL

Medicare and Medi-Cal

Now that ten months have passed since Medi-Cal and six months since Medicare became effective, it is appropriate to review and comment on the experiences of California physicians with these new laws. Considered the outstanding events in Medicine in 1966 the changes initiated by these statutes vitally affect all practicing physicians.

Medicare

From 1 July through 30 November 1966, the two Blue Cross plans in California had received almost 160,000 claims from hospitals and paid out nearly $60 million. The regulations have been modified so that one signature by the patient is now sufficient to support all claims for required services in a hospital. This requirement, we are told, has speeded up the processing of claims. The Blue Cross plans are further recommending that the requirement for the signature of a patient should be eliminated altogether, since it is often difficult for the hospital to obtain a signature, particularly in out-patient cases. The regulations have already been modified so that the Social Security Administration will now accept the signature of a relative, or even of a hospital official, if the patient is too ill to sign or has died.

Major problems that remain are caused by the complexity of the Medicare benefits. The beneficiaries do not understand to what they are and to what they are not entitled. Hospitals have found that billing for outpatient benefits under the law is most complicated.

By the first week in December 1966, California Blue Shield (CPS) had received a total of over 720,000 Part B Medicare claims and had paid out almost $7 million.

A substantial number of claim forms must still be returned for further information before they can be processed, resulting in an estimated backlog of approximately 140,000 claims over and above the normal number that are in the process of being paid.

Approximately 300,000 of the Medicare claims received by California Blue Shield through November 1966 were for patients who are eligible for both Medicare and Medi-Cal benefits. These "dual coverage" claims must be processed under the Medicare audit before they can be processed for Medi-Cal payment. This delay in processing and payment is, of course, a cause of considerable concern to many physicians and other providers of service. It is hoped that in the near future the law can be amended so that such claims need only be audited once and then paid with one check, issued either by the Social Security Administration or the State, rather than by two as at present. To do this it would be necessary for the two entities of government—federal and state—to make possible a bookkeeping or accounting record and credit of the proper amounts disbursed under both programs. This solution would be welcomed both by physicians and the carriers.

Medi-Cal

From 1 March through 30 November 1966, the Blue Cross plans processed more than 900,000

Medi-Cal claims. A breakdown of these claims by type of institution is as follows:

County hospitals	291,937
Community hospitals	338,694
Nursing homes	253,508
Mental and rehabilitation facilities	18,508
Home health agencies	23,556

Of the claims received, more than 83,000 were to be returned for revision or recheck. As the program matures, the number of claims that need to be returned will decrease. About $138 million was paid for claims that have been audited. In addition, claims totaling over $27 million for county hospitals were audited but not paid.

From March through November 1966, Blue Shield received nine and a half million claims, of which more than eight million had been paid and a total of over $98 million has been paid to physicians, dentists, pharmacists, podiatrists, optometrists, chiropractors and others under the program.

The four claimant categories were as follows:

	Claims Received	*Claims Paid*	*Total Amount Paid*
Physicians	2,558,813	2,014,460	$48,471,155
Other medical	680,603	533,992	12,373,365
Dental	373,540	277,626	16,917,682
Drugs	5,961,560	5,196,762	20,310,094

During November, 70 per cent payments were sent out on approximately one-half million claims for which eligibility had not been certified. The purpose of the 70 per cent payment was to relieve physicians and others of the financial burden imposed by the previous inability of Blue Shield to pay uncertified claims. When these partial payments were made, the physicians and others were sent a list of the claims in question.

Each physician was asked to check the data on the claim for accuracy and to correct it if necessary. When the correct information is returned to Blue Shield, eligibility of the claim will again be checked. When eligibility is verified, the balance of the payment will be made.

In the future, claims which cannot be matched against the eligibility file within 30 days after they are received will be returned to the physician or other provider so that such a backlog of unmatched claims will not again accumulate. All physicians and their office assistants should exercise great care in recording correct names, dates of eligibility, case numbers and aid categories in order to speed up verification by Blue Shield.

The former and present administrators of the program expressed deep concern about the total estimated cost of the program during the first fiscal year, which will end 30 June 1967. The following unexpected and unforeseen increases are causing major concern:

1. A substantial increase in the number of persons receiving nursing home care.
2. Substantial increases in the per diem cost of hospital and nursing home care.
3. The unknown and unresolved costs of physicians' services in county and teaching hospitals.
4. The unknown State cost of any increase in the medical care costs of counties for care rendered to persons in county hospitals.

It has been estimated that the cost of the program may overrun its budget by fifty million dollars. However, there are other estimates that the program will stay within its budgetary allowances.

Both the former and present administrators recommended that additional safeguards be placed upon nursing home utilization in order that the program can be made more financially stable. It is felt that the program benefits and utilization should be carefully watched and controlled rather than that rates of compensation be decreased. We commend this approach and hope that the new administrator of the program will continue to support this comprehensive, high quality medical care program.

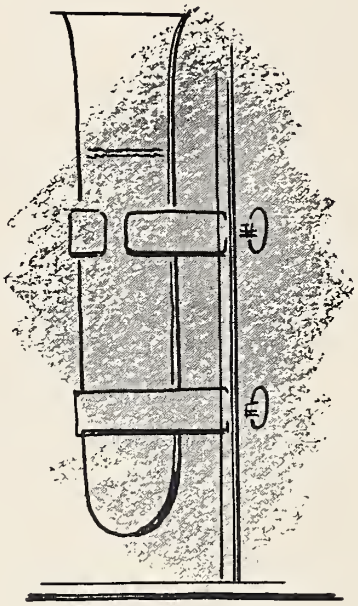
New Extended Care Benefits Under Medicare

Extended care benefits under Medicare for covered persons 65 and over became effective 1 January 1967. Under them a person may be admitted to a participating extended care facility for further treatment of a condition for which he was put in hospital. The hospital benefits under Part A of Medicare will pay for the full cost of covered services for the first 20 days and all but $5 per day for 80 additional days in each "spell of illness." Within each spell of illness the hospital benefits cover up to 90 days of hospital care and 100 days of extended care services.

The extended care service contemplated is distinct from acute hospital care on one hand, and on the other it is distinct from long-term care in a nursing home or home for the aged. It has been estimated that this Medicare benefit will not pay for more than one-fourth of the skilled nursing home beds in the country at any one time. It is not expected that all homes which have been considered skilled nursing homes will qualify and be certified as eligible for payment under the Medicare program.

Some problems need to be overcome to attain the potentialities of high quality extended care services. There are shortages of health personnel and obsolescence of equipment in some communities. Probably most of the facilities which will qualify in the early days of the program will be closely associated with hospitals.

There appears to be a need for and a challenge to physicians to become interested and involved in the growing development of high quality extended care facilities, in order that patients will receive the best possible care at the most economical rate within reason. Medical records in such facilities will receive more attention than in the past and medical committees will review the care rendered and the utilization patterns. This will not all happen overnight but there will surely evolve a broadening of a new level of care to be integrated with acute hospital care.



California Medical Association

NOTICES AND REPORTS

The Budgeting Process

THE BUDGETING PROCESS of the California Medical Association is complex and requires program planning as far in advance as 20 months.

The CMA fiscal year begins 1 July and ends 30 June. The House of Delegates, when it meets in March or April, approves the budget for the fiscal year that begins three months later, and the staff and Finance Committee begin work on the budget at least five months before that. This means up to 20 months can elapse between the time when the budget is begun and final expenditures are made. Often, during this time, unforeseen situations require immediate, unbudgeted action by the House of Delegates or the Council. However, during the past several years, total CMA operating expenses have been very close to the amounts budgeted.

In October, each staff coordinator works with committee chairmen to anticipate the activities of each committee during the next fiscal year. The cost of each project, including meeting and travel expenses, printing, postage, office services and advisory services, is estimated.

In November, the CMA Controller compiles the first draft of a budget based on these committee reports. In addition to committee expenses, he takes into consideration "fixed expenses"—office equipment and supplies, rent, insurance premiums, staff salaries, taxes and maintenance.

This first draft of the budget is presented to the Finance Committee in late November or early December. The committee reviews the budget, item by item, and decides what items will require Council policy decisions. These policy questions are presented and resolved at the December or January meeting of the Council, and a second draft of the budget is developed. The Finance Committee reviews the second draft and presents it to the Council for further consideration at the February meeting.

Between the February meeting of the Council and the meeting of the House of Delegates, the Finance Committee and members of the House of Delegates Reference Committee on Finance (No. 2) meet together to review the budget. Copies of the proposed budget are mailed to members of the

---

This article was prepared by the Finance Committee of the Council of the California Medical Association.

JAMES C. MacLAGGAN, M.D. . . . . President
JOHN G. MORRISON, M.D. . . . . President-Elect
WILLIAM F. QUINN, M.D. . . . . Speaker
JOSEPH W. TELFORD, M.D. . . . . Vice-Speaker
CARL E. ANDERSON, M.D. . . . Chairman of the Council
ALBERT G. MILLER, M.D. . . Vice-Chairman of the Council
HELEN B. WEYRAUCH, M.D. . . . . Secretary
DWIGHT L. WILBUR, M.D. . . . . Editor
HOWARD HASSARD . . . . Executive Director
General Office, 693 Sutter Street, San Francisco 94102 • 776-9400
ED CLANCY . . . . Southern California Office
1515 N. Vermont Avenue, Los Angeles 90027 • 663-8071

House of Delegates at least 30 days before the House meets.

During the Annual Session, Reference Committee No. 2 studies the proposed budget again, and holds open hearings. After the hearings, the Reference Committee presents its recommendations to the House of Delegates, which then votes on the budget and the committee's recommendations.

Thus, the amount of money needed to carry out the programs and accomplish the goals of the CMA is determined, and from this, the level of dues is established by the House of Delegates.

The Finance Committee and the CMA Controller are responsible for cost control and analysis.

Expenditures are reviewed throughout the year by members of the staff and the Finance Committee.

At the conclusion of each fiscal year, an independent firm of Certified Public Accountants audits the CMA financial records. Copies of the auditor's certified statements are given to councilors and members of Reference Committee No. 2, and the statements are published in the pre-convention issue of California Medicine.

In Memoriam

Brown, William Edward, Los Angeles. Died 25 November 1966, in Los Angeles, aged 65, of pneumonia. Graduate of the University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, 1932. Licensed in California in 1933. Doctor Brown was a member of the Los Angeles County Medical Association.

✣

Dieterle, Karl Lyonal, Los Angeles. Died 6 November 1966, in Los Angeles, aged 77, of carcinomatosis. Graduate of the College of Physicians and Surgeons, Medical Department, University of Southern California, Los Angeles, 1914. Licensed in California in 1914. Doctor Dieterle was a member of the Los Angeles County Medical Association.

✣

Goodman, Martin, North Hollywood. Died 3 December 1966, in North Hollywood, aged 42, of coronary artery disease. Graduate of the University of Vermont College of Medicine, Burlington, 1953. Licensed in California in 1956. Doctor Goodman was a member of the Los Angeles County Medical Association.

✣

Griner, Ernest C., Stockton. Died 30 October 1966, near Ely, Nevada, aged 72, of heart disease. Graduate of Hahnemann Medical College of the Pacific, San Francisco, 1917. Licensed in California in 1917. Doctor Griner was a member of the San Joaquin County Medical Society.

✣

Heilbronn, Alexander, Oakland. Died 25 November 1966, in Berkeley, aged 67, of acute pulmonary edema, acute extensive myocardial infarct, coronary artery atherosclerosis. Graduate of Hamburgische Universitat Medizinische Fakultät, Hamburg, Germany, 1924. Licensed in California in 1940. Doctor Heilbronn was a member of the Alameda-Contra Costa Medical Association.

✣

Horswill, Harry Lee, Tulare. Died 2 December 1966, aged 57. Graduate of the University of Oregon Medical School, Portland, 1936. Licensed in California in 1937. Doctor Horswill was a member of the Tulare County Medical Society.

✣

Johnson, William T., Alhambra. Died 5 November 1966, in Pasadena, aged 70, of acute myocardial infarction. Graduate of the University of Nebraska College of Medicine, Omaha, 1921. Licensed in California in 1927. Doctor Johnson was a member of the Los Angeles County Medical Association.

✣

Katz, Benjamin, Los Angeles. Died 6 November 1966, in Los Angeles, aged 83, of chronic and congestive heart failure. Graduate of First Moscow Medical Institute, Moscow, R.S.F.S.R., Russia, 1909. Licensed in California in 1922. Doctor Katz was a member of the Los Angeles County Medical Association.

Krull, Frank, Sacramento. Died 5 December 1966, in Sacramento, aged 93. Graduate of Cooper Medical College, San Francisco, 1900. Licensed in California in 1900. Doctor Krull was a member of the Sacramento County Medical Society, a life member of the California Medical Association, and a member of the American Medical Association.

✣

Lachman, Harry, Santa Monica. Died 1 December 1966, in Pacific Palisades, aged 61, of coronary artery disease. Graduate of the University of Maryland School of Medicine, Baltimore, 1931. Licensed in California in 1957. Doctor Lachman was a member of the Los Angeles County Medical Association.

✣

Lee, Lawrence Dewey, Glendale. Died 21 November 1966, aged 68, of arteriosclerosis and diabetes. Graduate of the University of Nebraska College of Medicine, Omaha, 1926. Licensed in California in 1936. Doctor Lee was a member of the Los Angeles County Medical Association.

✣

McCurdy, Theodore E. A., Berkeley. Died 25 November 1966, in Berkeley, aged 89. Graduate of Leonard Medical School, Raleigh, North Carolina, 1904. Licensed in California in 1923. Doctor McCurdy was a retired member of the Alameda-Contra Costa Medical Association and the California Medical Association, and an associate member of the American Medical Association.

✣

Moore, E. Nelson, Hollister. Died 22 November 1966, in Hollister, aged 65, of heart disease. Graduate of McGill University Faculty of Medicine, Montreal, Quebec, Canada, 1927. Licensed in California in 1941. Doctor Moore was a member of the San Benito County Medical Society.

✣

Pizey, Bryce A., San Pedro. Died 11 November 1966, in Harbor City, aged 53. Graduate of the College of Osteopathic Physicians and Surgeons, Los Angeles, 1936. Licensed in California in 1936. M.D. degree from California College of Medicine, 1962. Doctor Pizey was a member of the Los Angeles County Medical Association.

✣

Smith, Arthur E., Los Angeles. Died 18 November 1966, in Beverly Hills, aged 79, of coronary thrombosis. Graduate of Loyola University School of Medicine, Chicago, Illinois, 1918. Licensed in California in 1922. Doctor Smith was a member of the Los Angeles County Medical Association.

✣

Stewart, David, Covina. Died 21 November 1966, in Los Angeles, aged 45, of carcinoma of bile duct. Graduate of the University of Southern California School of Medicine, Los Angeles, 1953. Licensed in California in 1953. Doctor Stewart was a member of the Los Angeles County Medical Association.

STILLWELL, TYLER C., Oxnard. Died 16 November 1966, in Ventura, aged 49. Graduate of the University of Colorado School of Medicine, Denver, 1943. Licensed in California in 1946. Doctor Stillwell was a member of the Ventura County Medical Society.

✤

THOMAS, ROY E., Los Angeles. Died 26 October 1966, in Los Angeles, aged 85, of arteriosclerosis. Graduate of Rush Medical College, Chicago, Illinois, 1907. Licensed in California in 1914. Doctor Thomas was a member of the Los Angeles County Medical Association.

✤

WELCOME, EARL H., National City (San Diego County). Died in October 1966, aged 84. Graduate of Ensworth Medical College, St. Joseph, Missouri, 1903. Licensed in California in 1922. Doctor Welcome was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

application for HOTEL ACCOMMODATIONS

NINETY-SIXTH *Annual Session*

CALIFORNIA MEDICAL ASSOCIATION • APRIL 15-19, 1967

BILTMORE HOTEL, LOS ANGELES

House of Delegates Opening Session Saturday evening, April 15; Scientific Programs begin Sunday morning, April 16.

1. Fill in the form below **completely** for room accommodations at the CMA's 1967 Annual Session. There is only a limited number of rooms available. Your choice of accommodations will be better if your request is for rooms to be occupied by two or more persons.
2. Your reservation request should include the definite date and hour of your arrival and departure.
3. All reservations must be made through the CMA Housing Bureau, 693 Sutter Street, San Francisco, California 94102.
4. **DEADLINE** for housing: April 1, 1967.

HOTEL ROOM RATES*

	Single	Double and Twins	Suites
BILTMORE HOTEL			
515 So. Olive Street	10.50-17.50	15.50-21.50	30.00-55.00
MAYFLOWER HOTEL			Triples
535 So. Grand Avenue	8.50-13.50	12.00-17.50	22.00
STATLER-HILTON			Suites
930 Wilshire Boulevard	11.00-17.50	16.00-22.50	30.00-65.00

*Rates subject to change

Send to: California Medical Association—*Housing Bureau*
693 Sutter Street, San Francisco, California 94102

Please reserve the following accommodations for the CMA's 1967 Annual Session in Los Angeles, April 16-19, 1967. The first meeting of the House of Delegates begins Saturday evening, April 15; Scientific Programs begin April 16.

Single Bedroom $.................. Twin-Bedded Room $.................. Double Bedroom $..................

Small Suite $.................. Large Suite $.................. Other $..................

First Choice Hotel.................. Second..................

Arrival (date) .... Hours.................. a.m. p.m. Departure (date) .... Hour.................. a.m. p.m.

THE NAME OF EACH HOTEL GUEST MUST BE LISTED. Include the names and addresses of both persons for each twin-bedded or double room, and names and addresses of all other persons for whom you are requesting reservations.

....

....

....

....

—PLEASE PRINT—

Your Name .... Officer? ...... Delegate? ...... Alternate? ...... Speaker? ......

Address .... County ....

City and State.................. Zip No. ....

CALIFORNIA MEDICAL ASSOCIATION

1967 ANNUAL SCIENTIFIC ASSEMBLY

Biltmore Hotel • Los Angeles • April 15-19

Plan NOW to Attend

Three General Sessions on

ADVANCES IN DIAGNOSTIC TECHNIQUE

DISTINGUISHED SPEAKERS INCLUDE:

- ★ William A. Altemeier, M.D., Chief, Department of Surgery, University of Cincinnati College of Medicine, Cincinnati
- ★ Irvine H. Page, M.D., Director, Research Center, Cleveland Clinic, Cleveland
- ★ Herbert M. Stauffer, M.D., Professor and Chairman, Diagnostic Section, Department of Radiology, Temple University Science Center, Philadelphia
- ★ Jon V. Straumfjord, Jr., M.D., Professor and Chairman, Department of Clinical Pathology, Medical College, University of Alabama, Birmingham
- ★ George Zur Williams, M.D., Chief, Clinical Center, National Institutes of Health, Bethesda

SPECIAL CONFERENCES ON:

Cancer of the Colon and Rectum

Research Advances in Veterinary Medicine

The Impact of Medicare—Titles 18 and 19

Medicine and Religion—Moral Aspects of New Procedures

PLUS . . .

EIGHTEEN SCIENTIFIC SECTION PROGRAMS

SCIENTIFIC AND TECHNICAL EXHIBITS

COLOR TV AND FILM SYMPOSIA

PUBLIC HEALTH REPORT

Lester Breslow, M.D., M.P.H.
Director, State Department of Public Health

Despite California's rapid population increases, planning and development of water resources have met the pressing demands for domestic water supplies—a truly remarkable accomplishment. An equally remarkable accomplishment has been the continued safety of the water delivered to the bulk of California's population.

Further, the public health problems to be dealt with have multiplied out of proportion to the population spiral. This population acceleration, coupled with growing urbanization and increasing recreational activities, has created problems of producing and delivering a domestic water supply of high quality. In addition, public expectations for good quality water have increased significantly.

Surface water sources are being exposed to more potential sewage hazards owing to increased recreational activities and increased population. Of active concern to the State Department of Public Health are proposed small community sewage disposal projects in the upper watershed areas, heavy recreational activities on mountain streams and lakes, recreation on water supply reservoirs and enormous construction activities throughout the mountain areas.

In other areas of the state, urban development is encroaching upon local surface water sources and upon the thousands of domestic water wells located in highly urbanized areas. In addition to the encroachment upon surface water sources, ground water supplies are in a state of rapid change brought about by increasing water demands, lowering of ground water basins and recharge practices.

The staff of the department's Bureau of Sanitary Engineering has been alert to these problems and has been actively engaged in promoting high quality, safe and potable water supplies for Californians.

Urgent problems which confront all of the department's environmental health units include:

1. The rapid proliferation of sewage reclamation water projects, some of which have serious implications with regard to the safety of ground water sources.
2. The presence of chironomid larvae, nematodes, chronic high coliform counts, mineral content and taste and odor problems in four of the state's major water systems.
3. The transition of a substantial number of irrigation systems to domestic service.
4. The accidental spraying of pesticides over the raw water basins of domestic water systems.

Californians are served domestic water by some one thousand large water systems and more than four thousand small systems, in addition to numerous individual water sources that serve restaurants, resorts, parks, roadside establishments and isolated industrial premises.

Of particular concern to the department are the small water systems, many of which are becoming overtaxed by the demands of burgeoning residential populations, and which must expand their facilities.

The expansion of small systems often results in the development of problems which are difficult to correct and often stem from the lack of financial backing and capital investment.

Typical of the problems are water mains too small to provide adequate pressure, lack of sufficient water sources to meet demand, treatment units operating at excessive rates, dilapidated or nearly worn out equipment and poorly trained operating personnel.

The Bureau of Sanitary Engineering has joined with local health departments to upgrade the small water systems by providing consultation services and by conducting training programs for system personnel.

In all phases of water supply—source, treatment, and distribution—more intense problems are appearing. The Public Health Department has stepped up its activities to keep abreast of water quality needs. Thus far, we have been able to maintain a reasonably high degree of safety. Our efforts to maintain higher water quality standards have been accelerated, and programs for the continuance of surveillance to improve the more than five thousand water systems which serve the population have high departmental interest and priority.

INFORMATION

Statutory Consent for Chemical Test of Alcoholic Content of Blood, Breath or Urine in Drunk Driving Cases

HOWARD HASSARD, LL.B.*

THE CALIFORNIA LEGISLATURE, in 1966, amended the Vehicle Code (sections 13353 and 13354) to provide for a chemical test for sobriety in drunk driving cases. This article will describe the provisions of the law which are of interest to physicians, nurses and clinical laboratory technologists.

There is no indication that the legislature intended to change the laws regulating the practice of medicine. Its intent was to deal specifically with the problem of consent in connection with chemical tests for sobriety in drunk driving cases.

The new law has sometimes been referred to as the "Implied Consent Law." This title is somewhat misleading, for the person arrested may refuse to submit to the test (and, as a consequence of his refusal, take a six-month suspension of his driver's license); and if he does agree to take a test, he has an absolute right to choose whether it will be of his blood, his breath or his urine. Conversely, the officer in charge has a duty to provide whichever basic type of test the arrested person may select. An understanding of what the law is and what it is not is important to physicians, nurses and technologists.

Section 13353 provides that any person who drives a motor vehicle on a highway "shall be deemed to have given his consent to a chemical test of his blood, breath or urine" if he is arrested for committing an offense while driving the vehicle on the highway, under the influence of intoxicating liquor. The chemical test must be incidental to the arrest and administered at the direction of the "peace officer" or at the request of the person arrested.

A statement in writing by the peace officer that the person is under lawful arrest for a drunk driving offense on the highway and that this requested chemical test is incidental to the arrest, may be accepted by a physician, nurse or technologist, as a correct statement of fact.

As has been stated, the person arrested has the right to choose whether the test shall be of his blood, breath or urine. However, "any person who is dead, unconscious" or otherwise incapable of refusing to give consent or choose the type of test to be administered "shall be deemed *not* to have withdrawn his consent."

A blood test may *not* be required of:

(a) any person afflicted with hemophilia, or

(b) any person afflicted with a heart condition and using an anticoagulant under the direction of a physician.

It is recommended that if a conscious person states that he has either condition, he be given the breath or urine test rather than a blood test. An unconscious person should be checked to see that he does not have a "Medic-Alert" or similar identification stating he has either of these conditions. The physician, nurse or technologist who draws a blood specimen from a conscious or unconscious person should be sure bleeding has stopped before dismissing him from observation.

The taking of a breath or urine sample does not, in any way, constitute assault and battery by a physician, nurse or technologist. *Further, these samples will ordinarily be collected by the peace officer.* As a matter of general interest, the law requires that "the person tested shall be given such privacy in the taking of the urine specimen as will insure the accuracy of the specimen and, at the same time, maintain the dignity of the individual involved."

"Only a physician, registered nurse or duly licensed clinical laboratory technologist or clinical laboratory technician *acting at the request of a peace officer* may withdraw blood for the purpose of determining the alcoholic content therein" (Section 13354(a)).

---

*Of Peart, Hassard, Smith and Bonnington, General Counsel, California Medical Association.

Reprint requests to: California Medical Association, 693 Sutter Street, San Francisco 94102 (William Whelan, Associate Executive Director).

"No physician, registered nurse or duly licensed clinical laboratory technologist or clinical laboratory technician *shall incur any civil or criminal liability as a result of the proper administering of a blood test when requested in writing* by a peace officer to administer such a test" (Section 13354(d)).

Drawing of blood involves the penetration of tissue. Penetration of tissue is defined as a practice of medicine for which one needs a Physician's and Surgeon's Certificate—Business and Professions Code Section 2137. In the case of *Magit v. Board of Medical Examiners,* 57 Cal.2d 74 (1961), the court said that a licensed nurse, when acting under the direction and supervision of a licensed physician, is permitted to perform certain tasks which without such direction and supervision would constitute the illegal practice of medicine or surgery. Section 1242 of the Business and Professions Code authorizes a licensed clinical technologist to "perform venipuncture or skin puncture for test purposes upon the specific authorization" of a person licensed to practice the healing arts, such as a physician.

The law does not alter the usual professional liability principle as it relates to alleged negligence. The exemption from liability provision applies where there is "proper" administration of a blood test. There is no exemption for negligent administration of a blood test.

A registered nurse or laboratory technologist would perform the technique under the order of a physician or under written standing orders adopted by the hospital or organized agency after appropriate medical consultation.

If a physician, nurse or technologist, while performing the technique, observes medical or pathological symptoms indicating that the arrested person requires medical attention, the arresting officer should be so informed and a record made of this fact.

Section 1292 of the Business and Professions Code requires that laboratory tests in a clinical laboratory be made pursuant to the request of a person licensed under the provisions of the Business and Professions Code relating to the healing arts. A peace officer is not authorized to request a laboratory to make a blood or urine test. It would be legal and proper under the act for a physician to request a licensed laboratory to make a test of blood, breath or urine, when requested to do so by a peace officer.

The law permits the arrested person to request that an independent chemical test of blood, breath or urine be made at his own expense. It is our opinion that this is an absolute right. It is also provided that an arrested person may have the results of the test made available to him or his attorney.

Penal Code Section 817 defines a peace officer. It is a long and involved definition. For practical purposes, the peace officer most often involved with drunk driving violations on the highway will be a sheriff or deputy sheriff, a city or town police officer or a member of the California Highway Patrol. Such an officer may be identified by his name and badge number and will ordinarily be attired in a distinctive uniform with the insigne or name of his organization easily visible.

The law specifically exempts the physician, registered nurse or licensed clinical laboratory technologist from "civil or criminal liability" for the proper administration of a blood test when requested in writing by a peace officer. Attorneys for the California Hospital Association have advised hospitals that since a hospital is not specifically mentioned in the law, it would be prudent for them to require the written consent of the arrested person upon admittance. This would avoid later controversy concerning whether the person was exempt because he was afflicted with hemophilia or a heart condition, or whether the arrested person had chosen the blood test rather than a test of breath or urine, or had positively refused to authorize any test.

On the other hand, the California Attorney General's office feels that the place where the sample is taken—such as a hospital—would have implied immunity.

In summary, several conditions should be observed for the terms of the statute to apply:

1. A peace officer must request a blood, breath or urine sample in writing incident to an arrest.

2. A person must have been lawfully arrested for driving on the highway and committing an offense while under the influence of intoxicating liquor.

3. The person must have been permitted to choose between test of the blood, the breath or urine.

4. The arrested person may not be required to give a blood sample if he has hemophilia or a heart condition and is using an anticoagulant under the direction of a physician.

5. The taking of the blood sample must be done by a physician or at the request or authorization of a physician. When done by a registered nurse or a technologist in a hospital or other facility without the presence of a physician, written criteria should have been established for guidance.

6. The chemical test of the blood or urine sample can only be performed in a licensed laboratory at the request of a physician.

ACKNOWLEDGMENT: The author acknowledges, with appreciation, the assistance of William Whelan, Associate Executive Director, California Medical Association, in the preparation of this article.

Socialism In Saskatchewan: A Fiasco

(From an address by the Hon. W. Ross Thatcher, premier of Saskatchewan, before the Edison Electric Institute in San Francisco, June 8. Published in PG and E Progress, 43: October 1966. Reprinted with permission.)

SASKATCHEWAN FOR 20 YEARS—from 1944 to 1964—had a Socialist government—about the only one in North America, except Castro's.

In 1944, the Socialists said they would solve the unemployment problems by building government factories. They promised to use the profits to build highways, schools, hospitals and to finance better social welfare measures generally. Over the years they set up 22 so-called crown corporations. I wish that time permitted me to tell you of the fiasco that followed.

By the time we had taken over the government, 24 months ago, 12 of the crown corporations had gone bankrupt or been disposed of. Others were kept operating by repeated and substantial government grants.

During the whole period the Socialists waged war against private business. The making of profits was condemned as an unforgivable sin. What was the result? Investors simply turned their backs on the Socialists. Dozens of oil companies pulled up stakes and moved out. Gas exploration ground to a complete halt. Prospecting in our vast north became almost non-existent.

During the period Canada was experiencing the greatest economic boom in her history, Saskatchewan received only a handful of new factories. After 18 years of socialism there were fewer jobs in manufacturing than existed in 1945—this despite the investment of $500 million in crown corporations.

The Socialists promised a greatly expanded program of social welfare measures: there was to be "free" medical care, "free" hospitalization care, "free" drugs and so on. The money to finance these projects was to come from the profits of the crown corporations. Of course, in the over-all picture, there were no profits—rather there were colossal losses. Thus the welfare program had to be financed from taxation.

During the period more than 600 completely new taxes were introduced; 650 other taxes were increased. "Per capita" taxes in Saskatchewan were soon substantially out of line with our sister provinces—one more reason why industry located elsewhere.

Twenty years ago the Socialists promised to make Saskatchewan a Mecca for the working man. Instead, we saw the greatest mass exodus of people out of an area since Moses led the Jews out of Egypt. Since the war, 270,000 of our citizens left Saskatchewan to find employment elsewhere.

Finally our people decided they had been the Canadian guinea pig for socialism long enough. They threw them out.

It is the task of our new government to prove in the next few years that the private enterprise system can do more for our people than socialism.

If there are any Americans who think that socialism is the answer, I wish they would come to Saskatchewan and study what has happened to our province.

NEWS & NOTES

NATIONAL • STATE • COUNTY

LOS ANGELES

The eighth annual postgraduate course on **Cardiopulmonary Function and Physiology,** sponsored by the Council on Postgraduate Education of the American College of Chest Physicians will be held 6 to 10 February at the Ambassador Hotel, Los Angeles. For further information write American College of Chest Physicians, 112 East Chestnut Street, Chicago, Illinois.

* * *

Cedars-Sinai Medical Center is recipient of a $195,000 grant from the U.S. Public Health Service to establish a five-year **training program in cardiology,** it is announced by Dr. David Littauer, executive director.

Training will include intensive care, cardiac catheterization and cardiac physiology. Two resident physicians will participate for each of the first two years and three for each of the remaining three years.

SAN FRANCISCO

A total of 245 years of service in the field of medicine ended last month with the **retirement of five eminent San Francisco physicians** from the staff of **Southern Pacific Memorial Hospital.**

Bringing a close to their long and distinguished careers are 83-year-old Dr. Bernard Kaufman, Sr., the hospital's medical superintendent since 1948; 83-year-old Dr. Lloyd B. Crow, chief radiologist since 1921; 74-year-old Dr. Wilber F. Swett, chairman of the Department of Ophthalmology since 1932; 82-year-old Dr. Harold F. Unsinger, director of the Outpatient Department and assistant medical superintendent, and Dr. Thomas E. Gibson, 70, chief of the Department of Urology.

The five physicians were honored by the Southern Pacific Company and the Southern Pacific Memorial Hospital at a dinner 13 December.

* * *

J. Englebert Dunphy, professor and chairman of the Department of Surgery at the University of California San Francisco Medical Center, was recently elected **president of the International Federation of Surgical Colleges.** He succeeds Sir Harry Platt of the United Kingdom who had been president since 1958. Elections were held during the Federation's meeting in Warsaw, Poland.

Dr. Dunphy, who has been a member of the faculty of the School of Medicine, San Francisco, since 1964 is a world authority on tissue healing and has maintained his research activities throughout his academic and administrative career.

* * *

Chancellor Willard C. Fleming and Stuart C. Cullen, Dean of the School of Medicine, recently announced the appointment of **Dr. Walther Stoekenius** as professor of cell biology at the University of California San Francisco Medical Center. Dr. Stoekenius, a distinguished cell biologist, will be director of the Cardiovascular Research Institute's new Electron Microscope Laboratory. He will also be a member of the Department of Microbiology in the School of Medicine.

* * *

Dr. Donald L. Fink, assistant professor of pediatrics at the University of California San Francisco Medical Center, received the Ross Pediatric Education Award during the recent meeting of the Western Society for Pediatric Research. The award was given to Dr. Fink in recognition of his contributions to teaching and for his organization of the Pediatric Outpatient Clinic at the Medical Center as an exemplary teaching unit.

GENERAL

The American Academy of Allergy will present postgraduate courses, one on "Leukocytes" and one on "The Autonomic Nervous System," 18 and 19 February 1967, at the Holiday Inn-Riviera, Palm Springs, California, immediately before the annual meeting of the organization, which will be held at Palm Springs, 20 to 22 February.

Additional information may be obtained by writing to the American Academy of Allergy, 756 North Milwaukee Street, Milwaukee, Wisconsin 53202.

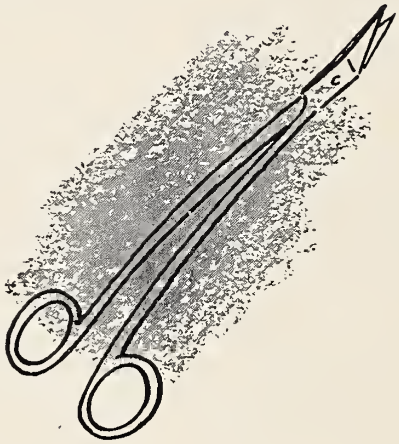
* * *

The Federation of Western Societies of Neurological Science will hold their annual meeting at the El Mirador Hotel, Palm Springs, California 2 to 5 March 1967. The meeting will be highlighted by a symposium on "Behavior of the Nervous System in Tissue Culture," which will take place on Saturday, 4 March from 9 a.m. to 5 p.m.

All inquiries concerning this meeting should be directed to: Theodore Kurze, M.D., Program Chairman, 1200 North State Street, Box 492, Los Angeles, California 90033.

* * *

The Colorado Winter Clinic of the Colorado Medical Society will be held at the Brown Palace Hotel in Denver, 28 February to 3 March. Scientific programs are planned each afternoon. The Yampa Valley Mail train leaves Union Station daily at 9:05 a.m. for Winter Park to accommodate those who wish to attend scientific meetings one day and ski another.



The Physician's BOOKSHELF

SURGERY IN AMERICA—From the Colonial Era to the Twentieth Century, Selected Writings. Edited by A. Scott Earle, M.D. W. B. Saunders Company, Philadelphia and London, 1965. 280 pages, $8.50.

Medical history has a singular fascination and excitement. This is a unique historical odyssey of American surgery. Original articles are presented dating from a 1710 account of the first elective surgical procedure performed in the North American Colonies through Dr. William S. Halsted's classic treatise on "The Training of the Surgeon." During these 200 years there have been many noteworthy contributions to the evolution of American Surgery. The articles comprising this concise volume are but a small percentage of many outstanding surgical achievements. A biographical sketch of each author accompanies his writing. This adds to the historical flavor and significance of the book. There are several articles on pioneering efforts in the field of vascular surgery, as well as early reports on the use of ether, and McBurney's original paper on his operation for appendicitis. It has been said that "the story of surgery's growth in this country has been told—and told well—by the men who contributed most to its progress.

DAVID SACHS, M.D.

* * *

NEURO-OPHTHALMOLOGY—Symposium of the University of Miami and the Bascom Palmer Eye Institute, Volume II—Compiled and edited by J. Lawton Smith, M.D., Associate Professor of Ophthalmology, Associate Professor of Neurosurgery, and Assistant Professor of Neurology, University of Miami School of Medicine, Miami, Florida. Illustrated. The C. V. Mosby Company, St. Louis, 1965, 278 pages.

This symposium edited by J. Lawton Smith does not stimulate the reader except for the chapters on Angiography of the ocular fundus by Edward Norton and techniques of fluorescein fundus photography by Johnny Justice Jr. and Raymond J. Sever. The contents of the rest of the symposium can be found in the latest texts on Neuro-ophthalmology. This is a disappointing fact in view of the rather distinguished list of contributors.

R. M. SINSKY, M.D.

* * *

HEMATOLOGIC PROBLEMS IN THE NEWBORN—Volume IV in the Series: Major Problems in Clinical Pediatrics—By Frank A. Oski, M.D., Assistant Professor of Pediatrics, University of Pennsylvania School of Medicine, and J. Lawrence Naiman, M.D., Assistant Professor of Pediatrics, Temple University School of Medicine. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105. 1966. 294 pages, illustrated, $11.00.

This is the fourth volume of a series entitled, "Major Problems in Clinical Pediatrics," under the general direction of Dr. Alexander Schaffer. The editor's own major interest can be deduced from the fact that three of the four volumes which have appeared so far deal with the newborn infant. The monographs published to date have set a very high standard and I am happy to report that the newest volume is of equal quality. Drs. Oski and Naiman have written a very lucid book which combines a presentation of the latest scientific advances in the field with a very clinical approach to the diagnosis and management of common hematologic problems in the newborn infant. The book provides a good deal of information about normal hematologic values of all sorts, and this alone would make it a most valuable reference book for the newborn nursery. The authors also provide many useful tables listing differential diagnoses, indications for therapy and logical ways of approaching clinical problems. In addition to presenting the normal development and metabolic features of the hematopoietic system, the chapters include a discussion of anemia, disorders of red cell metabolism, disorders of hemoglobin syntheses, erythroblastosis fetalis, coagulation problems, thrombocytopenia and finally, disorders of the white cells. An excellent and up-to-date bibliography completes the volume. The book is illustrated in black and white and the only improvement which comes to mind might be the inclusion of a few illustrations in color. The volume is highly recommended for students, house officers and for every physician who takes care of newborn infants and as a reference volume for every newborn nursery.

* * *

METABOLIC TOXEMIA OF LATE PREGNANCY—A Disease of Malnutrition—By Thomas H. Brewer, M.D., County Physician, Richmond Health Center, Richmond, Calif.; formerly, Instructor, Department of Obstetrics-Gynecology, University of California Medical Center, San Francisco, Calif. Charles C Thomas, Publisher, Springfield, Ill., 1966. 127 pages, $8.50.

In the introduction, the author states: "Scientific evidence will be presented which clearly indicates that toxemia of late pregnancy is a disease of nutritional deficiency mediated through hepatic dysfunction and that the bacterial flora of the maternal gastrointestinal tract, as well as the steroid hormones (estrogens and progesterone) produced by the placenta, play a contributing role in its pathogenesis." Having said that, Dr. Brewer might well have ended his book there, because the diatribe which follows is a somewhat illogical and jumbled polemic containing those bits of the literature, some out of context, which support his thesis together with a petulant rejection of "claims" which he cannot explain on the basis of malnutrition.

Brewer encounters much difficulty explaining such facts as the existence of hypertension, the presence of a renal lesion, the predominance of toxemia in primiparae, the presence of retinal edema, etc., on the basis of malnutrition or hepatic dysfunction. His method of dealing with these problems is both amusing and sad. Apropos of the predilection for primiparity, he writes: "The fact is neglected in such statistical analyses that . . . the large majority of primiparous patients do *not* develop the disease. By thus 'looking on the other side of the coin,' we can immediately challenge the concept that *primi-*

parity, per se, has any significant *physiological* role to play in the pathogenesis of [toxemia]." Using the same logic, he might well have concluded that the fact that a majority of malnourished pregnant women in many areas of the world, literally starved of protein and bordering on starvation, do *not* develop toxemia of late pregnancy rules out malnutrition as a *cause.*

Brewer dismisses the vasoconstriction of toxemia, and hence the hypertension, "as merely a clinical manifestation or end result of a metabolic hepatic disease of nutritional deficiency." The fact that diuresis occurs when human serum albumin is administered or after the patient delivers is evidence to the author that the renal lesion has no primary functional significance. As to the retinal edema described so often by Finnerty, Brewer has this to say: "So carried away with 'salt and water retention' have students of this disease become that they have seen 'mirages' in the retinae of 'toxemic' women and thought they were seeing 'water,' as a child sees such mirages while riding along the hot desert [sic] highway and thinks 'water.' "

In Chapter 10, entitled "Red Herrings in Toxemia Research," the author dismisses those theories which do not attribute a primary role to malnutrition. The concept developed by Ernest Page, for example, that "uterine ischemia" may play an important etiologic role is answered by saying: "We never find in Nature, in toxemic women such clamps on the abdominal aortas nor ligatures on the ovarian and uterine arteries. . . . To keep our interest and attention focused on the uterus and placenta is to remain forever in the dark about the pathogenesis of this disease." He goes on further to relate: "During the year I was associated with Dr. Page at the University of California, I was never able to free his mind of this trap of 'statistical associations.' " This is an interesting statement for one whose main "scientific evidence" in favor of malnutrition as the cause of toxemia is one of statistical association.

In the area of nutrition, it is well recognized by most students of toxemia that the disease is far more common in the impoverished, poorly nourished pregnant women. This treatise, through its contentiousness, may do as much to hinder as to accelerate progress in this highly important area of malnutrition and pregnancy.

* * *

CITANEST®—An International Conference Sponsored by ASTRA, Santos, Brazil, 17 to 18 September 1964—Edited by Sten Wiedling. This work is also published as Supplementum XVI, 1965, to Acta anaesthesiologica Scandinavica. Copies of this edition may be obtained by writing to Astra Pharmaceutical Products, Inc., Worcester, Massachusetts 01606. No price quoted.

This volume contains papers and discussions presented at an International Symposium held in 1964; the assembled panel is truly international consisting of over fifty participants most of whom are familiar, at least in name, to American readers. Outstanding authorities in regional anesthesia practice are well represented by such members as Bromage, Peere Lund, Crawford, Bonica, Sadove to mention a few of the North American participants only. The volume is comprehensive in scope and authorative in its field. The material is presented in four general sections; the first two on pharmacology, experimental and clinical; then a section on clinical toxicity and tolerance, and finally a section on clinical experience and investigation.

Six or eight major papers are presented in each section with general discussions after each two or three papers. The discussion portions are generally well edited although occasional errors of sequence do occur, i.e., a question quoted following an answer, but the knowledgeable reader will easily compensate for these lapses. The discussion sections are extensive and this reviewer formed the early impression that the participants were interested, animated, articulate, well prepared and anxious to take positive stands either in agreement or disagreement. The formal papers are extensive in scope and cover all facets concerned in the use of prilorcaine, the only criticism being the lack of captions to accompany the numerous figures of Peere Lund's first paper. The principal emphasis in this volume is on peridural anesthesia, both surgical and obstetrical. The effectiveness of prilorcaine is thoroughly presented and compared most often with lidocaine with agreement on the lower CNS toxicity of prilorcaine. Other uses of prilorcaine are also covered such as axillary block, topical anesthesia of tracheobronchial tree, spinal anesthesia, dental blocks and in all presentations there are recommendations for accepted safe doses. The occurrence of methemoglobinemia after prilorcaine is extensively reported with unresolved differences of opinion as to its seriousness. Small doses lessen the magnitude of methemoglobinemia but in patients with compromised oxygen transport the advantage of the lower CNS toxicity of prilorcaine must be balanced against a possible further decrease in oxygen transport.

The conclusions of this conference are conservative, sensible and constructive. This volume is useful not only to anesthesiologists but to surgeons or any physicians who use local anesthesia.

ROBERT BETHUNE, M.D.

* * *

SYNOPSIS OF NEUROLOY—Second Edition—Francis M. Forster, B.S., M.D., Professor and Chairman, Department of Neurology, University of Wisconsin School of Medicine, Madison, Wisconsin. The C. V. Mosby Company, St. Louis, 1966. 218 pages, $7.50.

This is defined as a "Synopsis," and it is indeeed a condensed textbook, following the usual type of presentation. History taking, examinations, and clinical entities. If well done, such a synopsis may be of real value to the beginning medical student, revealing how the signs of deranged physiologic function may be observed and used to determine the site or sites of such derangements, and how the natural history of disease entities allows a pathologic diagnosis to be made. This synopsis is designed for such students, but it is often too vague to stimulate careful observation, e.g., ". . . one is particularly careful to query into the review of systems in regard to vision, . . ."; it is too imprecise to spell out dangers, e.g., describing Queckenstedt's maneuver—"The maneuver will not be definitive and may seriously affect the patient's status." There is no discussion of methods of localization.

The index has defects, e.g., Walton's disease is shown in the index, but not referred to in the text and the commonly used eponym, Wilson's disease, is not in the index, but is in the text. The description of epilepsy is good, yet the description of diabetic neuropathy is unclear. The bibliography at the end of each chapter is useful.

This synopsis can be of value to medical students who need a rapid review of neurology, but is not a suitable introduction to neurology.

DONALD MACRAE, M.D.

tions and adverse reactions: The transitory drowsiness which
cur with hydroxyzine HCl usually disappears spontaneously
w days with continued therapy, or is correctable by dosage
on. Dryness of the mouth may be seen with higher doses.
ntary motor activity, including rare instances of tremor and
sions, has been reported, usually on higher than recom-
d dosage. **Hydroxyzine HCl may potentiate barbiturates,
ics such as meperidine, and other CNS depressants.** In con-
e use, dosage for these drugs should be decreased as much
. Because drowsiness may occur, patients should be cau-
against driving a car or operating dangerous machinery.
eral Solution Precautions and contraindications: This dos-
rm is intended only for I.M. or I.V. administration and
not, under any circumstances, be injected subcutaneously
a-arterially. When the usual precautions for I.M. injection
een followed, reports of soft tissue reactions have been
Vhen used intravenously, if given undiluted, minimal amounts
olysis (2-3 grams of liberated hemoglobin) will occur. If
with 50 cc. of normal saline and given during a period of
inutes or more, this phenomenon does not occur. Due to the
and infrequent phlebitis, the rate of injection must not
25 mg. per minute. A single I.V. administration in excess of
. is not recommended. Particular care should be used to
injection only into intact veins; a few instances of digital
ne occurring distal to the injection site have been attributed
dvertent intra-arterial injection or periarterial extravasa-
oth of which should be avoided. **Use in Pregnancy:** When
stered to rats at high dosage, hydroxyzine induced fetal
nalities. Until human clinical data are available adequate
blish safety in early pregnancy, hydroxyzine is contraindi-
n early pregnancy.

Teenagers' Ambition And Uric Acid Levels

A teenager's ambition, such as his drive to go to college, may be affected by the amount of uric acid in his blood.

This is a suggestion of a study of Pittsburgh, Pa., high school boys which appears in the November 14 *Journal of the American Medical Association.*

Several investigations have indicated that uric acid, a waste product of the body's metabolism, positively affects a person's drive, ambition, and achievement. Uric acid has nothing to do with general intelligence, however. High levels of the substance found in some persons' blood is purely a physiological phenomenon.

This latest study by University of Michigan investigators found that, among 62 high school boys, some who attended college did not differ markedly in ability or scholastic achievement from those who did not attend.

The college-attenders, however, did have significantly higher serum urate (uric acid) concentrations. The authors suggest that this is additional evidence of the relationship of serum uric acid levels to achievement-oriented behavior.

An earlier University of Michigan study (JAMA, February 7, 1966), found that the drive, achievement, and leadership of college professors was positively associated with uric acid levels.

The current study began in February 1961, when 138 high school junior and senior-class boys answered a brief questionnaire and contributed blood samples.

Four and a half years later, investigators restudied these boys, and divided them into three groups: those who completed college, those who attended, but did not complete college work, and those who did not attend.

Not surprisingly, many of those in the "completed college" category had received the highest scholastic marks in high school. In the other two groups, however, little difference was found in general ability.

The thing which set apart "attended college" boys from those who did not attend was their significantly higher serum urate concentrations. The authors suggest that these higher levels of uric acid may have boosted ambition.

"Given these findings, it would seem reasonable to argue that the group who attempted college differs from the (non-attending) group—not in ability or scholastic achievement, but in being more highly motivated to get additional education beyond high school," the author said.

The authors emphasize that the study relates only to high school boys, and suggest that serum urate's effects on behavior may relate only to

(Continued on Page 52)

★

Specialized Service

IN

PROFESSIONAL LIABILITY INSURANCE

is a high mark of distinction

THE
MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA
Professional Protection Exclusively since 1899

NORTHERN CALIFORNIA OFFICE:
Gordon C. Jones and John K. Galloway, Representatives
1518 Fifth Avenue, San Rafael, Telephone: 415-453-5140
Mailing Address: P.O. Box 1079, San Rafael 94902
SOUTHERN CALIFORNIA OFFICE:
Davis S. Spencer and Grady T. Dyer, Representatives
Suite 3, 33 East Huntington Drive, Arcadia, Tel. 213-681-5239
Mailing Address: P.O. Box 543, Arcadia 91008

Economical Long-term Therapy for Iron Deficiency Anemia

Tablets & Elixir

FAMOUS
Fergon®
brand of FERROUS GLUCONATE

C.M.A.P. CODE # 5261

BREON LABORATORIES INC.
Subsidiary of Sterling Drug Inc.
90 Park Avenue, New York, N.Y. 10016

Teenagers' Ambition And Uric Acid Levels

(Continued from Page 51)

males. Earlier studies which included girls were inconclusive.

Authors of the report are Stanislav V. Kasl, Ph.D.; George W. Brooks, M.P.H.; and Sidney Cobb, M.D., M.P.H., of the Institute for Social Research, University of Michigan, Ann Arbor.

Poison Antidote Information Is Readily Available to M.D.'s

When a child or adult is accidentally poisoned, the physician sometimes has the problem of quickly locating an antidote. The Public Health Service reports that this emergency arose nine times in the first six months of last year in the case of the drug, Deferoxamine.

Deferoxamine is being investigated as an antidote in acute iron poisonings among young children. However, the drug can be obtained only from a selected group of physicians who are investigating Deferoxamine under rigidly controlled conditions prescribed by the Food and Drug Administration.

As an illustration of the problem: Personnel at Baptist Hospital in Scottsdale, Arizona, had an urgent need for Deferoxamine for treating Joseph Babcock, age two. Joseph had consumed 50 tablets of a medicine heavy in iron content.

Leonard D. Gordon, pharmacist at the hospital, telephoned the Public Health Service's National Clearinghouse for Poison Control Centers to find where to obtain the drug. He reached Dr. John Crotty of the Clearinghouse at his home. The Clearinghouse, operated by the Public Health Service's Division of Accident Prevention in Arlington, Virginia, maintains a 24-hour consultation service to poison control centers.

Dr. Crotty told Mr. Gordon that the nearest supply was available at Albuquerque, New Mexico. Asked to assist, the Air Force flew the drug from Albuquerque to Scottsdale. The patient was treated and recovered.

The National Clearinghouse, which provides information on prevention and treatment of accidental poisonings to more than 550 poison control centers, had anticipated the problem of meeting a demand for Deferoxamine following publication of an article on the drug in 1963.

After a conference with the manufacturer, the names, addresses, and telephone numbers of physician investigators with a supply of the drug were supplied to the Clearinghouse. The Clearinghouse then notified all members of the Poison Control Center Network that information on sources of supply would be available at all times.

For information on Poison Information Centers in California, see listing on Page 72 of the advertising section.

what time is it?

For the past
two years
there's been
one new case
of active tuberculosis
reported for every
four thousand
of U.S. population.

it's time to tine.

Tuberculin, Tine Test
(Rosenthal) Lederle

Available in 5's and 25's.
Order now
from your pharmacist
or your Lederle
representative.

LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York Lederle
414-6—4046R

CLASSIFIED ADVERTISEMENTS

(Continued from Page 70)

REAL ESTATE FOR SALE

CONVALESCENT HOSPITAL SITE—NOVATO, CALIFORNIA: Choice five and one-half acre parcel with use permit. Ready for immediate development. Located 28 miles north of San Francisco, a growing community of approximately 28,000. Just one block from general hospital and new medical building. All utilities available. Priced low at $106,500. Tankersley Realty, 1106 Grant Avenue, Novato, California, or phone (415) 892-5008.

VACATIONS

SIX-DAY NORTHWEST SAILING VACATION aboard new, clean 60′ ketch all through the 172 Evergreen San Juan Islands. Learn to sail, fish for king salmon, gather oysters and clams—REST and RELAX—not over six guests, four may have exclusive. $150 per person pays for everything. See March 1965 California AAA magazine, MOTORLAND, for color-picture story, or write for picture brochure and name of doctor near you who has been on my cruise. Chris Wilkins, Orcas Island, Washington.

PHYSICIAN PLACEMENT SERVICE

The California Medical Association offers free placement assistance through the Physician Placement Service, 693 Sutter Street, San Francisco, California 94102. This service is for the use of all physicians seeking practice opportunities in California and for CMA members who are seeking an assistant or associate. A bulletin is published every other month. Opportunities in the Bulletin must be sponsored by a CMA member, an accredited California hospital or responsible persons in a California community. The Bulletin does not carry listings describing real estate for sale or office space for rent or lease or blind listings with box numbers.

POISON INFORMATION CENTERS

The location and telephone numbers (with area code in parentheses) of the seven official Poison Information Centers in California are listed below:

Fresno
Fresno Community Hospital (209) 233-0911; Night 233-7547

Los Angeles
Children's Hospital of Los Angeles (213) 664-2121

Oakland
Children's Hospital of the East Bay, Oakland (415) 654-5600
Alameda-Contra Costa Medical Association, Oakland (415) 652-8171

Orange
Orange County General Hospital, Orange (714) 633-9393, Ext. 273

San Francisco
Children's Hospital, San Francisco (415) 221-1200

San Jose
Santa Clara County Hospital, San Jose (408) 293-0262, Ext. 318 or 319; or 292-3141

Have you made your hotel reservation for the 1967 CMA Annual Session? You will find an application for Hotel Accommodations on Page 72 in the advertising section.

REFERENCES AND REVIEWS

Avoidable Mortality—A. M. Burgess, Jr. (25 Shattuck St., Boston), T. Colton, and O. L. Peterson, Arch. Environ. Health, 13:794-798 (December) 1966.

Passage of recent legislation concerning heart disease, cancer, and stroke has stimulated consideration of the merits of regional programs of medical care and has produced discussion as to whether or not the three disease groups represent the most effective expenditure of funds directed toward reduction of death rates. In many instances it appears that special provisions for treatment will be less effective than programs of prevention. Since losses in the productive years are particularly disadvantageous, the prominent categories of death in the young—infant mortality and accidental death—must not be overlooked. Reduction of the former, in particular, is not beyond the reach of presently available techniques.

* * *

Local Anesthesia for Peroral Endoscopy—J. R. Gaskill (Univ. of California Medical Center, Los Angeles), and D. R. Gilles, Arch. Otolaryng., 84:654-656 (December) 1966.

A technique is described for local anesthesia in endoscopy which utilizes superior laryngeal nerve block and topical nerve anesthesia of the pharynx and trachea. The procedure requires less time, reduces patient discomfort, and eliminates the possibility of toxic overdosage. In over 150 patients, anesthesia was uniformly satisfactory, and there were no complications attributable to the technique. It should not be used where the needle might pass through carcinoma or infected tissue.

* * *

Treatment of Enterococcal Endocarditis—W. Stille (I. Medizinische Universitätsklinik, Ludwig-Rehn-Str. 14, Frankfurt/Main, Germany), and W. Mondorf, Deutsch Med. Wschr., 91:1997-2002 (November 11) 1966.

Eight patients were treated for enterococcal endocarditis; despite intensive chemotherapy three died. In three patients ristocetin effected a cure, after other antibiotics had failed. In three other patients treatment with ampicillin resulted in a bacteriologically confirmed cure. High doses of ampicillin (10 to 20 gm daily), combined with streptomycin and probenecid, are the treatment of choice in enterococcal endocarditis. The importance of serial bacteriological controls is stressed.

* * *

Effect of Hypocapnia on Cardiac Output During Anesthesia—R. A. Theye, J. H. Milde, and J. D. Michenfelder (Section of Anesthesiology, Mayo Clinic and Mayo Foundation, Rochester, Minn.), Anesthesiology, 27:778-782 (November-December) 1966.

Hemodynamic and blood gas studies were carried out during anesthesia and operation during hypocapnia and normocapnia in nine patients. Without exception, cardiac output was greater during normocapnia than during hypocapnia. The available evidence suggests the presence of decreased myocardial contractility or distensibility, or both, during hypocapnia. These changes are believed to result from altered activity of the sympathetic nervous system.

"Yes, Doctor, the pain is gone."

■ Despite introduction of synthetic substitutes, efficacy of 'Empirin' Compound with Codeine remains unchallenged.

'Empirin'® Compound with Codeine Phosphate gr. ½ No. 3
Each tablet contains: Codeine Phosphate gr. ½ (Warning—May be habit forming), Phenacetin gr. 2½, Aspirin gr. 3½, Caffeine gr. ½.

Keeps the Promise of Pain Relief

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N. Y.

Officers OF THE CALIFORNIA MEDICAL ASSOCIATION

General Officers

President: JAMES C. MACLAGGAN, 525 Hawthorn Street, San Diego 92101. 714 233-6519
President-Elect: JOHN G. MORRISON, 333 Estudillo, San Leandro 94577. 415 483-1332
Past President: RALPH C. TEALL, 2626 L Street, Sacramento 95816. 916 448-1851
Speaker of the House of Delegates: WILLIAM F. QUINN, 1401 S. Hope Street, Los Angeles 90015. 213 749-0181
Vice-Speaker of the House of Delegates: JOSEPH W. TELFORD, 3255 Fourth Avenue, San Diego 92103. 714 295-5151
Chairman of the Council: CARL E. ANDERSON, 1150 Montgomery Drive, Santa Rosa 95405. 707 546-1922
Secretary: HELEN B. WEYRAUCH, 450 Sutter Street, San Francisco 94108. 415 392-6909
Editor: DWIGHT L. WILBUR, 655 Sutter Street, San Francisco 94102. 415 673-4080

Executive Director: HOWARD HASSARD, 693 Sutter Street, San Francisco 94102. 415 776-9400
General Counsel: PEART, HASSARD, SMITH and BONNINGTON, 44 Montgomery Street, San Francisco 94104. 415 982-8585

* * *

District Councilors

FIRST DISTRICT—Roger C. Isenhour, (1967), 2881 Fourth Avenue, San Diego 92103. San Diego County. 714 295-2137
SECOND DISTRICT—Imperial, Inyo, Mono, Orange, Riverside and San Bernardino Counties—
  Office No. 1—Henry V. Eastman (1967), 661 W. First Street, Tustin 92680. 714 544-4560
  Office No. 2—Frank C. Melone (1968), 527 N. Palm Avenue, Ontario 91761. 714 986-1104
THIRD DISTRICT—Los Angeles County—
  Office No. 1—Malcolm C. Todd (1968), 2865 Atlantic Avenue, Long Beach 90806. 213 426-1355
  Office No. 2—Elmer F. Gooel (1969), 465 N. Roxbury Drive, Beverly Hills 90210. 213 271-5745
  Office No. 3—Richard L. Taw (1967), 3741 Stocker Street, Los Angeles 90008. 213 293-7157
  Office No. 4—Lewis T. Bullock (1968), 3875 Wilshire Boulevard, Los Angeles 90005. 213 382-2209
  Office No. 5—Joseph P. O'Connor (1969), 595 E. Colorado Boulevard, Pasadena 91101. 213 792-2154
  Office No. 6—Franklin F. Ham (1967), 14556 Hamlin Street, Van Nuys 91401. 213 786-5711
  Office No. 7—Wilbur G. Rogers (1968), 1122 N. Brand Boulevard, Glendale 91202. 213 241-3125
  Office No. 8—Jean F. Crum (1969), 8355 E. 3rd Street, Downey 90241. 213 869-1091
  Office No. 9—Joseph F. Boyle (1967), 1136 W. Sixth Street, Los Angeles 90017. 213 482-9891
FOURTH DISTRICT—Joseph F. Maguire (1967), 2755 Loma Vista Road, Ventura 93003. San Luis Obispo, Santa Barbara and Ventura Counties. 805 648-2567
FIFTH DISTRICT—Ralph W. Burnett (1969), 515 Truxton Avenue, Bakersfield 93301. Calaveras, Fresno, Kern, Kings, Madera, Mariposa, Merced, San Joaquin, Stanislaus, Tulare and Tuolumne Counties. 805 327-4411
SIXTH DISTRICT—Monterey, San Benito, San Mateo, Santa Clara and Santa Cruz Counties.
  Office No. 1—Richard S. Wilbur (1967), 300 Homer Avenue, Palo Alto 94301. 415 321-4121
  Office No. 2—Albert G. Miller (1968), 77 San Mateo Drive, San Mateo 94401. 415 343-9074
SEVENTH DISTRICT—San Francisco County—
  Office No. 1—Malcolm S. M. Watts (1969), 909 Hyde Street, San Francisco 94109. 415 776-3838
  Office No. 2—Roberta F. Fenlon (1967), 490 Post Street, San Francisco 94102. 415 986-1391
EIGHTH DISTRICT—Alameda and Contra Costa Counties—
  Office No. 1—Harold Kay (1969), 411 Thirtieth Street, Oakland 94609. 415 832-4822
  Office No. 2—William F. Kaiser (1967), 3000 Colby Street, Berkeley 94705. 415 843-6515
NINTH DISTRICT—Carl E. Anderson (1967), 1150 Montgomery Drive, Santa Rosa 95405. Del Norte, Humboldt, Lake, Marin, Mendocino, Napa, Solano and Sonoma Counties. 707 546-1922
TENTH DISTRICT—James H. Yant (1968), 2600 Capitol Avenue, Sacramento 95816. Alpine, Amador, Butte, Colusa, El Dorado, Glenn, Lassen, Modoc, Nevada, Placer, Plumas, Sacramento, Shasta, Sierra, Siskiyou, Sutter, Tehama, Trinity, Yolo and Yuba Counties. 916 446-5278
ELEVENTH DISTRICT—Forest J. Grunigen (1967), 3919 Beverly Boulevard, Los Angeles 90004. Forty First Medical Society. 213 663-1161

SCIENTIFIC BOARD REPRESENTATIVE—William P. Longmire, Jr. (1967), UCLA Department of Surgery, Los Angeles 90024. 213 272-8911

* * *

COMMISSION ON *Medical Services*

Ralph Burnett, Bakersfield
Elmer F. Gooel, Beverly Hills (Secty.)
Arthur F. Howard, Fresno (Chmn.)
Charles B. Hudson, Oakland
Homer C. Pheasant, Los Angeles
Milo A. Youel, San Diego.

Committee on Fees

H. Dean Hoskins, Oakland	1967
Wallace A. Gerrie, Jr., Newport Beach	1967
Robert L. Watson, Jr., Los Angeles	1967
Roger C. Isenhour, San Diego	1968
Alfred J. Murrieta, Jr., Los Angeles	1968
Maurice S. Salomon, San Francisco	1968
Homer C. Pheasant, Los Angeles (Chmn.)	1969
Glenn A. Pope, Sacramento	1969
Leo Bell, San Mateo	1969
Joseph F. Boyle, Los Angeles	1969

CONSULTANT: William H. Thompson, San Mateo.

Committee on Federal Medical Care Programs

Charles B. Canby, Van Nuys	1967
Merlin A. Hendrickson, Rialto	1967
Ralph L. Hoffman, San Diego	1967
John S. McKee, Jr., San Diego	1968
Arthur Howard, Fresno (Chmn.)	1968

Committee on Veterans Affairs

Charles B. Hudson, Oakland (Chmn.)
Ben D. A. Miano, San Bernardino
Robert L. Watson, Los Angeles
CONSULTANT: Lloyd E. Gould, Napa.

Committee on Mediation

Eli A. Layon, Brawley	1967
Milo A. Youel, San Diego (Chmn.)	1967
Richard L. Taw, Los Angeles	1968
Albert G. Clark, San Francisco	1969
George Babbin, Sacramento	1969

Committee on Insurance and Prepayment

Oscar W. Hills, San Mateo	1967
Dave F. Dozier, Sacramento	1967
Ralph W. Burnett, Bakersfield (Chmn.)	1968
Stanley J. Murasky, Reseda	1968
Ivan Neubauer, San Jose	1969
Thayer A. Smith, Norwalk	1969

* * *

COMMISSION ON *Public Agencies*

Lewis T. Bullock, Los Angeles
Joseph P. Cosentino, Sacramento (Secty.)
William F. Kaiser, Berkeley
Howard W. Lindsey, Jr., San Mateo
Albert Miller, San Mateo (Chmn.)
Stanley A. Skillicorn, San Jose
Howell E. Wiggins, San Diego
Joseph P. O'Connor, Pasadena
CONSULTANT: Malcolm C. Todd, Long Beach.

Committee on Public Health

Robert L. Garrett, Vallejo	1967
Karl L. Schaupp, Jr., San Francisco	1967
Allen F. Sterling, San Bernardino	1967
Austin W. Matthis, Sacramento	1967
Albert G. Miller, San Mateo	1968
Stanley A. Skillicorn, San Jose (Chmn.)	1968
Alfred F. Kandlbinder, Monterey	1969
Robert Watson, Jr., Los Angeles	1969
Samuel S. Woolington, Long Beach	1969

Committee on Mental Health

Emmanuel Silver, Palo Alto	1967
Ira H. Degenhardt, San Rafael	1967
Rudolph B. Toller, Stockton	1967
John A. Stroud, III, Sacramento	1967
Alfred Auerback, San Francisco	1968
David H. Payne, Los Angeles	1968
Jack B. Lomas, Beverly Hills	1968
Joseph J. Furlong, San Francisco	1969
Stuart C. Knox, Los Angeles (Chmn.)	1969

Committee on Welfare Medical Care Programs

Clyde L. Boice, Palo Alto	1967
Edgar Rosen, Oakland	1967
Thomas N. Elmendorf, Willows	1967
Vincent P. Carroll, Laguna Beach	1967
Orrin S. Cook, Jr., Sacramento	1968
Robert F. Schell, San Rafael	1968
William F. Quinn, Los Angeles	1969
James Dalton, Santa Barbara	1969
Arthur F. Howard, Fresno	1969
Howard W. Lindsey, Jr., San Mateo (Chmn.)	1969

SUBCOMMITTEE ON ADOPTIONS: Walter Beckh, San Francisco; Fred M. Kay, Fullerton; Robert J. McNeil, Los Angeles, (Chmn.); Donald Minkler, Berkeley; Dale W. Ritter, Chico; Ben J. Siebenthal, Sacramento.

Committee on Occupational Health and Rehabilitation

John H. Leimbach, Jr., San Francisco	1967
Herbert A. Holden, San Leandro	1967
Howell E. Wiggins, San Diego (Chmn.)	1967
Edward Zaik, Los Angeles	1968
Packard Thurber, Jr., Los Angeles	1968
Thomas Gucker, III, Los Angeles	1968
Herbert A. Lints, Oakland	1969
Joseph N. Vizzard, Los Gatos	1969
Patrick J. Maloney, Merced	1969

* * *

COMMISSION ON *Community Health Services*

Wayne P. Chesbro, Berkeley
Charles G. Hutter, Jr., Hollywood
Gerald G. Jampolsky, Tiburon (Secty.)
Elmer R. Jennings, Long Beach
Harold Kay, Oakland (Chmn.)
George A. Martin, Redding
Pierre Salmon, San Mateo
Leopold J. Snyder, Fresno
Beverly G. Stewart, Bakersfield

Committee on Rural Health

Mitry P. Ajalat, Calexico	1967
Richard W. Johnson, Arbuckle	1967
Leopold J. Snyder, Fresno (Chmn.)	1968
Erwin P. Brauner, Visalia	1968
Carrol W. Goss, Lamont	1968
Franklin L. Murphy, Oroville	1969
Thomas Rossitto, Auburn	1969
M. Marlin Clark, Delano	1969

Committee on School Health

B. Otis Cobb, Martinez	1967
Gerald G. Jampolsky, Tiburon (Chmn.)	1967
Byron H. Demorest, Sacramento	1967
William A. Leovy, La Mesa	1968
Tom W. Robinson, Newport Beach	1968
Glenn Austin, Los Altos	1968
Ruth A. Frary, Watsonville	1968
Leslie M. Holve, Los Angeles	1969
Henry B. Bruyn, Berkeley	1969

Committee on Health Care of the Aged

Franklin F. Ham, Van Nuys	1967
Pierre Salmon, San Mateo (Chmn.)	1967
Charles E. Schoff, Jr., Sacramento	1968
Raymond D. Manchester, Visalia	1968
Leo A. Wagner, Novato	1969
Lloyd E. Gould, Napa	1969
Walter Batchelder, San Mateo	1969

Committee on Disaster Medical Care

Charles C. Henderson, San Mateo	1967
Donald E. Barker, Van Nuys	1967
Wayne P. Chesbro, Berkeley (Chmn.)	1968
Michael J. Hitchko, Eureka	1968
J. T. McNally, Stockton	1969
John Heald, San Francisco	1969

CONSULTANTS: Charles N. Earl, Arcata; Frank F. Schade, Los Angeles; Robert S. Johnson, Chico; Stephen Bruny, Fresno.

Committee on Automotive and Traffic Safety

George C. Snively, Sacramento	1967
Beverly G. Stewart, Bakersfield (Chmn.)	1967
Arnold R. Friesen, Pasadena	1968
Joseph F. Maguire, Ventura	1969
Thomas W. Lyons, La Mesa	1969
Robert L. Tour, San Francisco	1969

CONSULTANTS: Julian A. Waller, Berkeley; Julian M. Sether, Los Angeles.

Committee on Medical Aspects Of Sports and Physical Fitness

John F. Blinn, Stockton	1967
Robert K. Kerlan, Los Angeles	1967
George S. Buehler, Whittier	1968
Philip H. McFarland, Fullerton	1968
Tom W. Robinson, Newport Beach	1968
Charles G. Hutter, Jr., Hollywood (Chmn.)	1969
Arnold A. Nutting, San Rafael	1969

Committee on Environmental Health

George A. Martin, Redding	1967
Robert L. Hippen, San Diego	1967
Grace M. Talbott, San Francisco	1968
Francis M. Pottenger, Jr., Monrovia	1968
Reginald H. Smart, Los Angeles	1968
Douglas Campbell, San Francisco	1969
John R. Lee, Mill Valley	1969

Committee on Blood Banks

Robert W. Purvis, Modesto	1967
Osman W. Hull, Monterey	1967
James H. Yant, Sacramento	1967
Elmer Jennings, Long Beach (Chmn.)	1968

CONSULTANT: Melvin L. Sommer, Beverly Hills

COMMISSION ON *Communications*

Frederick Ackerman, Pleasant Hill
L. Morgan Boyers, Sacramento (Secty.)
John A. E. Bullis, Los Angeles
Roberta F. Fenlon, San Francisco
Elmer F. Gooel, Beverly Hills
Walter W. Hopps, Los Angeles
George Schwartz, Beverly Hills
Malcolm C. Todd, Long Beach (Chmn.)
Richard S. Wilbur, Palo Alto
EX-OFFICIO: James C. MacLaggan, San Diego; John G. Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Scientific Board

David A. Rytand, Palo Alto	1967
William W. Belford, San Diego	1967
Jerome W. Bettman, San Francisco	1967
John B. Dillon, Los Angeles	1967
Justin J. Stein, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
R. Kirklin Ashley, San Francisco	1967
Victor Richards, San Francisco	1967
David Rubin, Los Angeles	1967
Keith P. Russell, Los Angeles	1967
Edward J. Zaik, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Seymour J. Brockman, Los Angeles	1968
S. Austin Jones, Los Angeles	1968
Hobart M. Kelly, Riverside	1968
Saul J. Robinson, San Francisco	1968
Eugene Ziskind, Los Angeles	1968
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. deC. M. Saunders, San Francisco	1968
Herbert A. Holden, San Leandro	1968
David A. Wood, San Francisco	1968
Arthur A. Clinco, Los Angeles	1968
R. Theodore Bergman, Los Angeles	1968
Leland B. Blanchard, San Jose	1969
L. S. Goerke, Los Angeles	1969
Bert L. Halter, San Francisco	1969
Ellis Jones, Pasadena	1969
Carl McCandless, San Francisco	1969
J. Blair Pace, Oceanside	1969
Donald W. Petit, Los Angeles	1969
Glenn Pope, Sacramento	1969
Robert J. Prentiss, San Diego	1969
Edward B. Shaw, San Francisco	1969
Ralph E. Worden, Los Angeles	1969

COUNCIL REPRESENTATIVES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.
EX-OFFICIO: Dwight L. Wilbur, San Francisco.

Executive Committee

William W. Belford, San Diego	1967
Victor Richards, San Francisco	1967
Justin J. Stein, Los Angeles	1967
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. Dillon, Los Angeles	1969
Donald Petit, Los Angeles	1969

COUNCIL DESIGNEES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.

Committee on Nominations

Justin J. Stein, Los Angeles (Chmn.)	1967
Edward B. Shaw, San Francisco	1969
Robert J. Prentiss, San Diego	1969

Committee on Scientific Assemblies

John B. Dillon, Los Angeles (Chmn.)	1967
Victor Richards, San Francisco	1967
David A. Rytand, Palo Alto	1967
John B. deC. M. Saunders, San Francisco	1968
Ellis Jones, Pasadena	1969
Donald Petit, Los Angeles	1969

CONSULTANT: Claron Oakley, Los Angeles

Committee on Continuing Medical Education

John B. Dillon, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
Saul J. Robinson, San Francisco	1968
Donald W. Petit, Los Angeles (Chmn.)	1969
Bert L. Halter, San Francisco	1969
J. Blair Pace, Oceanside	1969

Committee on CALIFORNIA MEDICINE

David Rubin, Los Angeles	1967
R. Kirklin Ashley, San Francisco	1967

Committee on Scientific Information

Justin J. Stein, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Arthur A. Clinco, Los Angeles	1968
Edward B. Shaw, San Francisco (Chmn.)	1969
L. S. Goerke, Los Angeles	1969

Committee on Maternal and Child Care

Russell W. Mapes, Beverly Hills	1967
Keith P. Russell, Los Angeles (Chmn.)	1967
Forrest H. Howard, Garden Grove	1967
Charles M. Blumenfeld, Sacramento	1968
Herbert A. Holden, San Leandro	1968
Hobart M. Kelley, Riverside	1968
Leland Blanchard, San Jose	1969

Committee on Cancer

John W. Cline, San Francisco	1967
Robert W. Jamplis, Palo Alto	1967
David A. Wood, San Francisco (Chmn.)	1967
John G. Walsh, Sacramento	1968
Lewis W. Guiss, Los Angeles	1969
Robert Prentiss, San Diego	1969

SUBCOMMITTEE ON CANCER EDUCATION: Sol R. Baker, Beverly Hills; Robert W. Jamplis, Palo Alto (Chmn.); Maurice Galante, San Francisco; Justin J. Stein, Los Angeles.

SUBCOMMITTEE ON TUMOR TISSUE REGISTRY: John W. Cline, San Francisco (Chmn.); Lewis W. Guiss, Los Angeles; James E. Kahler, Los Angeles; Carl M. McCandless, San Francisco; Weldon K. Bullock, Los Angeles.

SUBCOMMITTEE ON CONSULTATIVE TUMOR BOARDS: Bert L. Halter, San Francisco; Carl L. Moore, Bakersfield; Victor Richards, San Francisco; John G. Walsh, Sacramento (Chmn.).

Committee on Adverse Drug Reactions

Edward R. Bloomquist, Glendale	1967
Ralph O. Wallerstein, San Francisco	1967
John F. Murray, Fresno (Chmn.)	1968
Russell J. Tat, San Francisco	1969
William W. Thayer, Stockton	1969

CONSULTANT: John W. Brown, Berkeley; Ralph W. Weilerstein, Berkeley.

SUBCOMMITTEE ON DANGEROUS DRUGS: Edward Bloomquist, Glendale; Norman A. Gale, San Diego; Edward T. Jewett, Santa Rosa; William F. Quinn, Los Angeles (Chmn.); Helen B. Weyrauch, San Francisco.
CONSULTANT: Ralph W. Weilerstein, Berkeley.

Committee on Cardiovascular Diseases

Harold I. Griffeath, San Francisco (Chmn.); Lewis T. Bullock, Los Angeles; David J. New, Modesto; George C. Griffith, Los Angeles.
CONSULTANT: John E. Connolly, Los Angeles.

* * *

BUREAU OF *Research and Planning*

J. Brandon Bassett, Oakland
John Sheehy, Riverside
Samuel Sherman, San Francisco (Chmn.)
H. Russell Fisher, Glendale
G. Roger Ellison, Pomona
EX-OFFICIO: Malcolm S. M. Watts, San Francisco; Ralph C. Teall, Sacramento.
CONSULTANT: Gerald W. Shaw, Santa Monica.

* * *

COMMISSION ON *Professional Welfare*

Bert L. Halter, San Francisco
George K. Herzog, Jr., San Francisco (Chmn.)
Francis E. West, San Diego
James H. Yant, Sacramento (Secty.)

Committee on Physicians Group Insurance

Robert J. Wescott, El Centro	1967
Robert X. DeMangus, Glendale	1967
John B. Long, Sacramento	1967
George K. Herzog, Jr., San Francisco (Chmn.)	1968
John Hogan, San Diego	1969
Homer C. Pheasant, Los Angeles	1969

Liaison Committee to Medical Schools

Samuel R. Irvine, Beverly Hills	1967
Francis E. West, San Diego	1967
Frank J. Novak, Menlo Park	1968
Stanley Cowell, Los Angeles	1968
Bert L. Halter, San Francisco (Chmn.)	1969
Alonzo Neufeld, Los Angeles	1969
Herbert Jenkins, Sacramento	1969

EX-OFFICIO: James C. MacLaggan, San Diego; John G. Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Medical Review and Advisory Committee

Eugene M. Webb, San Francisco	1967
James H. Yant, Sacramento (Chmn.)	1967
John B. Dillon, Los Angeles	1968
Carl Goetsch, Berkeley	1968
Donald Donisthorpe, Los Angeles	1969

CONSULTANT: Don Harper Mills, Long Beach.

Liaison Committee to State Bar of California

David Rubsamen, Berkeley	1967
John V. Fiore, Westchester	1967
Francis E. West, San Diego (Chmn.)	1968
Carl Goetsch, Berkeley	1968
Robert J. McNeil, Los Angeles	1969
Henry A. Brown, San Mateo	1969

* * *

COMMISSION ON *Allied Health Professions and Services*

Roger W. Barnes, Los Angeles (Secty.)
Forest J. Grunigen, Los Angeles (Chmn.)
Frank C. Melone, Ontario
Thomas W. Ledwich, Napa
C. Gerald Scarborough, San Jose
Richard H. Schug, Long Beach

Committee on Paramedical Personnel

Frank C. Melone, Ontario (Chmn.)	1967
Gregory Bard, San Francisco	1968
Elmer R. Jennings, Long Beach	1968
Gerson R. Biskind, San Francisco	1969
Joseph Schaefer, Santa Rosa	1969
Leonard M. Asher, Los Angeles	1969
Richard H. Schug, Long Beach (Chmn.)	1969

Committee on Other Professions

George S. Buehler, Whittier	1967
Burt L. Davis, Palo Alto	1967
C. Gerald Scarborough, San Jose (Chmn.)	1968
William D. Evans, Los Angeles	1968
J. Gordon Epperson, Oakland	1969
Lewis T. Bullock, Los Angeles	1969

Liaison Committee to California Medical Assistants Association

Leon O. Burke, Sacramento	1967
John C. Brennan, Pico Rivera	1967
H. Russell Irwin, San Diego	1967
Peary Benn Berger, Inglewood	1968
Judith D. Smith, Vallejo	1969
Sanford E. Feldman, San Francisco	1969
Richard H. Schug, Long Beach (Chmn.)	1969

CONSULTANTS: Leland W. Bonar, Redding; Henry Bodner, Van Nuys.

Committee on Medicine and Religion

Albert E. Long, Daly City	1967
Roger W. Barnes, Los Angeles (Chmn.)	1967
Max Nelson, National City	1968
Frank J. Glassy, Sacramento	1969
Richard J. Lescoe, Torrance	1969

Committee on Voluntary Health Agencies

H. Mason Hohl, Beverly Hills	1967
Thomas W. Ledwich, Napa (Chmn.)	1968
Harvey May Cordua, Jr., San Diego	1969
J. Brandon Bassett, Oakland	1969

* * *

Judicial COMMISSION

Donald A. Charnock, Los Angeles	1967
Albert E. Long, Daly City	1967
Donald Cass, Hollywood	1967
Floyd O. Due, Oakland	1968
Sidney J. Shipman, San Francisco	1968
Francis E. West, San Diego (Chmn.)	1968
Douglas G. Campbell, San Francisco	1969
W. Philip Corr, Riverside (Secty.)	1969
L. E. Wilson, Anaheim	1969

* * *

COMMISSION ON *Hospital Affairs*

William L. Argo, Fresno (Secty.)
James C. Doyle, Beverly Hills
Bert L. Halter, San Francisco (Chmn.)
John C. Lungren, Long Beach
Glenn Pope, Sacramento
John T. Saidy, San Mateo
Joseph W. Telford, San Diego
Frederic N. Tyroler, Los Angeles

Medical Staff Survey Committee

Robert M. Adams, San Leandro
W. L. Argo, Fresno
Daniel W. Black, Castro Valley
Rush M. Blodget, Jr., Redding
Bert L. Halter, San Francisco (Chmn.)
J. Ralph Hughes, San Diego
Wm. Kenneth Jennings, Santa Barbara
Donald O. Lagerlof, Los Angeles
John C. Lungren, Long Beach
William A. Newsom, San Francisco
James L. Rhee, Oakland
John T. Saidy, San Mateo
Wilfred J. Snodgrass, Santa Monica
Alexander Hatoff, Oakland
Shepard F. Palitz, Bakersfield
William L. Sumption, Santa Monica
A. Jerome Hurter, Pasadena
Ernest R. Barnett, Alhambra
Richard L. Taw, Los Angeles
Richard H. McDonald, Garden Grove
Robert M. Zweig, Riverside
L. Morgan Boyers, Sacramento
James O. Farley, Sacramento
Joseph W. Telford, San Diego
Paul Deeb, Redlands
Maxwell Goldstein, Fontana
Ray Seavers, Jr., San Bernardino
William Soldmann, La Mesa
Lloyd Milburn, San Francisco
Frank Solomon, San Francisco
Sanford Feldman, San Francisco
John Bazzano, San Francisco
Kenneth Allen, San Mateo
Martin Kohn, San Bruno
William Stephenson, Santa Barbara
Merlin Hendrickson, Rialto
Andrew D. Lucine, Campbell
Fred Haven Draper, San Jose
Wayne O. Buck, San Jose
William A. Reeves, Mt. View
A. A. Thurlow, Jr., Santa Rosa
John A. Carollo, Sabastopol
William Rector, Sacramento
Gerald W. Shaw, Santa Monica
Charles L. Lear, Norwalk
John R. Heckman, Marysville
Robert Buckley, Santa Monica
Woodrow W. Burgess, Sacramento
Ira H. Degenhardt, San Rafael
Arthur E. Gladman, Oakland
Norman Graff, San Mateo
Frank V. Hoffman, Huntington Beach
Robert E. James, Belmont
Clement O. Juul, Oakland
Seymour Kolko, San Mateo
Melvin M. Lipsett, Berkeley
Jack B. Lomas, Los Angeles
Edward A. Macklin, San Francisco
D. Stuart MacRobbie, Berkeley
Felix H. Ocko, Berkeley
William R. Sheehy, Berkeley
John A. Stroud, Sacramento
R. J. Sutherland, Oakland
Rudolph B. Toller, Stockton
Albert T. Voris, Belmont
G. Cameron Cray, Palo Alto

(Continued on Page 32)

Officers of Scientific Sessions

Members who contemplate presentation of scientific papers should promptly address the secretary of the proper section at the address shown below.

Allergy

Leo N. Meleyco..........Chairman
2040 Forest Avenue, San Jose 95128
Ernest J. Saslow..........Secretary
2011-18th Street, Bakersfield 93301
Samuel Ross..........Assistant Secretary
2946 Fresno Street, Fresno 93706

Anesthesiology

Thomas W. McIntosh..........Chairman
686 East Union St., Pasadena 91101
Donald B. Dose..........Secretary
5625 Linfield Ave., San Diego 92120
Donald A. Walker..........Assistant Secretary
35 Spring Lane, Tiburon 94920

Dermatology

Robert G. Walton..........Chairman
1700 McHenry Village Way, Modesto 95350
Samuel Ayres, III..........Secretary
405 N. Bedford Rd., Beverly Hills 90210
George T. Wilson..........Assistant Secretary
Welch and Pasteur Rd., Stanford Medical Plaza
Palo Alto 94304

General Practice

J. Blair Pace..........Chairman
408 Cassidy St., Oceanside 92054
Alfred F. Kandlbinder..........Secretary
302 Via Paraiso St., Monterey 93940
Allen F. Sterling..........Assistant Secretary
1285 D St., San Bernardino 92045

General Surgery

Seibert Pearson..........Chairman
2865 Atlantic Ave., Long Beach 90807
Albert G. Clark..........Secretary
450 Sutter St., San Francisco 94108
Robert T. Hood, Jr..........Assistant Secretary
2010 Wilshire Blvd., Los Angeles 90057

Industrial Medicine and Surgery

Leon R. Rudnick..........Chairman
15921 E. 14th St., San Leandro 94578
Raymond O. Oja..........Secretary
525 Golden Gate Ave., San Francisco 94102
James G. Forgerson..........Assistant Secretary
Metropolitan Life Insurance Company
600 Stockton St., San Francisco 94108

Internal Medicine

Robert L. Paver..........Chairman
384 Post St., San Francisco 94108
John L. Benton, Jr..........Secretary
1216 Wilshire Blvd., Los Angeles 90017
Edgar Rosen..........Assistant Secretary
1340 K St., Sacramento 95814

Obstetrics and Gynecology

Lester T. Hibbard..........Chairman
1930 Wilshire Blvd., Los Angeles 90057
Warren E. Jones..........Secretary
1380-45th Street, Sacramento 95819
James W. Ravenscroft..........Assistant Secretary
2330 First Ave., San Diego 92101

Ophthalmology

Victor G. Fellows..........Chairman
595 Buckingham Way, San Francisco 94117
Wendell C. Irvine..........Secretary
3875 Wilshire Blvd., Los Angeles 90005
Robert E. Hopkins..........Assistant Secretary
4255 Pacific Ave., Stockton 95207

Orthopedics

David C. Monsen..........Chairman
2300 S. Hope St., Los Angeles 90007
Arthur R. Hartwig..........Secretary
490 Post St., San Francisco 94102
Vernon L. Smythe..........Assistant Secretary
3875 Wilshire Blvd., Los Angeles 90005

Otolaryngology

G. Howard Gottschalk..........Chairman
8618 S. Sepulveda Blvd., Los Angeles 90045
Charles P. Lebo..........Secretary
490 Post St., San Francisco 94102
Irving L. White..........Assistant Secretary
911 San Antonio Dr., Long Beach 90807

Pathology

Frank R. Dutra..........Chairman
20103 Lake Chabot Rd., Castro Valley 94546
William H. Kern..........Secretary
679 S. Westlake Ave., Los Angeles 90057
Donald L. Bittner..........Assistant Secretary
Bay Area Laboratories, 2245 Post St.,
San Francisco 94115

Pediatrics

Richard L. Anderson..........Chairman
636 Harris St., Eureka 95501
Sherrod C. Swift..........Secretary
14619 Whittier Blvd., Whittier 90605
Robert J. Harvey..........Assistant Secretary
909 Hyde St., San Francisco 94109

Physical Medicine

Rene Cailliet..........Chairman
4900 Sunset Blvd., Los Angeles 90027
Anton A. Tratar..........Secretary
2360 Clay St., San Francisco 94115
Elizabeth Austin..........Assistant Secretary
Los Angeles County Hospital
1200 N. State St., Los Angeles 90033

Preventive Medicine

Henrik L. Blum..........Chairman
P. O. Box 871, Martinez 94554
Maxwell Rosenblatt..........Secretary
Los Angeles County Health Department,
220 N. Broadway, Los Angeles 90012
Russell B. Watson..........Assistant Secretary
Orange County Health Department, Santa Ana
92701

Psychiatry and Neurology

Werner M. Mendel..........Chairman
1934 Hospital Place, Los Angeles 90033
Ames Fischer..........Secretary
Langley Porter Inst., 401 Parnassus Ave.,
San Francisco 94122
Howard S. Barrows..........Assistant Secretary
2025 Zonal Ave., Los Angeles 90033

Radiology

Victor G. Mikity..........Chairman
1200 State St., Los Angeles 90033
Vernon R. Gee..........Secretary
2020 Court St., Redding 96001
Richard P. Storrs..........Assistant Secretary
2131 West 3rd St., Los Angeles 90057

Urology

Carl K. Pearlman..........Chairman
1518 N. Sycamore, Santa Ana 92701
James L. Goebel..........Secretary
51 Loma Linda, Ross 94957
Robert O. Pearman..........Assistant Secretary
5353 Balboa Blvd., Encino 91316

Delegates and Alternates

TO THE AMERICAN MEDICAL ASSOCIATION

DELEGATES		ALTERNATES
Francis E. West, San Diego	(1966-1967)	Vincent P. Carroll,* Laguna Beach
Samuel R. Sherman, San Francisco	(1966-1967)	Albert G. Miller, San Mateo
Henry Gibbons, III, San Francisco	(1966-1967)	A. B. Sirbu, San Francisco
John M. Rumsey, San Diego	(1966-1967)	Carl E. Anderson, Santa Rosa
Eugene F. Hoffman, Los Angeles	(1966-1967)	Joseph F. Boyle, Los Angeles
Warren L. Bostick, Los Angeles	(1966-1967)	Walter H. Brignoli, St. Helena
J. B. Price, Santa Ana	(1966-1967)	Donald C. Dodds, Oakland
Ralph C. Teall, Sacramento	(1966-1967)	Robb Smith, Orange Cove
James C. Doyle, Beverly Hills	(1966-1967)	Herman H. Stone, Riverside
Wilbur G. Rogers, Glendale	(1966-1967)	Carl M. Hadley, San Bernardino
Charles B. Hudson, Oakland	(1966-1967)	Robert L. Watson, Jr., Los Angeles
John G. Morrison, San Leandro	(1967-1968)	Frederick Ackerman, Pleasant Hills
William F. Quinn, Los Angeles	(1967-1968)	Dudley M. Cobb, Los Angeles
Robert C. Combs, San Francisco	(1967-1968)	Emmett Rixford, San Francisco
Homer C. Pheasant, Los Angeles	(1967-1968)	James C. MacLaggan, San Diego
Alfred J. Murrieta, Los Angeles	(1967-1968)	Harold Kay, Oakland
Burt L. Davis, Palo Alto	(1967-1968)	Leon P. Fox, San Jose
Arlo A. Morrison, Ventura	(1967-1968)	Henry Brown, San Mateo
James E. Feldmayer, Exeter	(1967-1968)	Charles Grayson, Sacramento
O. W. Wheeler, Riverside	(1967-1968)	Wilbur Bailey, Los Angeles
Malcolm C. Todd, Long Beach	(1967-1968)	Neil C. Hamel, Encino
J. E. Vaughan, Bakersfield	(1967-1968)	Joseph P. O'Connor, Pasadena

(Delegates and Alternates to the AMA are elected for terms of two calendar years. Each office is scheduled for election at the Annual Session of the second year shown above.) **Council appointee.*

Other Organizations and Medical Schools

Board of Medical Examiners of the State of California

Secretary..........Shelby M. Hicks, M.D.
1021 O Street, Room A202, Sacramento 95814
San Francisco..........30 Van Ness Ave., 94102
Room 2100
Los Angeles..........107 South Broadway 90012
Sacramento..........1021 O Street, Room A547 95814

The Public Health League of California

Executive Secretary..........Ben H. Read
955 South Western Ave., Los Angeles 90006
Assistant Executive Secretary..........Al Paul Putnam
693 Sutter Street, San Francisco 94102
San Francisco..........693 Sutter Street 94102
415 474-1061
Los Angeles..........955 South Western Ave. 90006
213 731-6397
Sacramento..........1127-11th Street, 95814
916 444-7496

Department of Public Health of the State of California

Director..........Lester Breslow, M.D.
2151 Berkeley Way, Berkeley 94704
Berkeley..........2151 Berkeley Way 94704
415 843-7900
Sacramento..........1927-13th Street 95814
916 445-2264
Los Angeles..........417 South Hill Street 90013
213 620-2900

Medical Schools in California

University of California School of Medicine, Medical Center, San Francisco 94122. Dean: Stuart C. Cullen, M.D.
Stanford University School of Medicine, 300 Pasteur Drive, Palo Alto 94304. Dean: Robert J. Glaser, M.D.
University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 90033. Dean: Roger O. Egeberg, M.D.
Loma Linda University School of Medicine, Loma Linda 92354. Dean: David Burdg Hinshaw, M.D.
University of California at Los Angeles, School of Medicine, Hilgard Avenue, Los Angeles 90024. Dean: Sherman M. Mellinkoff, M.D.
University of California, California College of Medicine, 1721 Griffin Avenue, Los Angeles 90031. Dean: Warren L. Bostick, M.D.
University of California, San Diego, School of Medicine, La Jolla 92038. Acting Dean: Robert D. Tschirgi, M.D.
University of California, Davis, School of Medicine, Davis 95616. Dean: Charles John Tupper, M.D.

California Medical Education and Research Foundation

Directors: Ralph C. Teall, M.D., President; Carl E. Anderson, M.D., Vice-President; Howard Hassard, Secretary; James C. MacLaggan, M.D.; Harold Kay, M.D.; William F. Quinn, M.D.; E. B. Shaw, M.D.

Roster — CALIFORNIA COMPONENT MEDICAL SOCIETIES

Society secretaries are requested to notify California Medicine promptly when changes are indicated in their roster information

ALAMEDA-CONTRA COSTA Medical Assn., 6230 Claremont Ave., Oakland 94618. Meets quarterly.
Frederick W. Ackerman........................President
2100 Monument Blvd., Pleasant Hill 93542
Herbert A. Holden..............................Secretary
1436 Clarke St., San Leandro 94577

BUTTE-GLENN Medical Society, P. O. Box 1008, Chico 95927. Meets Fourth Thursday.
Walter S. Mazen..................................President
1617 Esplanade, Chico 95926
John B. Copeland................................Secretary
1423 Magnolia, Chico 95926

FORTY FIRST Medical Society, Suite 821, 1515 N. Vermont Ave., Los Angeles 90027
Paul D. Yates.......................................President
844 Hermosa Ave., Hermosa Beach 90254
Jordan M. Phillips................................Secretary
11239 S. Lakewood Blvd., Downey 90241

FRESNO County Medical Society, 1118 Divisadero St., Fresno 93721. Meets Second Tuesday, 6:30 p.m., California Hotel.
Theodore Steinberg ....President
155 N. Thesta St., Fresno 93701
Jack Schiff ....Secretary
167 N. Thesta St., Fresno 93701

HUMBOLDT-DEL NORTE County Medical Society. Meets Second Thursday.
Wells C. Carey.....................................President
2822 E St., Eureka 95501
Max Todd ....Secretary
616-14th St., Arcata 95521

IMPERIAL County Medical Society, P. O. Box 518, Imperial 92251. Meets Second Tuesday, 8 p.m., Pioneer Memorial Hospital, Brawley.
O. G. Fais..President
1745 S. Imperial Ave., El Centro 92243
Elmer C. Werner..................................Secretary
1745 S. Imperial Ave., El Centro 92243

INYO-MONO County Medical Society. Meets Fourth Tuesday except December, January, February.
Milton R. Jones, Jr...............................President
Box 38, Lone Pine 93545
Robert W. Denton ....Secretary
611 W. Line St., Bishop 93514

KERN County Medical Society, 2603 G Street, Bakersfield 93301. Meets Third Tuesday, 7:30 p.m., Society Office, 2603 G Street, except June, July, August.
Keith Spaulding ....President
2521 "G" St., Bakersfield 93301
James Friend ....Secretary
1921-18th St., Bakersfield 93301

KINGS County Medical Society, P. O. Box 1003, Hanford 93230. Meets Second Thursday, 7:00 p.m. Place to be announced.
James Dean ....President
912 N. Douty St., Hanford 93230
Willard S. Bridwell ....Secretary
1507 N. 11th Ave., Hanford 93230

LASSEN-PLUMAS-MODOC-SIERRA County Medical Society. Meets on call.
Kenneth G. Korver ....President
50 N. Roop St., Susanville 96130
James McCullough ....Secretary
50 N. McDow St., Susanville 96130

LOS ANGELES County Medical Assn., 1925 Wilshire Blvd., Los Angeles 90057.
Joseph F. Boyle....................................President
1136 W. Sixth St., Los Angeles 90017
Homer C. Pheasant..............................Secretary
2300 S. Hope St., Suite 300,
Los Angeles 90007

MARIN Medical Society, 1601 Second St., Suite 106, San Rafael 94901. Meets First Thursday, 7:00 p.m.
Robert Mills ....President
711 D St., San Rafael 94901
William A. Galeno ....Secretary
712 D St., San Rafael 94901

MENDOCINO-LAKE County Medical Society.
Frank H. Dailey...................................President
719 S. Dora, Ukiah 95482
Patrick R. Allanson ....Secretary
765 S. Dora St., Ukiah 95482

MERCED-MARIPOSA Medical Society, P. O. Box 586, Merced 95340. Meets Fourth Thursday; meeting place changes.
Patrick J. Maloney...............................President
P. O. Box 586, Merced 95340
Robert J. Schiffler................................Secretary
P. O. Box 586, Merced 95340

MONTEREY County Medical Society, P. O. Box 308, Salinas 93903. Meets First Thursday.
Joseph E. Turner ....President
1010 Cass St., Monterey 93940
Thomas S. Elliott ....Secretary
101 Maple St., Greenfield 93927

NAPA County Medical Society, P. O. Box 2158, Napa 94559. Meets Second Wednesday.
Richard W. Neil ....President
1375 Main St., Helena 94574
Victor J. Bouillon ....Secretary
3020 Beard Rd., Napa 94558

ORANGE County Medical Association, 300 S. Flower, Orange 92666. Meets First Tuesday, 7:00 p.m.
Wallace A. Gerrie................................President
1901 Westcliff Dr., Newport Beach 92660
David I. Nielsen ....Secretary
400 Westminster Blvd., Newport Beach 92660

PLACER-NEVADA County Medical Society. Meets Second Wednesday.
F. Harold Johnson...............................President
305 Oak Ridge Dr., Roseville 95678
Arthur R. Weaver ....Secretary
1212 High St., Suite 106, Auburn 95603

RIVERSIDE County Medical Association, 4175 Brockton Ave., Riverside 92501. Meets Second Monday, 7:30 p.m., Spanish Arts Gallery, Mission Inn.
Roger W. Ridley ....President
6969 Brockton Ave., Riverside 92506
Richard W. Trotter ....Secretary
6876 Magnolia Ave., Riverside 92506

SACRAMENTO County Medical Society, 5380 Elvas Ave., Sacramento 95819. Meets Third Tuesday, 8:30 p.m. Location of meetings varies.
James W. Martin ....President
2811 L St., Sacramento 95816
James O. Farley ....Secretary
3428 Watt Ave., Sacramento 95821

SAN BENITO County Medical Society. Meets once a month, except July and August. Time and place to be announced.
Robert D. Quinn ....President
930 Sunset Dr., Hollister 95023
Roderick L. Yip ....Secretary
891 Sunset Dr., Hollister 95023

SAN BERNARDINO County Medical Society, 1875 N. D St., San Bernardino 92405. Meets First Tuesday, 7:30 p.m., Harold's Club, Fontana.
Harold Hill ....President
219 Cajon Ave., Redlands 92373
Nicholas P. Krikes ....Secretary
25060 Base Line, San Bernardino 92410

SAN DIEGO County Medical Society, 3427-4th Ave., San Diego 92103. Meets Second Tuesday. Location of meetings varies.
John Wanless ....President
3427 Fourth Ave., San Diego 92103
W. C. Herrick ....Secretary
233 A St., San Diego 92101

SAN FRANCISCO Medical Society, 250 Masonic Ave., San Francisco 94118.
Albert G. Clark....................................President
450 Sutter St., San Francisco 94108
Harry W. Sawyer.................................Secretary
909 Hyde St., San Francisco 94109

SAN JOAQUIN County Medical Society, 445 W. Acacia St., P. O. Box 230, Stockton 95201. Meets First Thursday, 8:15 p.m.
Thomas Huff ....President
512 W. Harding Way, Stockton 95204
Dora A. Lee ....Secretary
2420 N. California St., Stockton 95204

SAN LUIS OBISPO County Medical Society, P.O. Box 319, San Luis Obispo 93402. Meets Third Saturday, 7:00 p.m., Madonna Inn, San Luis Obispo 93402.
Robert Buck ....President
P. O. Box 319, San Luis Obispo 93402
Billy W. Mounts ....Secretary
P. O. Box 319, San Luis Obispo 93402

SAN MATEO County Medical Society, 122 El Camino Real, San Mateo 94401. Meets Third Tuesday.
Howard W. Lindsey, Jr........................President
323 N. San Mateo Dr., San Mateo 94401
Martin M. Kohn ....Secretary
841 W. San Bruno Ave., 94066

SANTA BARBARA County Medical Society, 300 W. Pueblo St., Santa Barbara 93105. Meets Second Monday of the month, University Club, 1332 Santa Barbara St., Santa Barbara.
T. K. Hill...President
1421 State St., Santa Barbara 93105
Casimir Domz ....Secretary
317 W. Pueblo St., Santa Barbara 93105

SANTA CLARA County Medical Society, 700 Empey Way, San Jose 95128. Meets First Monday.
Gerald Besson ....President
700 Empey Way, San Jose 95128
Alexander MacKenzie ....Secretary
700 Empey Way, San Jose 95128

SANTA CRUZ County Medical Society. Meets every Second Month, Second Tuesday. Time, place to be announced.
W. W. Hoskins....................................President
30 Brennan St., Watsonville 95076
William Blade ....Secretary
1510 Seabright Ave., Santa Cruz 95062

SHASTA-TRINITY County Medical Society. Meets Second Monday of the month. Place to be announced.
Faylon M. Brunemeier ....President
2020 Court St., Redding 96001
Eugene A. Nutley ....Secretary
2625 Edith Ave., Redding 96001

SISKIYOU County Medical Society. Meets Sunday on call.
Robert L. Anderson ....President
504 Hillcrest Dr., Yreka 96097
John P. Geyman ....Secretary
705 S. A St., Mt. Shasta 96067

SOLANO County Medical Society, 631 Tennessee St., Vallejo 94599.
Tuesday, 8:00 p.m., at different meeting places.
Robert L. Garrett.................................President
1512 Napa St., Vallejo 94590
Keith E. Vincent ....Secretary
2121 Redwood St., Vallejo 94594

SONOMA County Medical Society, 1625 Franklin Ave., Santa Rosa 95404. Meets Second Thursday.
John S. Dunn ....President
576 B St., Santa Rosa 95401
Elizabeth Buckley ....Secretary
1140 Sonoma Ave., Santa Rosa 95405

STANISLAUS County Medical Society, 1125-13th St., Modesto 95354. Meets Third Tuesday of the month, 7 p.m., Hotel Covell, Modesto.
Howard Kuhl ....President
1700 McHenry Ave., Modesto 95350
Jeanne I. Miller ....Secretary
709-18th St., Modesto 95354

TEHAMA County Medical Society. Meets at call of President.
W. Scott Davis ....President
P. O. Box 118, Los Molinos 96055
M. Blair McGee ....Secretary
217 Corona Ave., Corning 96021

TULARE County Medical Society, 1640 West Mineral King. P. O. Box 16, Visalia 93278.
Irving M. Schor...................................President
P. O. Box 1151, Visalia 93277
George Tiss ....Secretary
P. O. Box 7, Visalia 93277

VENTURA County Medical Society, 3291 Loma Vista Rd., Room 40, Ventura 93003. Meets Second Tuesday each month, 7:30 p.m., Los Posas Country Club, Camarillo.
Joseph F. Gstettenbauer.......................President
3003 Loma Vista Rd., Ventura 93003
Franklin K. Helbling ....Secretary
3291 Loma Vista Rd., Room 40, Ventura 93003

YOLO County Medical Society. Meets First Wednesday.
Benjamin R. Robinson.........................President
650 Third St., Woodland 95695
Corbin Young ....Secretary
101 Main St., Winters 95694

YUBA-SUTTER-COLUSA County Medical Society.
John R. Heckman.................................President
714-14th St., Marysville 95901
Norman K. Noordhoff ....Secretary
320 G St., Marysville 95901

Officers — CALIFORNIA PHYSICIANS' SERVICE — 1966-67
BLUE SHIELD

Officers:

Chairman of the Board	Richard S. Wilbur
Vice-Chairman of the Board	Milo A. Youel
Secretary	Wilbur G. Rogers
Treasurer	Bert L. Halter

BOARD OF *Trustees:*

Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Roberta F. Fenlon*	San Francisco
Donald R. Fitch	Glendale
Mr. Thomas Hadfield	San Francisco
Bert L. Halter	San Francisco
Donald C. Harrington	Stockton
M. M. Haskell	Long Beach
Carl Goetsch	Berkeley
Mr. Philip S. Magruder	Flintridge
Gregory C. Murray	Los Angeles
Wilbur G. Rogers*	Glendale
Mr. Paul Schrade	Los Angeles
William H. Thompson	San Mateo
Richard S. Wilbur*	Palo Alto
Milo A. Youel	San Diego

---

*Appointed by CMA Council

BOARD COMMITTEES:

Executive Committee

Richard S. Wilbur, Chairman	Palo Alto
Carl Goetsch	Berkeley
Bert L. Halter	San Francisco
Mr. Philip S. Magruder	Flintridge
Wilbur G. Rogers	Glendale
William H. Thompson	San Mateo
Milo A. Youel	San Diego

Finance Committee

Bert L. Halter, Chairman	San Francisco
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Donald R. Fitch	Glendale
Mr. Philip S. Magruder	Flintridge

Medical Policy Committee

Wilbur G. Rogers, Chairman	Glendale
Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Roberta F. Fenlon	San Francisco
Carl Goetsch	Berkeley
Mr. Thomas Hadfield	San Francisco
M. M. Haskell	Long Beach
Donald C. Harrington	Stockton
Gregory C. Murray	Los Angeles

Togetherness....

...can be rough when epidemics of nausea and vomiting strike a family. Emetrol offers prompt, safe relief. It is free from toxicity[1] or side effects[2,3] and will not mask symptoms of serious organic disorders.

1. Bradley, J. E., *et al.*: J. Pediat. *38*:41 (Jan.) 1951.
2. Bradley, J. E.: Mod. Med. *20*:71 (Oct. 15) 1952.
3. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. *65*:311 (Feb.) 1953.

RORER WILLIAM H. RORER, INC.
Fort Washington, Pa.

Emetrol®
phosphorated carbohydrate solution
emesis control

Measles Vaccine Is Safe and Effective

Extensive clinical tests show that a type of measles vaccine now in use consistently gives a high degree of immunity with only one immunization and without causing rashes or illness.

The safety and effectiveness of the vaccine should make the task of eliminating measles in this country somewhat easier.

A report of tests on 14,000 children in the U.S. and Mexico appears in the January 2 *Journal of the American Medical Association.*

The commercially available vaccine is termed a "highly (or further) attenuated live vaccine."

Two other types of measles vaccine are available and are effective. One type, however, has caused rash and temperature in 40 to 50 per cent of all children immunized, the report said. The second type does not cause significant reactions, but a series of injections is necessary to produce satisfactory measles protection. In some cases, this immunity has been relatively short-lived.

Clinical tests of the highly attenuated type of vaccine were done in Chicago, Cleveland, Detroit, Norfolk, Va., and Mexico City.

"It was very unusual," the report said, "for a vaccinated child to show more than two or three symptoms usually seen with natural measles." In most cases, these symptoms were a mild rash or a slight temperature.

"The effectiveness of this vaccine as an immunizing agent appears to be outstanding," the report said.

Vaccination against measles should be by now a "routine procedure," the report pointed out.

The American Medical Association has urged individual physicians and local and state medical societies to lead the drive to eliminate measles in this country. Ridding the nation of measles is possible in the foreseeable future, said a recent statement of the AMA's Council on Environmental and Public Health.

The Council made these recommendations:

—All children should be immunized when approximately 12 months old.

—Eventually, all measles-susceptible children should be immunized, but the first effort should be to protect infants and those entering kindergarten and the first two years of elementary school.

—Older children should be immunized only on an individual basis, and not as part of a mass vaccination program. An exception, the Council said, is when large numbers are measles-susceptible, as in isolated geographic regions.

Those who cooperated in the massive vaccine test include Anton J. F. Schwarz, M.D., and John T. Anderson, M.D., of the Pitman-Moore Division of Dow Chemical Co.; Manuel Ramos-Alvarez, M.D., Hospital Infantil de Mexico, Mexico City;

(Continued on Page 17)

individuality

. . . that quality which distinguishes one person from another —is a primary consideration in the planning of Devereux residential treatment programs for mentally retarded and emotionally disturbed children. In recognition of the unique nature of each child, Devereux Schools, since its inception in 1912, has sought to meet the needs of children on the basis of this individuality. . . through the provision of individual and group psychotherapies, educational and vocational guidance and the development of a therapeutic environmental setting.

DEVEREUX SCHOOLS
UNDER THE DEVEREUX FOUNDATION
A NONPROFIT ORGANIZATION

Helena T. Devereux — *Founder and Consultant*
Edward L. French, Ph.D. — *President and Director*

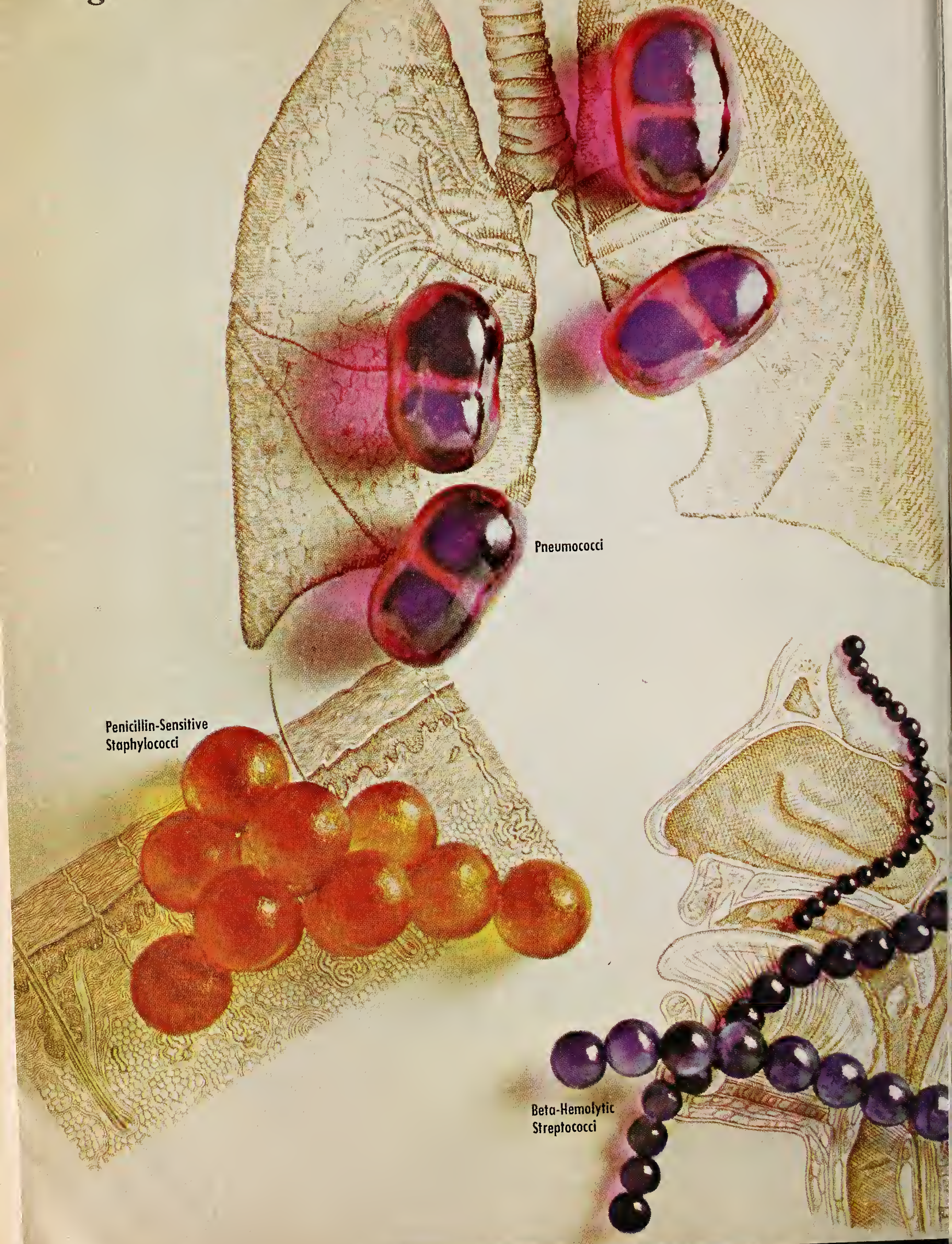
FOR INFORMATION AND LITERATURE

Santa Barbara (Box 1079), California
Keith A. Seaton, Director of Admissions

Devon, Pennsylvania and Rutland, Massachusetts
Charles J. Fowler, Director of Admissions
Devereux Schools, Devon, Penna. 19333

Victoria (Box 2666), Texas
Richard D. Grant, Registrar

Against these three major pathogens...


Pneumococci
Penicillin-Sensitive Staphylococci
Beta-Hemolytic Streptococci

Measles Vaccine Is Safe and Effective

(Continued from Page 9)

Morton B. Andelman, M.D., Chicago Board of Health Clinics; James F. Crosby, M.D., Norfolk, Va., General Hospital; James A. McKay, M.D., Western Reserve University, Cleveland, and Morris Medalie, M.D., Detroit Metropolitan Hospital, Detroit.

College Freshmen and Smoking

When 3,567 freshmen at the University of Illinois, Urbana, were questioned about their smoking habits, the investigators found these things:

—There was an inverse association between grade average and smoking: Of students with an "A" average, only 16.7 per cent smoked, while 59.1 per cent of "E" average (the lowest grade) students smoked.

—Sixty per cent of the freshmen did not smoke, and 43 per cent of those who smoked wanted to stop.

—The amount of allowance seemed to have a relationship to smoking. When parents provided full financial support, the number of freshmen who smoked was above average. The average weekly spending money was slightly over $8. When it was more than $5, the percentage of smokers was above average. Only 20 per cent of those who received sole financial support from a scholarship smoked.

—Of those who smoked 12 months ago, 56 per cent had increased the number of cigarettes smoked per day. More than 27 per cent smoked a pack a day or more.

These and other findings are reported in the January 2 *Journal of the American Medical Association*. The study, by Dorothy F. Dunn, Ph.D., of the University's Student Health Center Department of Health Science, was financed by a grant from the AMA Education and Research Foundation.

Eight out of 10 freshmen believed students were adequately informed regarding the potential dangers of cigarettes, and nine in 10 believed that the cigarette smoker had a greater chance of developing lung cancer than the nonsmoker.

It appears that the majority of students may be more ready to tighten university smoking regulations than their campus elders, Dr. Dunn said.

She urged that colleges and universities reconsider their smoking regulations, and that they institute programs to take the social pressure to smoke off freshmen.

"How much longer can colleges and universities enjoy the status quo and collect revenue from campus cigarette sales knowing that smokers may pay later with years of disability or premature death? Freshmen who do not wish to smoke comprise a group toward which universities should focus effort in prevention," she said.

...but doctor,
I eat like a bird!

IN WEIGHT CONTROL

OBETROL®

Each OBETROL-10 tablet contains: Methamphetamine Saccharate 2.5 mg., Methamphetamine Hydrochloride 2.5 mg., Amphetamine Sulfate 2.5 mg., Dextro-amphetamine Sulfate 2.5 mg. (OBETROL-20 tablets contain twice this potency) Pat. #2748052.

This combination of amphetamines may be useful as an adjunct in the management of certain forms of obesity where an appetite depressant is indicated.

CONTRAINDICATIONS: Hypertension, advanced arteriosclerosis, coronary artery disease, cardiac arrhythmias, peripheral vascular disease, states of undue restlessness, anxiety, excitement, agitated depression, hyperthyroidism, idiosyncrasy to amphetamine, concomitant administration of a monoamine oxidase inhibitor. PRECAUTIONS: Use with caution in individuals with anorexia, insomnia, vasomotor instability, asthenia, psychopathic personality, a history of homicidal or suicidal tendencies, and individuals who are known to be hyperreactive to sympathomimetic agents, or emotionally unstable individuals who are known to be susceptible to drug abuse. Certain monoamine oxidase inhibitors may potentiate the action of Obetrol. SIDE EFFECTS: The most common side effects attended with the use of amphetamines include nervousness, excitability, euphoria, insomnia, dryness of mouth, nausea, vertigo, constipation, and headache. DOSAGE AND ADMINISTRATION: Initial adult dose is one-half to one 'Obetrol-10' tablet daily, preferably one-half to one hour before meals. This may be gradually increased to one 'Obetrol-10' or 'Obetrol-20' tablet one to three times daily as indicated. SUPPLIED: Tablets scored, in bottles of 100, 500, and 1000.

REQUEST SAMPLES AND LITERATURE

OBETROL PHARMACEUTICALS
Div. of Rexar Pharmacal Corp. Brooklyn, N.Y. 11207

OBETROL PHARMACEUTICALS • BROOKLYN, N.Y. 11207

Dr. ____________________

Address ____________________

City ____________ State ____________ Zip ______

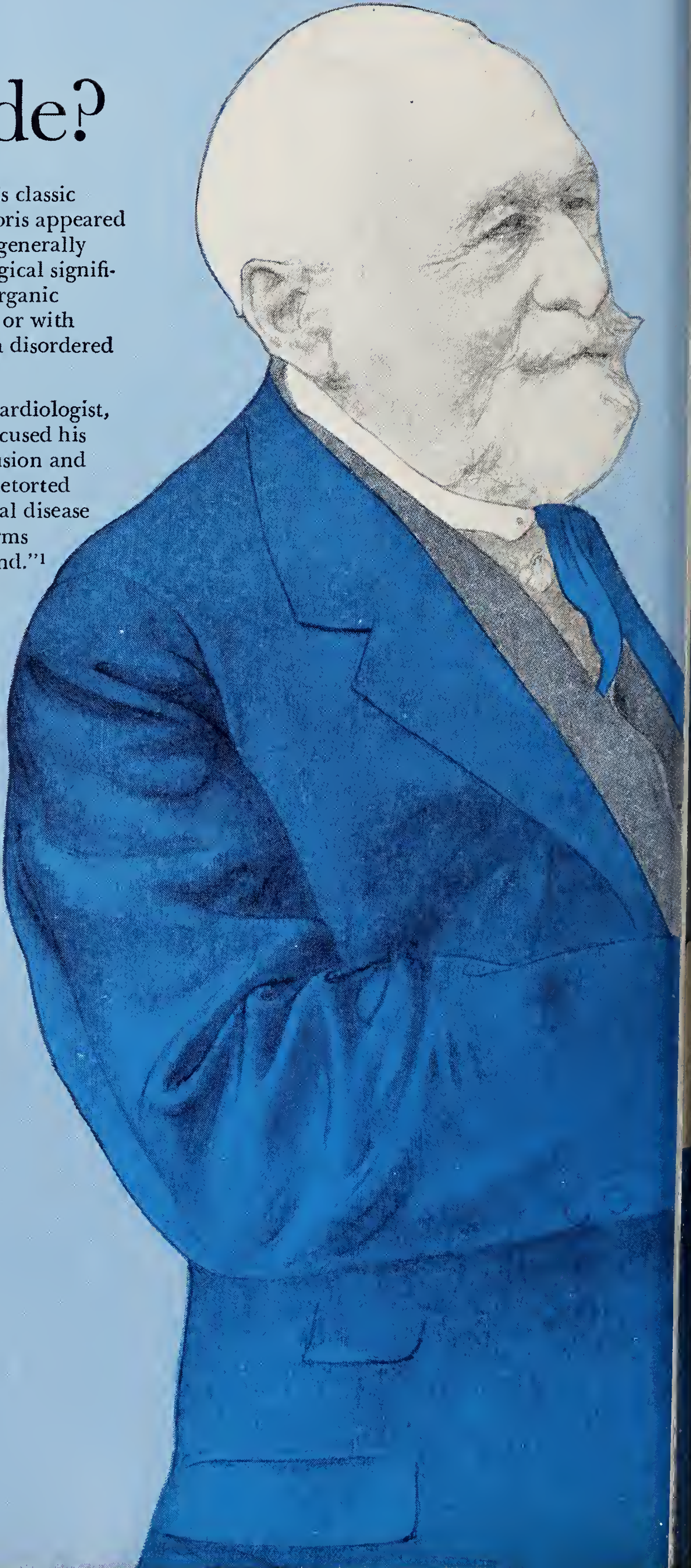
Vignettes of angina pectoris
no. 2 in a series:

Allbutt condemns Victorian complacency

angina or angstzustände?

Fully a century and a half after Heberden's classic description, this statement on angina pectoris appeared in a leading British medical journal: "Its generally admitted symptoms have no fixed pathological significance; they may be associated with grave organic disease of the heart and coronary arteries, or with nothing more serious than a heavy meal, a disordered stomach, or excessive smoking...."[1]

This article so incensed the noted British cardiologist, Sir Clifford Allbutt (1836-1925), that he accused his medical contemporaries of fostering confusion and complacency about the dread condition. Retorted Allbutt: "In other words, trivial and mortal disease are much the same thing, and technical terms may be blown about like straws in the wind."[1]



Mediatric®

Designed for the "metabolically spent"

Nutritional reinforcement for those who can't – or won't – eat properly...balanced amounts of estrogen and androgen to counteract declining gonadal hormone secretion and its sequelae of premature degenerative changes...mild antidepressant for a gentle "mood" uplift...

The estrogen component in MEDIATRIC is PREMARIN® (conjugated estrogens-equine), the natural estrogen most widely prescribed for its superior physiologic and metabolic benefits.

MEDIATRIC also provides *nutritional reinforcement—blood-building factors and vitamin supplementation*. It contributes a *gentle "mood" uplift* through methamphetamine HCl.

Three different dosage forms—Liquid, Tablets, and Capsules—offer convenience and variety.

MEDIATRIC Liquid

Each 15 cc. (3 teaspoonfuls) contains:

*Conjugated estrogens-equine (Premarin®)	0.25 mg.
Methyltestosterone	2.5 mg.
Thiamine HCl	5.0 mg.
Cyanocobalamin	1.5 mcg.
Methamphetamine HCl	1.0 mg.

Contains 15% alcohol

MEDIATRIC Tablets and Capsules

Each MEDIATRIC Tablet or Capsule contains:

*Conjugated estrogens-equine (Premarin®)	0.25 mg.
Methyltestosterone	2.5 mg.
Ascorbic acid	100.0 mg.
Cyanocobalamin	2.5 mcg.
Intrinsic factor concentrate	8.0 mg.
Thiamine mononitrate	10.0 mg.
Riboflavin	5.0 mg.
Niacinamide	50.0 mg.
Pyridoxine HCl	3.0 mg.
Calc. pantothenate	20.0 mg.
Ferrous sulfate exsic	30.0 mg.
Methamphetamine HCl	1.0 mg.

*Orally active, water-soluble conjugated estrogens derived from pregnant mares' urine and standardized in terms of the weight of active, water-soluble estrogen content.

MEDIATRIC helps keep the older patient alert and active; helps relieve general malaise, easy fatigability, vague pains in the bones and joints, loss of appetite, and lack of interest usually associated with declining gonadal hormone secretion.

CONTRAINDICATION: Carcinoma of the prostate, due to methyltestosterone component.

WARNING: Some patients with pernicious anemia may not respond to treatment with the Tablets or Capsules, nor is cessation of response predictable. Periodic examinations and laboratory studies of pernicious anemia patients are essential and recommended.

SIDE EFFECTS: In addition to withdrawal bleeding, breast tenderness or hirsutism may occur.

SUGGESTED DOSAGES: *Male and female:* 3 teaspoonfuls of Liquid, 1 Tablet, or 1 Capsule, daily or as required.

In the female: To avoid continuous stimulation of breast and uterus, cyclic therapy is recommended (3 week regimen with 1 week rest period—Withdrawal bleeding may occur during this 1 week rest period).

In the male: A careful check should be made on the status of the prostate gland when therapy is given for protracted intervals.

SUPPLIED: No. 910 – MEDIATRIC Liquid, in bottles of 16 fluidounces and 1 gallon. No. 752 – MEDIATRIC Tablets, in bottles of 100 and 1,000. No. 252 – MEDIATRIC Capsules, in bottles of 30, 100, and 1,000.

16 fl. oz.
No. 910
MEDIATRIC LIQUID
Ayerst
MEDIATRIC TABLETS
SHAKE WELL BEFORE USING

Mediatric®

steroid-nutritional compound

AYERST LABORATORIES, NEW YORK, N.Y. 10017 • Montreal, Canada

6636

OFFICERS

OF THE CALIFORNIA MEDICAL ASSOCIATION

(Continued from Page 5)

Committee on Health Facilities Planning

Laurence A. Mosier, Garden Grove	1967
John W. Gates, Visalia	1967
Joseph W. Telford, San Diego	1967
Jean F. Crum, Downey	1968
Glenn A. Pope, Sacramento (Chmn.)	1968
Richard Taw, Los Angeles	1968
James W. Dalton, Santa Barbara	1969
Paul C. Doehring, Glendale	1969
Pierre Salmon, San Mateo	1969

CONSULTANTS: Robert F. Schell, San Rafael; Jack Lomas, Beverly Hills.

COUNCIL COMMITTEES

Finance Committee

Harold Kay, Oakland (Chmn.); Roger C. Isenhour, San Diego; Frank C. Melone, Ontario; Joseph W. Telford, San Diego; Malcolm C. Todd, Long Beach.

Committee on Legislation

Dan O. Kilroy, Sacramento (Chmn.); Stuart C. Knox, Los Angeles; Harold E. Wilkins, Downey; John Rumsey, San Diego; Samuel R. Sherman, San Francisco.

Advisory Board to Woman's Auxiliary

James C. MacLaggan, San Diego (Chmn.); John G. Morrison, San Leandro; William F. Quinn, Los Angeles; Carl E. Anderson, Santa Rosa; Eugene Hoffman, Los Angeles.

Benevolence Fund Operating Committee

Clyde L. Boice, Palo Alto (Chmn.); Alexander Fraser, San Francisco; Elizabeth Mason Hohl, Los Angeles; Don C. Musser, San Francisco; George Wolf, Fresno; Dudley M. Cobb, Jr., Los Angeles.

MEDICAL OFFICER
Pacific Missile Range
Point Mugu, California

The Pacific Missile Range, Point Mugu, California, has an opening for Medical Officer (General Practice), GS-13 (Federal Civil Service) at a salary of $14,595 per annum.

Incumbent will be responsible to the Senior Medical Officer in providing outpatient care to authorized medical care recipients. Duties will involve care of active duty military personnel, authorized categories of civilian personnel, and military dependents.

Further information may be obtained by writing to the Medical Department, Code 672, Pacific Missile Range, Point Mugu, California; or call collect area code 805, 488-3511, extension 8137 (CAPT Niforopulos or LCDR Tanner).

U.S. Citizenship Required
An Equal Opportunity Employer

On U.C. Extension Courses In Continuing Medical Education

University of California Extension, the statewide education service which provides an intellectual bridge between the University and more than 200,000 adult citizens, the largest educational organization of its kind, is marking its 75th anniversary.

In a 1965-66 annual report issued to officers, regents and friends of the University by Paul H. Sheats, statewide Dean of University Extension, he noted that Los Angeles accounted for 46.3 per cent of total University Extension registrations. Offering 3,208 programs, the local office recorded 98,663 registrations, and a student full-time equivalent total of 9,280, an increase of 24.3 per cent over the previous reporting period.

Noteworthy in the Los Angeles report were the contributions of Continuing Education in Medicine and Health Sciences, an innovation of the 1940s. About 25 per cent of courses offered by this department during 1965-66 were new. Medical Television Network programs, as a result of a grant from the American Cancer Society, expanded and broadened medical topics in relation to psychiatry. Four programs for nurses were added. Last year the Network also began dubbing programs onto videotapes for distribution to hospitals outside broadcast range, greatly increasing the viewing audience, the report stated.

Two symposia presenting results of current research—one concerning birth control, the other on drugs—presented nationally known speakers. Proceedings of the birth control conference will be made permanent in a soon-to-be-published volume entitled "Birth Control—A Continuing Controversy." Another innovation was the planning and administration of the annual meeting of the American Physiological Society, with an attendance of more than 1,000.

Partially supported by a grant from the Kellogg Foundation, Extension's Western Center for Continuing Education in Hospitals and Related Health Institutions was organized in 1965 to meet the training needs of health care personnel, the report explained. Assisted by the center, University Extension at Los Angeles activities included a three-day institute in dietetics and nutrition for extended care facilities, organized with the California Dietetic Association, the California Association of Nursing Homes and the Southern California Nursing Home Association; and a three-day conference dealing with the administrator's role in the development of medical evaluation committees for nursing homes.

A total of 37 community organizations participated in programs presented by Continuing Education in Medicine and Health Sciences during the past year, the report concluded.

Course in Medical Care Offered by UC Extension

A course for health services administrators, physicians, nurses and related health welfare and insurance personnel in "Medical Care: Basic Issues and Current Problems" will be offered by University of California Extension's Continuing Education in Medicine and Health Sciences.

The course will meet on Tuesday, February 21 to May 2, 7 to 9:30 p.m., at Extension's Downtown Center, 1100 South Grand Avenue. Co-sponsored by the UCLA School of Public Health, it will examine patterns of medical care organization in the U.S. and their implications with respect to demand for and delivery of health services.

Principles and methods of health insurance, voluntary health organizations, public assistance medical care programs, and private purchase of medical care are among the basic issues and problems to be studied. Emphasis will be on the concept of organization as it may be applied to medical care.

Instructor will be Donald M. Dubois, Dr.P.H., assistant professor of medical care organization, School of Public Health.

For more information contact Continuing Education in Medicine and Health Sciences, Room 15-39, Rehabilitation Center, University of California, Los Angeles, California 90024, or telephone 272-8911 or 478-9711, extension 4345.

Reports Agree: No Big Reduction In Fats Is Warranted

No radical changes in the American diet are warranted on the basis of present knowledge about the role of food fats in cardiovascular disease.

This conclusion was drawn in a recent report on "Dietary Fat and Human Health," issued by the Food and Nutrition Board of the National Academy of Sciences-National Research Council (NAS-NRC).

It is essentially one of the major conclusions reached by the Council on Foods and Nutrition of the American Medical Association in reports issued in 1962 and 1965. The NAS-NRC report is a revised and updated version of a document first published in 1958.

The 1965 report by the AMA Council reaffirmed its 1962 position that "there is not sufficient information available at the present time to warrant a change in the American diet aimed at preventing heart disease in the general population."

General agreement also is found in the NAS-NRC and AMA Council reports on criteria for limiting food fats in the diet.

The Council on Foods and Nutrition said in its 1965 annual report:

(Continued on Page 34)

DID 1966 TAXES HURT?

START YOUR 1967 TAX PLANNING NOW!

Commercial Finance Co. has several prime properties which it must liquidate at the earliest possible moment. These properties include income units, commercial, and land. There are inherent tax advantages in owning any of these. Bonus depreciation, prepaid interest, and capital gain appreciation to mention a few.

OUR LOSS IS YOUR GAIN!!

WE must dispose of these excess properties. As a publicly held company, we can't take advantage of these tax benefits, but YOU can.

NAME YOUR TERMS. CASH TALKS!

DO IT NOW.

CLIP THE COUPON AT THE RIGHT.

Why Not Form a Syndicate with Your Associates?

LUMBERMANS MORTGAGE CO.
306 Mendocino Ave.
Santa Rosa, Calif. 95404

Yes, I am interested in receiving information on properties which you now have and all future properties you may acquire. I am particularly interested in the following types of properties:

INCOME ☐ DR.____________________
LAND ☐
COMMERCIAL ☐ ____________________
RESIDENTIAL ☐
INDUSTRIAL ☐ ____________________
TRUST DEEDS ☐

★

Specialized Service
IN
PROFESSIONAL LIABILITY
INSURANCE

is a high mark
of distinction

THE
MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA
Professional Protection Exclusively since 1899

NORTHERN CALIFORNIA OFFICE:
Gordon C. Jones and John K. Galloway, Representatives
1518 Fifth Avenue, San Rafael, Telephone: 415-453-5140
Mailing Address: P.O. Box 1079, San Rafael 94902
SOUTHERN CALIFORNIA OFFICE:
Davis S. Spencer and Grady T. Dyer, Representatives
Suite 3, 33 East Huntington Drive, Arcadia, Tel. 213-681-5239
Mailing Address: P.O. Box 543, Arcadia 91008

Easy on the Budget... Easy on the Mother

Tablets & Elixir
For Iron Deficiency Anemia

FAMOUS
Fergon®
brand of FERROUS GLUCONATE

C.M.A.P. CODE # 5261

BREON LABORATORIES INC.
Subsidiary of Sterling Drug Inc.
90 Park Avenue, New York, N.Y. 10016

Reports Agree: No Big Reduction In Fats Is Warranted

(Continued from Page 33)

"The Council notes that after puberty, American men, and to a lesser extent women, show a progressive rise in serum betalipoprotein content and the associated cholesterol, phospholipid, and triglycerides until age 60. Present knowledge suggests that this rise is associated with an increasing risk of coronary disease in men, whereas women are relatively protected until after menopause. The Council believes that the development of coronary disease in young American men may be attributed to a combination of unfavorable genetic and environmental factors. One environmental factor of importance to the genetically predisposed person may be the American diet."

The Council recommended that physicians advise persons with an abnormal excess of fatty materials in the blood to undertake diet therapy aimed at lowering the concentration.

"In addition," the Council said, "it recommends that physicians consider offering similar diet modifications to young men vulnerable to coronary disease in an attempt to prevent the rise of serum lipids which, in time, might put them into high-risk categories."

The Food and Nutrition Board of NAS-NRC points out in its report that: (1) Fats play a vital role in nutrition, and any drastic reduction in fat consumption would alter body metabolism in unpredictable and possibly harmful ways; (2) no positive case has ever been made against dietary fats as a direct cause of cardiovascular disease; (3) nevertheless, the attractiveness and availability of rich or fatty foods place the American consumer in some danger of overnutrition and dietary imbalance.

Therefore, the Board says: "Until we learn more about which fats are desirable nutritionally, the Board recommends that the American consumer should partake of the foods that make up a varied, adequate, and not overly rich diet and maintain a normal body weight by judicious control of caloric intake and by daily exercise."

For many Americans, says the Board, a moderate reduction in total fat intake and some substitution of polyunsaturated for saturated fat may be indicated for purposes of dietary balance. "The degree to which this is done must be judged on an individual basis and, in adjustment of the diet, other changes of caloric and nutrient intake must be taken into consideration," according to the Board.

The AMA Council recommends that adjustment of dietary intake and the ratio of saturated to polyunsaturated fats be made under the supervision of a physician.

Does he really care? Is he alert, encouraged, positive and optimistic about getting out of bed and back to work soon?

Or is he giving in to the depressing impact of confinement?

When functional fatigue complicates convalescence, Alertonic can help...

Pleasant-tasting Alertonic is pipradrol hydrochloride—an effective cerebral stimulant whose gentle analeptic action helps counteract the apathy and inertia that so often delay convalescence—together with an excellent vitamin and mineral formula, in a satisfying 15% alcohol vehicle.

Nothing fosters confidence and a sense of well-being better than your own personal warmth, understanding and encouragement together with Alertonic to help insure prompt response.

Adequate dosage is important: Prescribe Alertonic—one tablespoonful t.i.d., 30 minutes before meals...tastes best chilled.

And for your patient's sake, prescribe Alertonic in the convenient, economical one-pint bottle.

Alertonic®

Available Only On Prescription

Each 45 cc. (3 tablespoonfuls) contains: alcohol, 15%; pipradrol hydrochloride, 2 mg.; thiamine hydrochloride (vitamin B_1) (10 MDR*), 10 mg.; riboflavin (vitamin B_2) (4 MDR), 5 mg.; pyridoxine hydrochloride (vitamin B_6), 1 mg.; niacinamide (5 MDR), 50 mg.; choline,† 100 mg.; inositol,† 100 mg.; calcium glycerophosphate, 100 mg. (supplies 2% MDR for calcium and for phosphorus) and 1 mg. each of the following: cobalt (as chloride), manganese (as sulfate), magnesium (as acetate), zinc (as acetate), and molybdenum (as ammonium molybdate).

*Multiple of adult Minimum Daily Requirement supplied.
†The need for these substances in human nutrition has not been established.

Indications: 1. Functional fatigue such as that often associated with: a depressing life experience or stressful time of life; advancing years; convalescence; limited activity or confinement. 2. Poor appetite and vitamin-mineral deficiency as they occur in: patients having faulty eating habits; geriatric patients who are losing interest in food; patients convalescing from debilitating illness or surgery.

Contraindications: As with other drugs with CNS stimulating action, Alertonic is contraindicated in hyperactive, agitated or severely anxious patients and in chorea or obsessive compulsive states.

Side effects: Reports of overstimulation have been rare. Patients who are known to be unduly sensitive to the effects of stimulant drugs should be observed carefully in the initial stages of treatment.

Dosage: Adults, 1 tablespoonful; children (over 15 years old), 1 to 2 teaspoonfuls; children (4 to 15 years old), 1 teaspoonful. To be taken three times daily 30 minutes before meals.

Merrell

THE WM. S. MERRELL COMPANY
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215

6-7907

Preventi-Care or Preventi-Thought?

The name of the game is The Name when it comes to labeling federal activities in the field of health care, says an editorial in the January 2 *Journal of the American Medical Association.*

If the name of the program is right, it's difficult to oppose it and avoid being labeled a "bad guy," the editorial points out.

Who, for instance, wants to oppose "The Drug Abuse Control Amendments of 1965?" To do so implies that one favors drug abuse.

A closer look at the bill, however, reveals it is not concerned with drug "abuse," but with control and distribution of stimulant and depressant drugs.

"The floodgates have opened, and we may anticipate a deluge of dramatic titles which are capsulated value judgments rather than descriptive labels," says the editorial.

The following bills recently became law or are awaiting Presidential signature: "The Child Safety Act of 1966," "The Water Quality Act," and the "Economic Opportunity Act Amendments."

The newest catch title is "Preventicare," a newly proposed federal program for multiphasic health screening of all persons over 50 years of age.

"Now there, truly, is a press agent's dream (title)," the editorial said, "which will enlighten no one but will appeal to all who seek easy solutions."

Said the editorial:

"Physicians are not opposed to the constructive recommendations incorporated in many of these acts. Indeed, the American Medical Association has vigorously supported some of the bills cited. We believe, however, that prejudgment inhibits constructive debate. Neither sugar-coated titles nor the opposite strategy of scare campaigns serve the populace well in the long run. Medical legislative titles presented to the Congress and to a mature public should be characterized by a forthright approach if medical problems of this decade are to be solved most effectively."

Your public relations problem has been our prime consideration in collection procedures during two generations of ethical service to the Medical Profession.

✶

THE DOCTORS BUSINESS BUREAU

Since 1916

821 MARKET ST., SAN FRANCISCO 94103
GArfield 1-0460

"COCA-COLA" AND "COKE" ARE REGISTERED TRADE-MARKS WHICH IDENTIFY ONLY THE PRODUCT OF THE COCA-COLA COMPANY.

Coke
TRADE MARK
Coca-Cola
For the taste you never get tired of.

California Medicine

MEDICAL PROGRESS

Hirsutism and Virilization

RICHARD J. SHERINS, M.D., AND RICHARD HORTON, M.D., *Los Angeles*

■ *In normal females, androstenedione from both the adrenal cortex and ovary, as a result of peripheral conversion, is the source of the majority of biologically active testosterone in the circulation. The control of the secretion of precursor steroid and androgenic hormone (testosterone) in females is not clear at this time. There are a number of possibilities to explain various types of hirsutism and virilization. The presence of true virilization indicates a significant disorder and requires complete investigation.*

The presence of increased amounts of 17-ketosteroids in the urine implicates the adrenal cortex as a source of the pathologic manifestations. The suppressibility of elevated 17-ketosteroids with cortisol analogues aids in distinguishing between adrenal hyperplasia and autonomous neoplasm of the adrenal cortex.

By far the most common entity in this area is simple hirsutism without virilization. Although our knowledge of this disorder is quite incomplete, conservative management is indicated. Further progress in this field is rapidly occurring. An informed clinician can do an adequate job of diagnosis and treatment with the clinical and laboratory tools generally available.

TREMENDOUS advances in our knowledge of adrenal cortical and gonadal physiology and pathology have occurred in the last 25 years. Although many aspects of this field have been extensively investigated, the limitations of methodology have only recently allowed progress in our understanding of androgen metabolism. This review will of necessity deal with current concepts of physiology and classification. Since the knowledge in this field is so new and changing, this paper must be considered more a progress report, than an elucidation of fixed truths.

Androgens are substances which cause the development of the primary and secondary sexual tissues of the male and analogous structures in the female. Androgens have general anabolic effects upon muscle, skin and its appendages, fat distribution, behavior, and growth rate. These hormones

---

From the Department of Medicine, UCLA School of Medicine, Los Angeles. This work was supported by NIH grant AM 10019 and American Cancer Society P-404. Dr. Horton is the recipient of U.S.P.H.S. Career Development Award K3-AM-15955-01.

Reprint requests to: Department of Medicine, UCLA School of Medicine, Los Angeles 90024 (Dr. Horton).

interact on DNA-RNA exchange of information at the nuclear level and thereby influence protein synthesis.

Of the known androgenic substances, only testosterone, a C-19 steroid, and to a much lesser degree, androstenedione, appear significant (Chart 1). It is a general belief that hirsutism (excess body hair) in the female and virilization (the masculinization of a female, with clitoral hypertrophy, excess muscle development, hair recession, and amenorrhea) are dose effects of androgen. As little as 2 mg per day of testosterone can virilize an adult woman.

Recent work by one of the authors has indicated that there is a pronounced rise in circulating androgen between prepuberty and puberty. The amount of androgen necessary to virilize a prepubertal girl is probably much less than that required for an adult.

Although historically the Zimmerman method for estimating urinary ketosteroids opened up the *in vivo* steroid field in the 1930's, it has taken a long time to put the ketosteroids and biologically active androgens into their proper place in medicine. The urinary and plasma 17-hydroxycorticoids or ketogenic steroids are measures of cortisol (hydrocortisone) metabolism and will not be discussed further.

The 17-ketosteroids in urine include a considerable number of steroids which possess a ketone group at the 17 position, allowing the formation of a blue-red color with dinitrobenzene. It is important to realize that all of these substances are metabolites of parent steroid hormones and that they possess little or no biological activity and are present in a conjugated, water-soluble form as glucuronosides or sulfates. A minor fraction (10 to 15 per cent) is made up of breakdown products of cortisol from the adrenal gland. The majority of these urinary metabolites in both sexes are derived from a steroid, dehydroisoandrosterone (Chart 1), and its sulfate. They have minor androgenicity and are secreted by the adrenal cortex in amounts equal to or exceeding the secretion of cortisol (15 to 25 mg a day).[8] Thus, the overwhelming majority of the urinary 17-ketosteroids are derived from the adrenal cortex and represent weakly androgenic substances. Nevertheless, as will be described later, the urinary 17-ketosteroid determination widely available to the practicing physician can be used to rule *in* certain disorders.

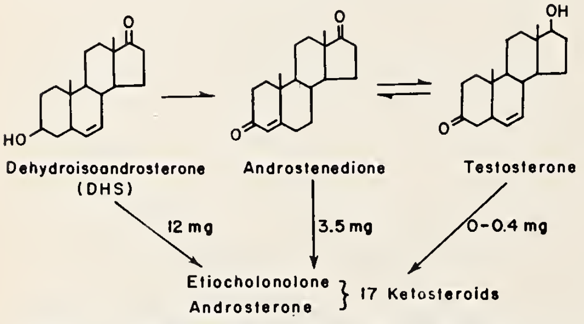


Chart 1.—The steroid precursors of the urinary C19-O$_2$ ketosteroids and production rates in normal females.

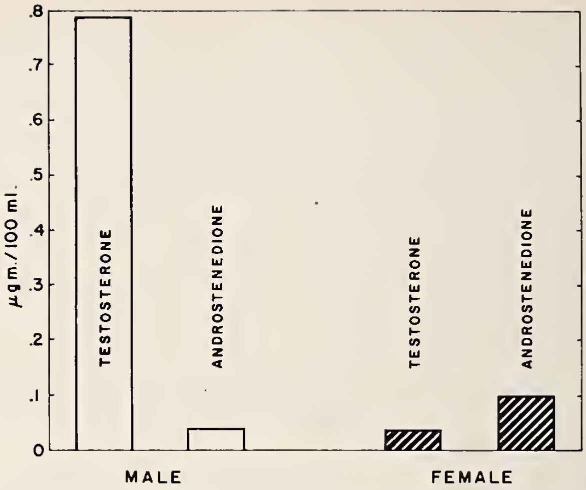


Chart 2.—Testosterone and androstenedione in human plasma.

In recent years, the use of isotopes and chromatography for steroid assay has permitted accurate and sensitive measurement of hormones in biological fluids. Adult male and female urine contains 80 mcg and 8 mcg a day of testosterone glucuronoside respectively.[2] In blood, testosterone is present in a mean plasma concentration of 0.7 mcg per 100 ml in males but only 0.04 mcg per 100 ml in females (Chart 2).[6,9] The total daily production of testosterone which enters the circulation in a normal adult human is approximately 7 mg for males but only 0.4 mg for females. There appears to be a direct feedback to the pituitary, since luteinizing hormone (usually in the commercial form of chorionic gonadotropin) will increase plasma testosterone, while synthetic testosterone analogues will reduce endogenous testosterone secretion.[4,5]

In males, the system appears relatively simple in that the adult Leydig cell of the testes secretes essentially all the testosterone that enters the blood. The female—as we all know—is a much more complex creature. Although the story is not complete, the following will attempt to summarize the current work. An adult female secretes, from the adrenal glands and the ovaries, about 3.5 mg a day of the weakly androgenic steroid androstenedione. This steroid is rapidly metabolized by the liver, as are most steroids. A glance at the chart of the structure of this steroid will indicate that it differs from the potent androgen testosterone only in the presence of a 17-ketone group instead of a 17-hydroxyl. This leads to the quip that a hydrogen ion is the only thing standing between a boy and a girl. Peripheral tissue has the capacity for making this interconversion. Most of this peripherally synthesized testosterone is conjugated, inactivated and excreted, but a fraction of it does enter the blood, where it provides about two-thirds of the total testosterone present in the plasma of females (Chart 3).[3]

Thus the possible source of excessive androgen in a female is:

- Increased secretion of testosterone by adrenal glands
- Increased secretion of testosterone by ovaries
- Increased secretion of androstenedione by adrenals
- Increased secretion of androstenedione by ovaries
- A combination of the above possibilities
- Abnormal conversion rates of androstenedione to testosterone

Since most of the testosterone synthesized in the liver does not enter the blood and circulate throughout the body as a biologically active hormone, the use of urinary testosterone metabolites in females gives very crude estimations of blood testosterone, and this approach should be interpreted with caution. In normal males the urinary approach does reflect circulating testosterone.

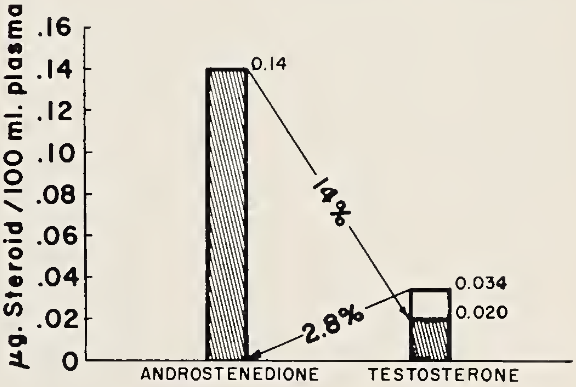
Virilizing disorders almost certainly indicate the presence of relatively large amounts of testosterone in blood. The clinical findings of virilization include most of the following: Deepening of the voice, severe acne, recession and balding of head hair, excessive facial and body hair, flushing of face and chest, amenorrhea, clitoral hypertrophy, sexual hyperactivity, increased muscle development and strength and changes toward a masculine habitus. The presence of a number of these findings warrants extensive clinical and laboratory diagnostic studies, since no better indicator of elevated testosterone could be obtained than this type of clinical bioassay.

The differential diagnosis of virilization includes various neoplastic or hyperplastic states of the adrenal cortex and ovary. Exogenous androgen or the various anabolic agents can mimic this process.

Cushing's disease, whether bilateral hyperplasia, adenoma or carcinoma, should be mentioned, although this is principally a problem of cortisol hypersecretion which involves signs and symptoms primarily of cortisol excess and can be diagnosed by the presence of elevated cortisol in plasma, lack of diurnal variation and increased urinary metabolites (17-hydroxycorticoids or ketogenics).

Congenital adrenal hyperplasia is a virilizing syndrome due to a biosynthetic defect in cortisol production which is overcome by increased stimulation of pituitary ACTH, but at the price of heightened secretion of dehydroisoandrosterone, androstenedione and other steroid precursors. As a result, in patients with this condition the ketosteroids are higher than in other persons of the same age group, and there is increased androstenedione and testosterone in the blood. In urine, both the 17-ketosteroids and pregnanetriol are increased. A physiological dose of glucocorticoid such as prednisone 2.5 mg three times a day or dexamethasone 0.5 mg twice a day will suppress the ketosteroids and pregnanetriol and serve as both diagnostic test and long-term therapy.

Rarely, an adenoma or carcinoma of the adrenal cortex will be present and lead to production of biologically active androgens. In this neoplastic disorder of the adrenal cortex, there is considerable disorganization of steroid biosynthesis, leading to production of large amounts of dehydroiso-



FEMALE PERIPHERAL BLOOD

Chart 3.—The contribution of androstenedione to plasma testosterone in females. The hatched areas represent androstenedione secretion and contributions. The arrows represent the rates of conversion. The clear area represents secreted testosterone.

androsterone and its sulfate. Hence the urinary 17-ketosteroids are almost always strikingly elevated, typically to between 40 and 150 mg a day (normal 5 to 15 mg). As is generally true of a neoplasm, no central nervous system pituitary control of secretion rate is apparent and even large doses of a cortisone analogue such as dexamethasone (2 mg four times a day) will not suppress the elevated ketosteroids.

The ovary may be the site of the abnormal androgen secretion. The Stein-Leventhal or polycystic ovary syndrome is a rather variable disorder whose essence is menstrual abnormalities, relative infertility and usually hirsutism with only occasional and mild degrees of virilization. The polycystic ovary and perhaps the adrenal glands secrete moderate amounts of testosterone and androstenedione. Since the ovary is the primary site of disease, minimal increases in dehydroisoandrosterone secretion are present and the urinary 17-ketosteroids are normal or only slightly elevated. The diagnosis can be made by the overall picture. Ovarian sclerocystosis, usually with enlargement, can be determined by careful pelvic examination or culdoscopy.

Prednisone in low dosage (5.0 to 7.5 mg a day in divided doses) has been effective in the majority of cases, especially in fertility control. Clomiphene (50 mg twice a day) for five to seven days, perhaps by altering the ratio of secreted pituitary gonadotropin, causes ovulation in up to 75 per cent of patients, and conception occurs in approximately half of them. Wedge resection, an unknown stimulus, is less often utilized at the present time as a therapeutic approach.

Again as in the adrenal glands there are very rare masculinizing tumors which carry a plethora of names based upon histological appearance (arrhenoblastoma, luteoma, thecoma, among others) although endocrinologically they behave similarly. These tumors secrete a potent androgen, probably testosterone. The ketosteroids are normal or low. A significant number of these masculinizing ovarian tumors are not palpable by the bimanual examination. However, the presence of frank virilization should lead to complete diagnostic studies, including exploratory laparotomy if necessary. To recapitulate, the presence of normal or low urinary 17-ketosteroids implicates the ovary. The masculinizing ovarian tumor is rare. Perhaps one or two a year are seen at major university centers. Very rarely an undifferentiated carcinoma or adrenal rest of the ovary will also secrete dehydroisoandrosterone, elevate the urinary 17-ketosteroids and resist suppression with cortisol analogues.

A recurrent theme in this discussion is the use of the urinary 17-ketosteroid assay. Although not a representation of androgen level, the ketosteroids can be used to implicate and rule in adrenal disease. The suppression with exogenous steroid generally differentiates between hyperplasia and autonomous adrenal tumor. The finding of normal or low urinary ketosteroid suggests an ovarian source (Chart 4).

This brings us to the major entity, at least in terms of the physicians' experience. Perhaps 90 per cent of all cases of hirsutism are "idiopathic" or simple hirsutism. Patients with simple hirsutism, whether obese or of normal body build, have varying degrees of increased facial and body hair and frequently mild acne and oily skin. No other signs or symptoms of virilization are present. A few patients have menstrual abnormalities, but associated psychological factors are usually involved. Most endocrinologists, especially those active in this particular field, feel that these patients represent a diverse group of mild disorders. This includes hereditary influences, obesity with some hypothalamic abnormality, local factors bearing on the hair follicle and perhaps mild abnormalities in androgen secretion or peripheral metabolism. A minor group have slightly elevated urinary ketosteroids, but otherwise normal adrenal function. The entire group suppress normally (>50 per cent) with small doses of a cortisol analogue administered for one week.

The major point here is to recognize the most common syndrome in the androgen area. An adequate investigation should include a careful pelvic

Chart 4.—Female Androgens.

URINARY METABOLITES

(a) *Urinary androsterone and 5β-androsterone* arise mainly from androstenedione produced from dehydroisoandrosterone. This androstenedione is poorly converted to testosterone in the liver and even less to testosterone in the blood.

(b) *Urinary testosterone glucuronide* arises mainly from secreted androstenedione. This testosterone is poorly transferred to testosterone in the blood.

BLOOD TESTOSTERONE

(c) *Sixty per cent blood testosterone* arises mainly from secreted androstenedione. This testosterone may be produced extrahepatically or in the liver.

(d) *The remaining 40 per cent of the blood testosterone* may be secreted, or produced in the liver from dehydroisoandrosterone, or both. It is most likely to be secreted testosterone.

examination, the exclusion of virilizing signs and the presence of normal urinary 17-ketosteroids (or suppressibility if slightly elevated to 50 per cent of initial value).

Even though the disorder is not completely understood at this time, making a positive diagnosis is important. Some endocrinologists have suggested long-term steroid suppression. The authors are not convinced of the efficacy of this approach. Only occasionally will hair growth be significantly reduced by this means. Reliance must be placed at this time on electrolysis and reassurance to the patient that this is not a serious disorder. The facial hair can be removed and controlled. On the research horizon is the possibility of using antiandrogens,[1] or other drugs.

No mention has been made of the use of a plasma testosterone estimation. This determination is available in a number of research laboratories in this country (including the authors') and also available in a few specialized commercial laboratories. The authors feel at this time that this procedure remains a research tool. In the near future, in selected cases of virilization a practical approach may be to study the effect of corticotropin or gonadotropin on plasma testosterone and androstenedione to determine the site and responsiveness to appropriate stimuli. The presence of virilization unquestionably indicates elevated testosterone. Simple hirsutism may or may not be associated with elevated testosterone levels. What appears to count are the clinical indications as noted above for an aggressive approach including culdoscopy or laparotomy. This attitude has been reached as a result of both clinical and research experience.[7,10]

However, it is certainly helpful to have further documentation and quantitation. The major point of this paper is to correlate the research findings with the everyday clinical problem and to point out that proper diagnosis can almost always be made with the widely available tools that an informed physician and a typical clinical laboratory can effectively use.

REFERENCES

1. Boris, A., and Stevenson, R. H.: Further studies on an antiandrogen, Endocrinology, 78:549, 1965.

2. Camacho, A., and Migeon, C. J.: Studies on origin of testosterone in urine of normal adult subjects and patients with endocrine disorders, J. Clin. Invest., 43:1083, 1964.

3. Horton, R., and Tait, J. F.: Androstenedione production and interconversion rates in blood and studies of the sites of conversion to testosterone, J. Clin Invest., 45:301, 1966.

4. Hudson, B., Coghlan, J. P., Dulmanis, A., and Wintour, M.: The measurement of testosterone in biological fluids in evaluation of androgen activity, Proc. 2nd Internation Congress of Endocrinology Excerpta Medica, 83: 1127, 1965.

5. Korenman, S. G., Wilson, H., and Lipsett, M. B.: Testosterone production rates in normal adults, J. Clin. Invest., 42:1753, 1963.

6. Lamb, E. J., Dignam, W. J., and Simmer, H.: Plasma androgens in women, Acta Endocr., 45:243, 1964.

7. Lipsett, M., and Korenman, S. G.: Androgen metabolism, J.A.M.A., 190:147, 1964.

8. MacDonald, C., Gonzalez, O., Vande Wiele, R. L., and Lieberman, S.: Excretion of radioactive 17-ketosteroid conjugates following intravenous administration of ^{3}H dehydroisoandrostene, J. Clin. Endocr., 23:665, 1963.

9. Riondel, A., Tait, J. F., Gut, M., Tait, S. A. S., Joachim, E., and Little, B.: Estimation of testosterone in human peripheral blood, J. Clin. Endocr., 23:620, 1963.

10. Vermeulen, A.: Androgens in normal and pathological conditions, Excerpta Medica Series No. 101, The Netherlands, 1966.

Needle Aspiration Biopsy of Localized Pulmonary Lesions

G. MELVIN STEVENS, M.D., GLEN A. LILLINGTON, M.D., AND
JOHN F. WEIGEN, M.D., *Palo Alto*

■ *Needle aspiration biopsy of localized pulmonary lesions is a direct, safe, relatively inexpensive method of diagnosis which is applicable for both in-patients and out-patients. It should be performed under direct visualization with image amplifier guidance. Proper application of this method can greatly increase diagnostic yield, diminish the diagnostic interval and expense and minimize the risks involved.*

NEEDLE ASPIRATION BIOPSY of the lung offers a virtually painless, simple and relatively inexpensive method of diagnosis of many localized pulmonary lesions. The use of a small caliber biopsy needle and image amplifier fluoroscopic guidance has made lesions as small as one centimeter in diameter accessible for biopsy, regardless of whether they are in the peripheral or central portion of the lung. Biopsy of mediastinal lesions by the same technique is perfectly feasible.[6,13]

The advantages of this procedure were first described by Ménétrier in 1886,[10] and have been pointed out by other investigators since.* An unfounded belief prevails that complications are frequent and that dissemination of malignant tumor or infection along the needle tract is common. The experience of several investigators refutes this theoretical concern.[2,4,6,9,11]

It is the purpose of this paper to describe our very favorable early experience in the diagnosis of a variety of intrapulmonary lesions, in which no specific diagnosis had been established despite extensive investigations. We believe that more widespread knowledge and use of needle aspiration biopsy is to be encouraged.

Method

With the patient lying on the x-ray table in the optimal position for aspiration of the lesion, fluoroscopic localization is carried out. The tip of a hemostat is placed in contact with the skin directly over the lesion, during moderate inspiration, to indicate the appropriate needle entry site. A small metallic marker, fixed to the skin by tape, then replaces the hemostat. After fluoroscopic confirmation that it is accurately placed, the marker is pressed into the skin so that an imprint remains when it is removed. Skin preparation with germicidal solution follows and the site of entry is infiltrated with local anesthetic down to the pleural surface. A minute bistoury blade stab wound is made in the skin to diminish skin friction on the needle shaft so that variations in lung consistency can be easily felt. A No. 18 lumbar puncture needle was initially used for biopsy. More recently this has been replaced by a No. 18 thin walled aortography type needle. Needles of four-inch and six-inch lengths will suffice in most cases. The needle is inserted under direct fluoroscopic control, while held by a two-

---

From the Department of Radiology and the Department of Internal Medicine, Chest Disease Section, Palo Alto Medical Clinic, Palo Alto.

Presented before the Section on Radiology at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: Department of Radiology, Palo Alto Medical Clinic, 300 Homer Avenue, Palo Alto 94301 (Dr. Stevens).

*Reference Nos. 1 to 6, 8, 12, 13, 15.

piece radiolucent balsa wood holder which allows the physician's hands to remain outside the fluoroscopic field. The stylus is kept in place until the peripheral edge of the lesion is reached. It is then withdrawn as the needle is advanced into the lesion. A 20 ml Luer-Lock syringe is attached to the needle, and constant suction is applied while the needle is advanced farther into the lesion. The yield is considerably improved by rotation of the needle while one or two short in-and-out penetrations are made. Vigorous suction with the syringe is maintained during the entire withdrawal of the needle.

The success of the biopsy is dependent upon insertion of the needle to the proper depth so that the tip actually enters the lesion. Since biplane image amplifiers are not often available, more indirect methods to determine depth may be necessary. Preliminary study and measurements of the posteroanterior and lateral chest films, with allowance made for magnification, permit a fairly accurate estimate of the depth of the lesion. After the needle is inserted, under fluoroscopic control, to the depth believed appropriate, the relationship of the needle tip to the mass can be checked by having the patient breathe normally. Coordinated synchronous movement of the lesion and needle tip can be seen. As a further check, one can rotate the patient into right and left 45° oblique positions and recheck the needle position in the new planes. Frequently the increased resistance of the lesion will be appreciated as the needle enters its surface. Once impaled, small lesions in the lung parenchyma can be moved by manipulation of the biopsy needle, thus confirming penetration of the needle to the proper depth.

Two or three smears are made from the material expressed from the needle and are prepared for cytologic examination. An air dried smear should be stained for acid-fast organisms if tuberculosis seems a reasonable possibility. Cultures are taken from the material in the syringe or needle and appropriate media for aerobic and anaerobic bacteria, mycobacteria and fungi are inoculated. The residual material in the needle or syringe is washed out by aspirating Bouin's fixative solution and ejecting it in a specimen bottle. Tiny tissue particles are spun down and paraffin sections made. The residue is then passed through a millipore filter and further sections are made.

Three expiration posteroanterior chest films are taken routinely, the first one immediately following withdrawal of the needle, the second at one hour and the third at 24 hours. Although pneumothorax may not be visible at once, it may appear after several hours. This possibility is discussed with the patient, who is permitted to go home after one hour if no complication is present.

TABLE 1.—*Results of Needle Aspiration Biopsy for Diagnosis of Pulmonary Lesions*

	No. of Biopsies	*No. of Patients*
Malignant change diagnosed	15*	14
Non-malignant (confirmed by surgical operation, culture or disappearance of lesion)	14	10
Non-malignant (substantiated by clinical course and radiographic stability)	3	2
Probable false negative (known primary, multiple lesions)	2	2
False negative (biopsy benign initially but lesion proved malignant on subsequent needle biopsy)	4	3
False positive	1	1
	39	32†

---

*Bilateral malignant change diagnosed in one patient.

†Three "false negative" patients are included also under "malignant change diagnosed" due to subsequent needle biopsy findings.

Results

To date biopsy has been carried out on 30 lesions in 29 patients (bilateral lesions in one patient). Four patients had two such procedures, and three had three. In none of these patients had a diagnosis been established before the biopsy, although most had had extensive diagnostic investigation. The principal problem in most patients was to determine the presence or absence of malignant disease. The results are shown in Table 1.

The false positive result shown in the table was the result of misinterpretation of postpulmonary infarct metaplasia as carcinoma. The rapidly diminishing size of the lesion, however, prevented radiation therapy or surgical intervention. Death from other causes was followed by autopsy study of the infarct.

Complications

Thirteen of 39 biopsies resulted in radiographically demonstrable pneumothorax. In most cases the patients were unaware of the pneumothorax and in all but one instance it resolved spontaneously within 48 hours. In the patient who was aware of the complication, the lung was 30 per cent collapsed, and closed intercostal drainage was

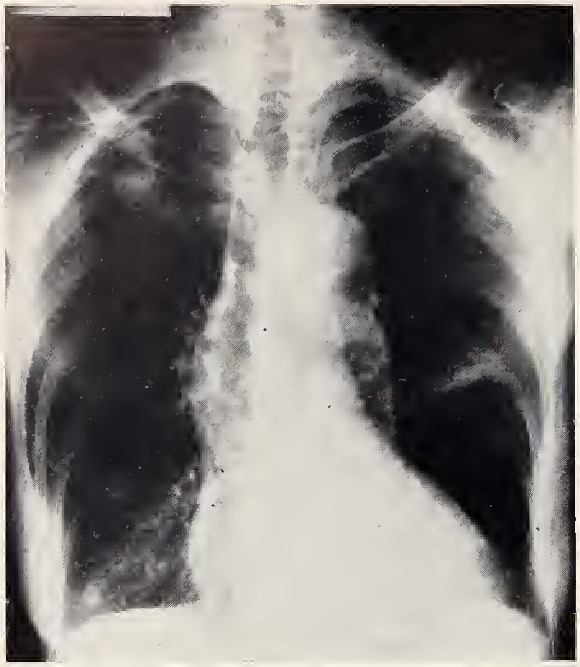


Figure 1.—Needle aspiration biopsy of right and left pulmonary lesions proved the presence of bilateral alveolar cell carcinoma and the futility of surgical operation.

maintained for 48 hours. Four patients had brief, minimal hemoptysis following biopsy, and one additional patient noted recurrent minimal hemoptysis over a period of several days. No other complication was encountered. No implants of malignant lesions along the needle tract have been observed, although it should be mentioned that the follow-up period was brief, varying from 15 months to two months.

Reports of Cases

The following case histories illustrate the usefulness of needle aspiration biopsy of the lung.

Case 1.—A 68-year-old woman with advanced multiple sclerosis was noted to have a linear "scar" in the left midlung field in 1962. An x-ray film of the chest in 1965 showed that the "scar" on the left was unchanged, but a new circumscribed lesion, 1.5 cm in diameter, was present in the right subapical area (Figure 1). Needle aspiration biopsy of the lesion in the right lung showed alveolar cell carcinoma. Despite the serious operative risk, surgical excision was considered, as solitary localized alveolar cell tumors are sometimes cured by local removal. Needle biopsy of the "scar" in the left lung, however, also revealed alveolar cell carcinoma, proving that the disease was bilateral. This information forestalled a useless thoracotomy. The patient died four months later.

Case 2.—A 44-year-old man was shown in 1965 to have an asymptomatic noncalcified nodule measuring 2.5×3 cm in the right lung (Figure 2). This nodule had been noted first three years previously, and it appeared to have increased slightly in size. Surgical removal in 1965 was postponed because thyrotoxicosis was present. Later the same year, needle aspiration biopsy revealed chronic inflammatory cells, and cultures of the aspirated material were positive for atypical mycobacteria (Runyon Group IV) after four weeks of growth. Six months later the lesion was smaller.

Case 3.—A 70-year-old man with aortic stenosis and coronary artery disease was found to have left hilar enlargement in November 1964. Bronchogenic carcinoma was suspected, but extensive diagnostic studies, including multiple sputum cytological studies, bronchoscopy, scalene node biopsy, bronchography and pulmonary angiography, were negative for malignant change. Exploratory thoracotomy was considered contraindicated because of the heart disease and the likelihood that the lesion would prove unresectable if malignant. In July 1965 (Figure 3) the lesion was larger, but cytological examinations of sputum were still negative. In November 1965, needle aspiration biopsy of the left hilum showed "oat cell" bronchogenic carcinoma and radiotherapy was begun.

Discussion

Lauby and associates[6] said that the development of other diagnostic techniques, such as cytologic examination of sputum and bronchial washings, angiograms and scalene node biopsy, has steadily diminished the need for needle biopsy. Sabour and coworkers[12] take a contrary view, which we share, that needle biopsy should be a commonplace procedure in the diagnosis of localized pulmonary disease and should frequently substitute for a long, conventional diagnostic work-up and diminish greatly the need for diagnostic thoracotomy. An exception to this is the occasional pulmonary lesion which clearly should be removed no matter what its identification. In such a case, there would seem to be little justification for any diagnostic investigation of the lesion, once the patient's condition is found satisfactory for surgical operation.

Diagnosis by culture or cytologic examination of sputum remains the least expensive and least hazardous method of precise diagnosis when positive information is obtained. Because of the nonspecificity of tomography and angiography, the relatively lower yield and the greater patient discomfort of scalene node biopsy and bronchoscopy, the increased risk of thoracotomy, and the greater expense of each of these methods, we believe that needle aspiration biopsy is often preferable. If the diagnosis of malignant change is established, bronchoscopy and scalene node biopsy may become necessary to evaluate operability.

At present, we consider a negative or nonspecific biopsy as unreliable and proceed with thoracotomy if advisable. There is, of course, no reason that a second or a third needle biopsy should not be performed if the diagnosis in inconclusive. Indeed, in the three patients in the present series with false negative biopsy, subsequent needle biopsy proved the presence of malignant tumor. Just as in cytologic studies of sputum, the yield is improved with additional specimens. Biopsy specimens can be taken from different parts of the lesion on the same day. When necessary, smears of the aspirated material can be stained and examined immediately to check the adequacy of the biopsy.

Needle biopsy is particularly advantageous in the diagnosis of metastatic pulmonary lesions, inoperable primary carcinoma and bronchogenic carcinoma in poor risk patients. The information also is useful in planning preoperative radiation therapy. A further advantage is that patients whose disease is established as benign or infectious are spared thoracotomy.

While our experience with needle biopsy has been limited to localized lesions, Manfredi and

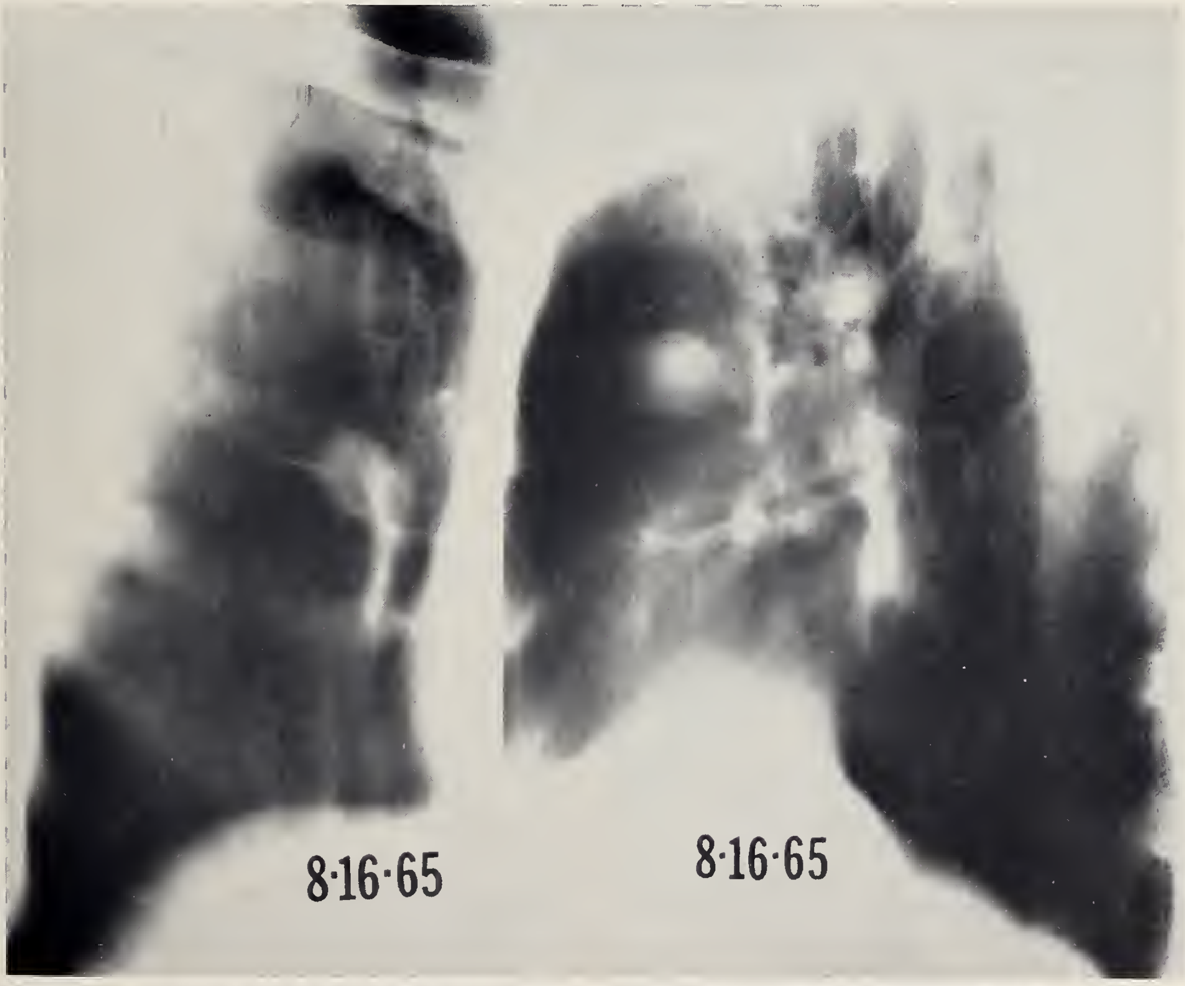


Figure 2.—*Left*, posteroanterior tomogram of right hilar nodule in 44-year-old man. Nodule proved on needle biopsy and culture to be due to atypical mycobacteria. The accessibility of deep seated small lesions to needle biopsy is illustrated. *Right*, lateral tomogram of lesion shown on left.

associates,[7] Smith,[14] and Krumholz and Weg[5] have recently reported on the usefulness of needle biopsy in diffuse pulmonary lesions. These investigators reported 66 per cent, 80 per cent and 86 per cent yields, respectively, using modified Franklin-Silverman or Vim Silverman needles. It would seem that larger coring-type needles would be more effective in diffuse lesions, whereas a smaller spinal type needle is suitable and probably less hazardous for aspiration biopsy of localized lesions, particularly those near the hilum.

If one discounts the occurrence of asymptomatic, self-limiting pneumothorax and hemoptysis, the complications reported by nearly all observers have been very infrequent. Occasionally, symptomatic or persistent pneumothorax appears which must be treated, and on very infrequent occasions moderate bleeding occurs. The frequency of pneumothorax in the present series was somewhat higher than in most, but we believe this to be the result of routinely taking immediate post-biopsy, one-hour and 24-hour expiration chest films. Rare reports of convulsions believed due to air embolism in patients after needle biopsy while erect have appeared.[6,15] None is mentioned in patients who were reclining. Deaths following needle aspiration biopsy have been reported.[6,15] Only one of the deaths, however, seems clearly the result of the procedure, and in this case the tension pneumothorax which followed was not recognized. Death was probably preventable.

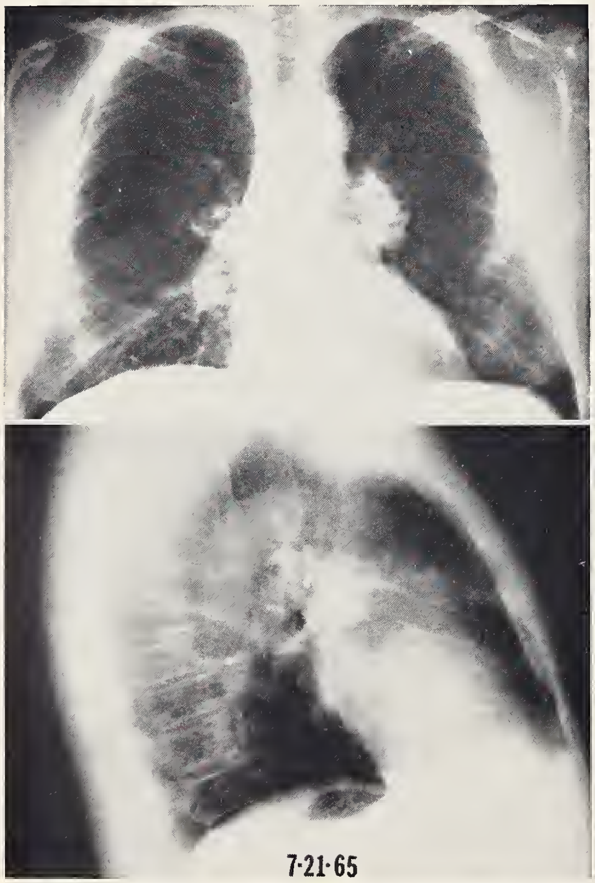


Figure 3.—*Upper*, posteroanterior view of left hilar tumor in 70-year-old man. After all other diagnostic procedures had failed, needle aspiration biopsy proved this to be an oat cell bronchogenic carcinoma. *Lower*, lateral view of same case.

If pus is obtained on aspiration, Lauby and coworkers[6] immediately inject antibiotics into the cavity and along the needle tract upon withdrawal. Prompt recognition of acid-fast organisms on smears allows treatment to start immediately and diminishes the likelihood of dissemination along the needle tract. We have not had occasion to use such therapy.

Thirty-three of 39 biopsies reported herein were performed on outpatients. The absence of pain during the procedure is impressive. No patient refused re-biopsy because of discomfort during an earlier biopsy.

Contraindications

Absolute contraindications to needle biopsy are rare. Needle biopsy is inadvisable for uncooperative patients (this could be overcome by anesthesia), or in the presence of hemorrhagic diathesis or severe pulmonary hypertension (which increases the risk of bleeding), or for patients who refuse definitive therapy no matter what the biopsy result.

REFERENCES

1. Aronovitch, M., Chartier, J., Kahana, L. M., Meakins, J. F., and Groszman, M.: Needle biopsy as an aid to the precise diagnosis of intrathoracic disease, Canad. Med. Ass. J., 88:120, January 1963.

2. Craver, L. F., and Binkley, J. S.: Aspiration biopsy of tumors of lung, J. Thoracic Surg., 8:436, 1939.

3. Franseen, C. C.: Aspiration biopsy with a description of a new type of needle, New Engl. J. Med., 224: 1054, 1941.

4. Gledhill, E. Y., Spriggs, J. B., and Binford, C. H.: Needle aspiration in the diagnosis of lung carcinoma; report of experience with 75 aspirations, Amer. J. Clin. Path., 19:235, 1949.

5. Krumholz, R. A., and Weg, J. G.: Percutaneous needle biopsy of the lung, J.A.M.A., 195:38, January 1966.

6. Lauby, Vincent W., Burnett, W. Emory, Rosemond, George P., and Tyson, R. Robert: Value and risk of biopsy of pulmonary lesions by needle aspiration, J. Thor. Cardiov. Surg., 49:159, January 1965.

7. Manfredi, Felice, Rosenbaum, David, and Behnke, Roy H.: Percutaneous needle biopsy of the lung in diffuse pulmonary diseases, Ann. Intern. Med., 58:773, 1963.

8. Martin, H. E., and Ellis, E. B.: Aspiration biopsy, Surg. Gynec. Obstet., 59:578, 1934.

9. Martin, H. E., and Stewart, F. W.: Advantages and limitations of aspiration biopsy, Amer. J. Roentgenol., 35:245, 1936.

10. Ménétrier, P.: Cancer primitif du poumon, Bull. Soc. Anat. de Paris, 11:643, 1886.

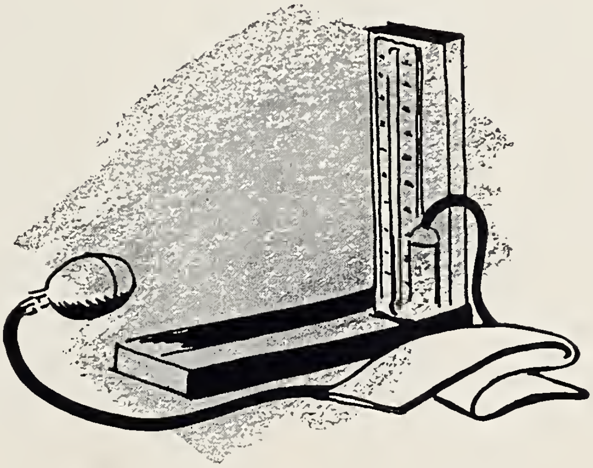
11. Nordenstrom, Bjorn: Thoracic Clinic Karolinska Sjukhuset, Stockholm, Sweden (Personal communication).

12. Sabour, M. S., Osman, L. M., Le Golvan, P. C., and Ishak, K. G.: Needle biopsy of the lung, Lancet, 2:182, 1960.

13. Schiessle, W., and Könn, G.: La ponction-biopsie transpariétale du poumon et du médiastin. Technique et résultats, J. Franc. Med. Chir. Thor., 19:593, 1965.

14. Smith, W. G.: Needle biopsy of the lung. With special reference to diffuse lung disease and the use of a new needle, Thorax, 19:68, January 1964.

15. Woolf, C. R.: Applications of aspiration lung biopsy with a review of the literature, Dis. Chest, 25: 286, March 1954.



The Use of Tumor Registry Data

SIDNEY J. CUTLER, SC.D., *Bethesda, Maryland*

■ *The End Results Group, sponsored by the National Cancer Institute, coordinates a national cooperative program for evaluating the results of cancer therapy. Three central tumor registries (in California, Connecticut and Massachusetts) plus nine individual hospital registries (in university hospitals) are participating in the program.*

During the period 1940-59, there was an encouraging increase in five-year survival rates among patients with carcinomas and sarcomas (solid tumors). Preliminary data for 1955-59 suggest that the upward trend is leveling off. A smaller absolute increase in five-year survival rates occurred among patients with lymphomas and leukemia. These increases in patient survival rates coincided with more frequent use of surgical operation for solid tumors and more frequent use of chemotherapy for lymphomas and leukemia.

THERAPEUTIC POLICY must ultimately be evaluated from the experience of the general run of patients with a specified form of cancer. Recognition of this principle has led to the organization of cancer registration and patient follow-up programs in many hospitals. Ideally, all hospitals and physicians in a community, in a region or in a country supply information on all patients in a defined population. This permits the calculation of incidence rates, which makes possible comparison of trends in cancer incidence with trends in patient survival. However, even though a registry system may not encompass every case diagnosed in a defined population, judicious use of data pertaining to a broad spectrum of patients can provide useful information concerning the results of cancer therapy and factors influencing prognosis.

The California Tumor Registry, which is a central registry collecting information on individual cancer patients from 53 hospitals in various parts of the State, is an outstanding example of a well organized, excellently run registry system. The monograph on cancer registration and survival in California, published by the State Department of Public Health in 1963,[5] serves as a standard of excellence in the imaginative and judicious use of central registry data.

The End Results Group, sponsored by the National Cancer Institute, U.S. Public Health Service, is fortunate to have the California Tumor Registry as a member. The End Results Group coordinates a national cooperative program for evaluating the end results of cancer therapy. Three central tumor registries (in California, Connecticut and Massachusetts) plus nine individual hospital registries (in university hospitals) are participating in the program. The extent to which the institutions participating in this cooperative program are representative of all hospitals and clinics in the United States is not known. They do, however, represent a very broad experience and provide a basis for examining trends in cancer therapy and patient survival.

From the Biometry Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland.

Presented as part of an Interagency Conference on Cancer Diagnosis, Treatment and Control in California at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: Biometry Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20014.

The participating registries submit a punched card annually for each cancer patient seen in their hospitals and clinics. The punched cards contain information on the characteristics of the patient (sex, race and age), the nature of the disease (primary site, histological type and stage at diagnosis), date of diagnosis and treatment, date of last contact, follow-up status and survival time. The confidentiality of information on individual patients is completely protected, since patients are identified by case number only. The information available on punched cards may be used to identify cases for special studies calling for detailed review of hospital records.

I would like to review briefly with you the major trends regarding patient survival obtained through examination of data pertaining to 325,000 cancer patients in whom the disease was diagnosed during the 20-year period 1940-1959. I will then discuss an interesting observation concerning cancers of the colon and rectum, derived from examination of routinely collected tumor registry data. In addition, I will present examples of special studies carried out within the framework of tumor registry programs, one on cancer of the lung, designed to elucidate findings from routinely collected data, and one on prognostic factors in cancer of the female breast.

Trends in Patient Survival

The trend in patient survival is described in terms of relative survival rates. The survival rate observed in a group of patients reflects mortality not only from the disease under study but also deaths due to all other causes. The risk of dying from causes other than the specific cancer under study varies with the sex and age characteristics of the patient group and with calendar time. The relative survival rate adjusts for "normal" mortality and thus makes possible comparisons of the survival experience of groups of patients that differ with respect to sex, age and calendar period of observation. It is defined as the ratio of the observed survival rate to the expected rate for a group of people in the general population similar to the patient group with respect to sex, age and calendar period of observation. The relative survival rate is the probability of escaping the mortality risk due to the specific cancer under study.

TABLE 1.—*Trends in Cancer Survival Rates (5-year)—1940-1959*

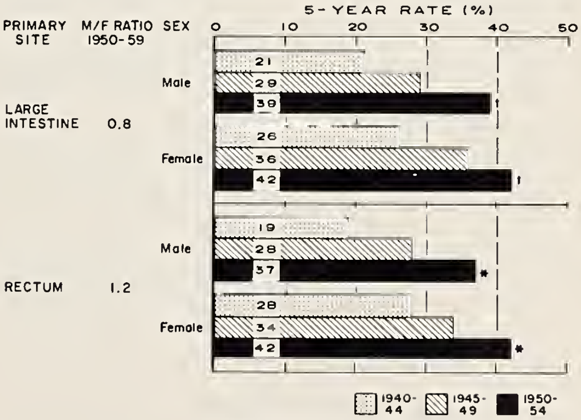
	5-Year Survival Rate (Per Cent)		
	All Malignant Disease	*Solid Tumors*	*Leukemia and Lymphomas*
1940-44	35	36	16
1945-49	42	43	17
1950-54	45	47	19
1955-59*	45	47	20

*Preliminary.
Source: End Results Report No. 2.[5]

The trend in patient survival rates for all patients with cancer is summarized in Table 1. There was an encouraging increase in five-year survival rates among patients with carcinomas and sarcomas (solid tumors), from 36 per cent for patients in whom the diagnosis was made in 1940-44 to 47 per cent for patients with the disease diagnosed in 1950-54. Preliminary data for 1955-59 suggest that the upward trend is leveling off. A smaller absolute increase in five-year survival rates occurred among patients with lymphomas and leukemia from 16 per cent for patients with diagnoses in 1940-44 to 20 per cent for those in whom the diagnosis was made in 1955-59. These increases in patient survival rates coincided with more frequent use of surgical operations in treating patients with carcinomas and sarcomas, and more frequent use of chemotherapy in treating patients with lymphomas and leukemia. Survival data for several of the more frequent forms of cancer are shown in Charts 1 through 5.

Colon and Rectum

Five-year survival rates for cancers of the colon and rectum increased from about 25 per cent in patients diagnosed 1940-44 to about 40 per cent for patients diagnosed in 1950-54 (Chart 1). The



†Preliminary rates for 1955-59 indicate that the increase in survival appears to be continuing.

*Preliminary rates for 1955-59 indicate that survival appears to be leveling off.

Chart 1.—Five-year relative survival rates for patients with cancers of the large intestine and rectum. Source: End Results in Cancer, Report No. 2.[5]

trend was similar for both sites of origin and for both men and women. Preliminary data for 1955-59 suggest that the upward trend is continuing for cancers of the colon but appears to be leveling off for cancers of the rectum.

The upward trend in survival for both sites coincided with a sharp increase in the proportion of patients treated surgically, from about 45 per cent in 1940-44 to about 75 per cent in 1955-59. The proportion of cases classified as localized remained unchanged at around 40 per cent. This is probably a reporting artifact, reflecting more accurate classification of extent of disease resulting from more frequent use of both therapeutic and exploratory operation.

Stomach and Lung

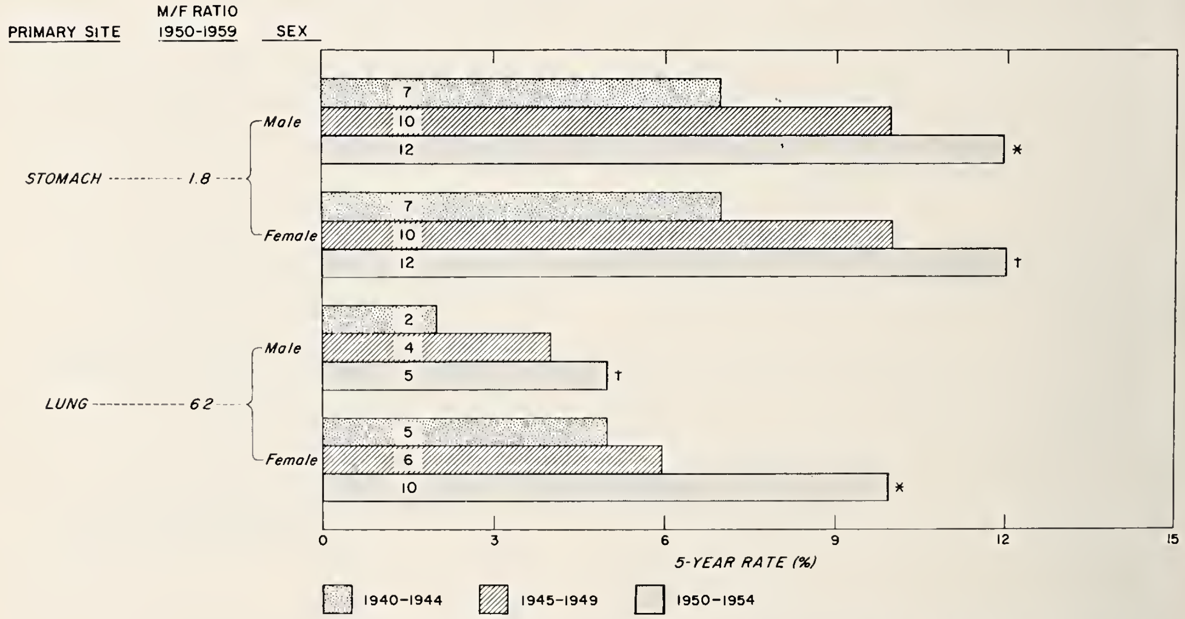
Although prognosis for patients with cancers of the stomach and of the lung remains poor, there has been some improvement in survival (Chart 2). These two forms of cancer were grouped on the same chart to illustrate an interesting sex difference. The survival rates for male and female patients with stomach cancer were identical. In contrast the survival rates for women with lung cancer were higher than for men. This contrast shows up more sharply when restricted to patients with localized disease. In recent years, somewhat less than 20 per cent of cancers originating in the stomach and the lungs were classified as localized. Among patients with localized stomach cancer the survival rates were similar for men and women—39 per cent at five years for patients with disease diagnosed 1950-54. In contrast, the five-year survival rate for patients with localized lung cancer was 17 per cent for men, 32 per cent for women. The factors involved in the sex differential for lung cancer are discussed in a separate section.

Female Reproductive System

Survival rates for women with cancers of the breast, uterine cervix, uterine corpus and ovary have increased (Chart 3). The increases for cancer of the uterine cervix and corpus were particularly impressive. Although the reported increase in the proportion of localized cases of uterine cancer has been small, it is likely that there has been a significant increase in the proportion of "early localized" cases with more frequent use of vaginal cytology for case finding.

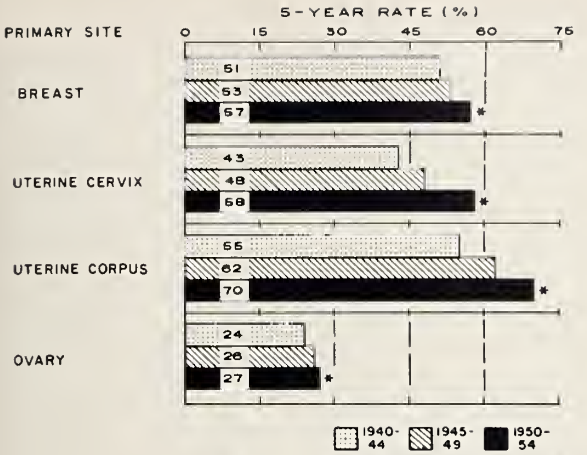
Acute Leukemia

There was a dramatic increase in survival rates for patients with acute leukemia (Chart 4), but it must be noted that the rates shown are one-year rates. Few patients with acute leukemia were alive three years after diagnosis. The observed increase in survival coincided with a major change in thera-



† PRELIMINARY RATES FOR 1955-59 INDICATE THAT THE INCREASE IN SURVIVAL APPEARS TO BE CONTINUING.
* PRELIMINARY RATES FOR 1955-59 INDICATE THAT SURVIVAL APPEARS TO BE LEVELING OFF.

Chart 2.—Five-year relative survival rates for patients with cancers of the stomach and lung. Source: End Results in Cancer, Report No. 2.[5]

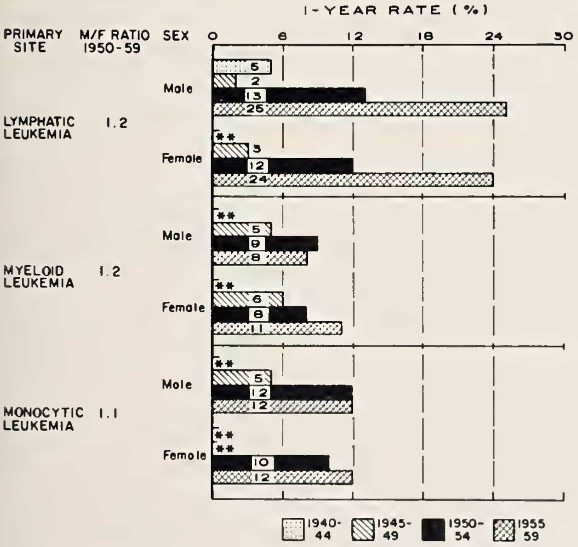


*Preliminary rates for 1955-59 indicate that survival appears to be leveling off.

Chart 3.—Five-year relative survival rates for women with cancers of the breast, uterine cervix, uterine corpus and ovary. Source: End Results in Cancer, Report No. 2.[5]

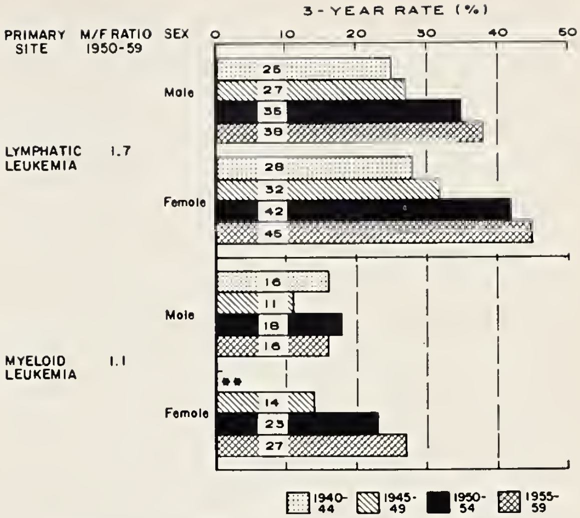
peutic approach. Chemotherapy was introduced in the late 1940's; by the late 1950's two-thirds of the patients with acute leukemia were receiving chemotherapy. Although chemotherapy is now the treatment of choice for all forms of acute leukemia, there has been a much more dramatic change in survival among patients with lymphatic leukemia.

Survival rates for children with acute leukemia have increased substantially more than for adults. Recent data indicate a decided gradient in survival of patients with acute leukemia with respect to age.



** The number of cases are too small to yield reliable rates.

Chart 4.—One-year relative survival rates for patients with acute leukemia. Source: End Results in Cancer, Report No. 2.[5]



** The number of cases too small to yield reliable rates

Chart 5.—Three-year relative survival rates for patients with chronic leukemia. Source: End Results in Cancer, Report No. 2.[5]

For example, among male patients with acute leukemia diagnosed 1955-59, the median of months survived, by age at diagnosis was as follows:

Age at Diagnosis	*Median of Months Survived*
0-9	8.1
10-19	5.4
20-44	3.3
45-64	2.2
65 & over	1.6

Chronic Leukemia

Among patients with chronic leukemia there was an encouraging increase in survival among patients with the lymphatic type (three-year survival rates are shown). Among patients with chronic myeloid leukemia, survival rates increased among women but not among men. The reason for the sex difference is not known. (See Chart 5.)

The more favorable survival rates for patients with lymphatic leukemia is not associated with age; the age distributions for the chronic lymphatic and myeloid leukemia are very similar. Although there was an increase in the proportion of chronic leukemia patients receiving chemotherapy it is noteworthy that even in the most recent period, 1955-59, a very substantial proportion of patients received no specific anti-leukemic therapy—48 per cent for lymphatic leukemia, 33 per cent for myeloid leukemia. Thus, the more favorable prognosis for chronic lymphatic leukemia appears to be due, at least in part, to the nature of the disease.

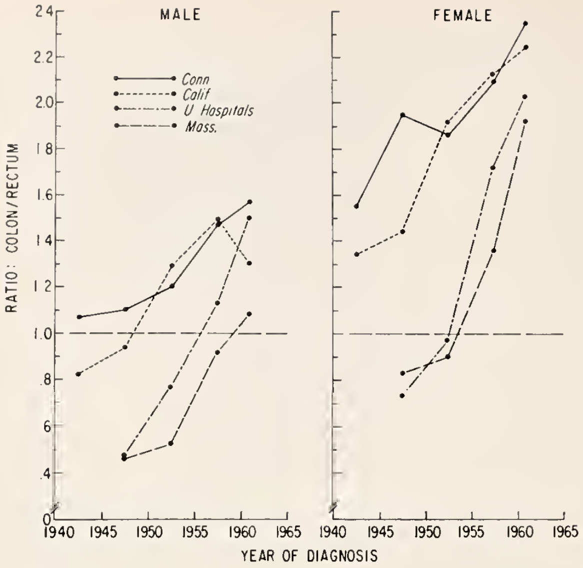


Chart 6.—Ratio of colon to rectum cancers reported by the End Results Group, 1940-1962. Source: Axtell and Chiazze.[1]

Incidence of Cancers of the Colon and Rectum

In the course of summarizing the trend in the survival experience of patients with cancers of the colon and rectum, it was noted that a change appeared to be taking place in the relative frequency of cancers at these two sites. As shown in Chart 6, there has been a continuing increase from the early 1940's to the early 1960's in the ratio of cancers of the colon to cancers of the rectum. This occurred among both male and female patients, in each of the three centralized registry systems, and in the individual (university) hospital registries.

Since the totality of cases reported by the End Results Group cannot be related to a population base, the sharp upward trend in the ratio of cancer of the colon to cancer of the rectum might be a reflection of selective patient referral policy. However, available data from Connecticut and upper New York State, that have cancer reporting sys-

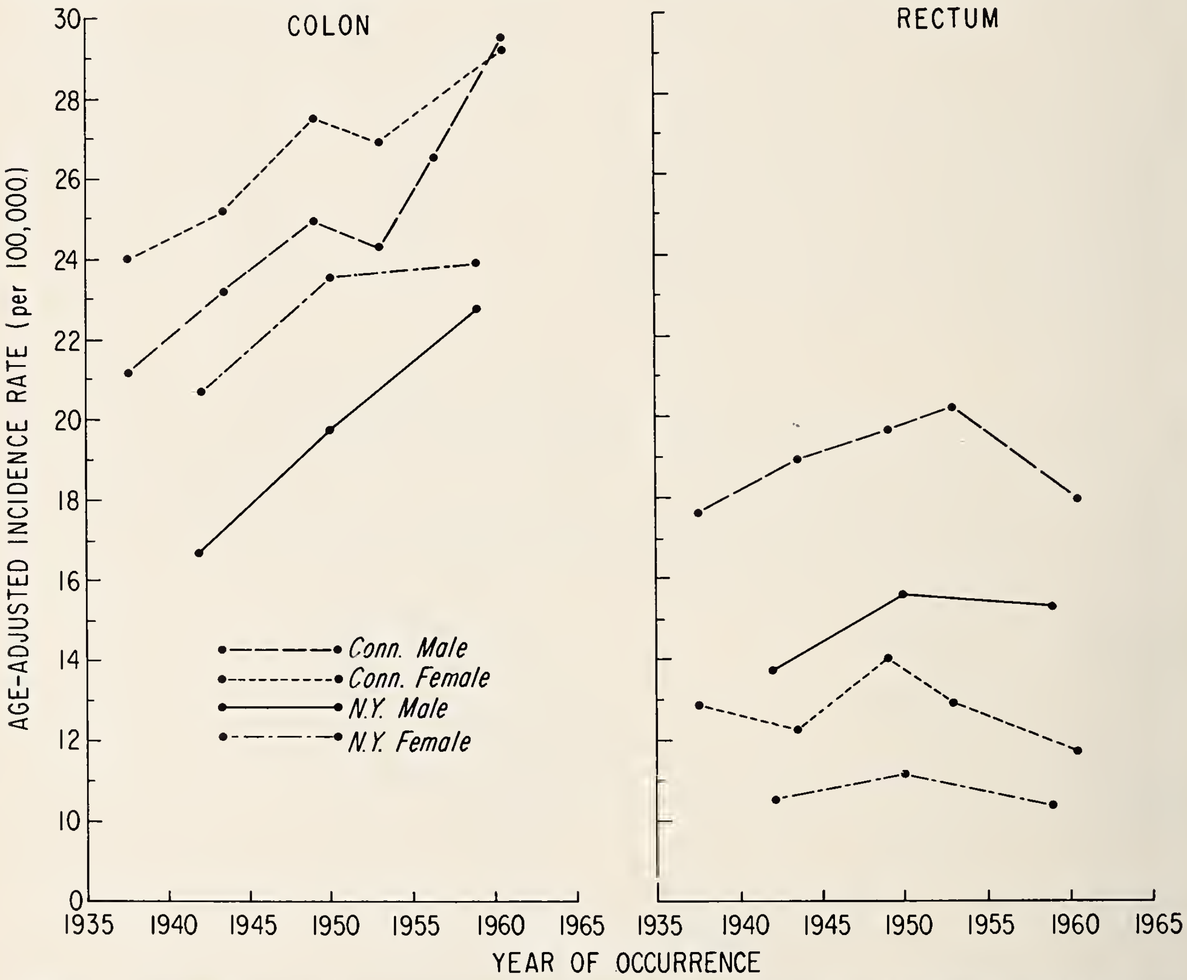


Chart 7.—Incidence rates* for cancers of colon and rectum in Connecticut and New York State. Source: Axtell and Chiazze.[1]

*Age adjusted on United States population for 1950.

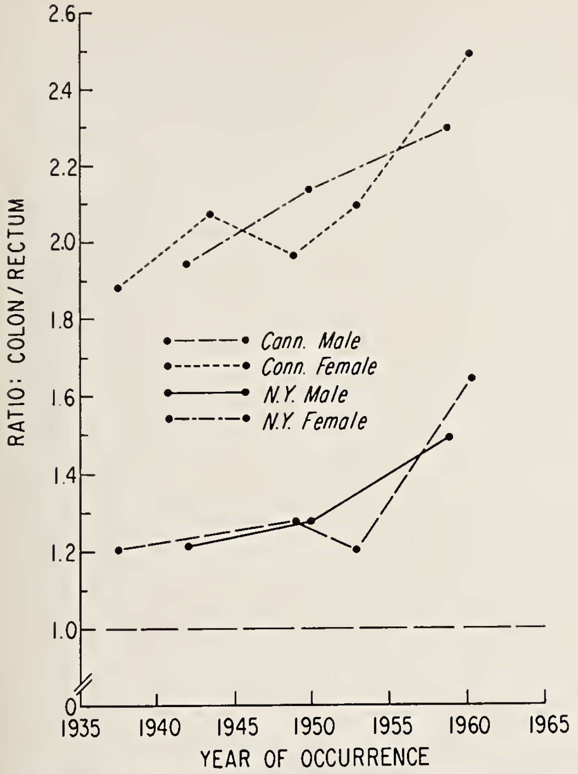


Chart 8.—Ratio* of incidence rates for cancers of the colon and rectum: Connecticut and New York State. Source: Axtell and Chiazze.[1]

tems for defined geographic areas, indicate that the trend in the colon : rectum ratio reflects different trends in the rate of incidence. As shown in Chart 7, the rate of incidence of cancers of the colon has been increasing, whereas the incidence of cancer of the rectum has been relatively stable. The trend in the ratio of incidence rates for cancers of the colon and rectum (Chart 8) is consistent with the trend based on absolute numbers (Chart 6).

The observed trend might have occurred because of changing criteria in the assignment of primary site for tumors originating in the vicinity of the rectosigmoid junction. If such a factor had been operating, we would expect an increase in the proportion of cancers of the colon assigned to the sigmoid colon. No such change took place. Cancers of the sigmoid colon accounted for about 43 per cent of cancers of the colon in men and about 38 per cent in women throughout the period of study. It thus appears that the reported increase in the incidence of cancer of the colon is real. The increased incidence of cancer of the colon coincides with a decrease in the reported incidence of cancer of the stomach. It is reasonable to hypothesize that some of the factors associated with a decrease in incidence of stomach cancer are also associated with the increased incidence of cancer of the colon. Such a hypothesis may provide a reasonable basis for etiologic research on cancers of the digestive tract.

Lung Cancer in Men and Women

A striking difference between survival rates for men and women with cancers of the lung was first reported by Mersheimer and Ederer in 1960.[4] In examining pooled data from 99 hospitals participating in the End Results Evaluation Program, these investigators found that among surgically treated patients with localized disease, the survival rate was substantially higher in women. A review of records was undertaken several years later to confirm these findings.[2] A total of 1,015 patients' records were reviewed, 511 for male and 504 for female patients.

Treatment (initial course) was found to be similar for men and women: (a) Two hundred and seven men and 209 women were treated surgically, and of these 32 men and 37 women also received radiation or chemotherapy; (b) Two hundred and ten men and 205 women were treated by radiation, with or without chemotherapy; (c) Ninety-four men and 90 women received only chemotherapy. Survival curves for surgically and non-surgically treated patients are shown in Chart 9. Among surgically treated patients, the curve for women is consistently higher by several percentage points. In contrast, the two curves for patients treated by other

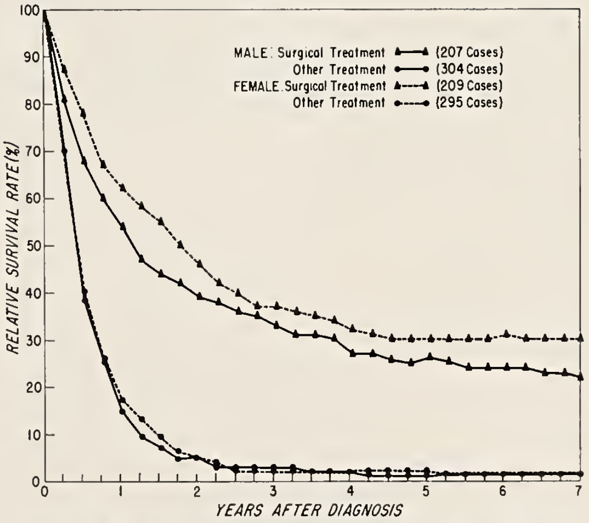


Chart 9.—Relative survival rates for male and female patients with cancer of the lung, by type of treatment. Source: Connelly Cutler and Baylis.[2]

---

*Based on age-adjusted incidence rates.

means are almost identical and at a much lower level.

Among surgically treated patients, a higher proportion of women than men had localized tumors —43 and 32 per cent, respectively. This partially explains the higher survival rate among surgically treated women but, as is shown in Chart 10, the survival curve for women with localized tumors is substantially higher than the corresponding curve for men.

A number of factors were examined for possible association with the observed survival advantage in favor of women, among patients with localized tumors:

Age—The distribution of cases with respect to age at diagnosis was similar for men and women.

Histological Type—Adenocarcinomas and alveolar cell tumors occurred much more frequently in women than in men; they accounted for 58 per cent of localized cases in women, compared with only 19 per cent in men. However, among patients with localized adenocarcinomas and alveolar cell tumors, the survival rate was much higher in women than in men—55 per cent against 16 per cent at five years (relative rates). Among patients with localized epidermoid or undifferentiated tumors, the survival rates were closer together—41 per cent for men, 48 per cent for women.

Extent of Tumor—Among patients with localized disease, 79 per cent of women had tumors limited to one lobe compared with 58 per cent of men. This was in part due to the more frequent occurrence of adenocarcinomas and alveolar cell tumors in women. Three out of four adenocarcinomas and alveolar cell tumors in both men and women were limited to one lobe. However, even among patients with epidermoid tumors, women more frequently had lesions limited to one lobe—83 per cent against 56 per cent in men.

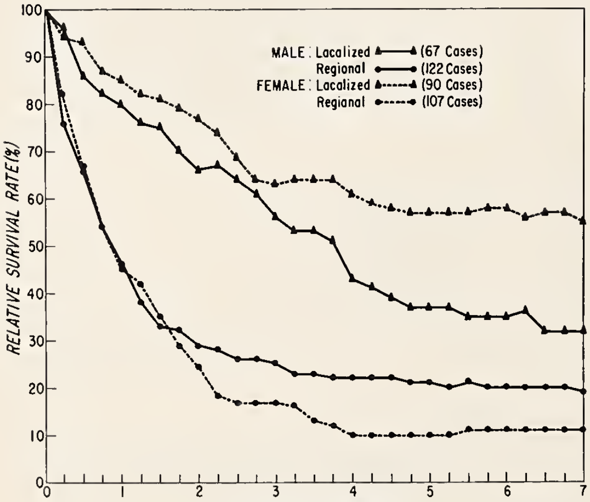


Chart 10.—Relative survival rates for surgically treated lung cancer patients, by sex and stage of disease. Source: Connelly, Cutler and Baylis.[2]

The more frequent occurrence of tumors of limited extent in women only partially explains the survival advantage in favor of women, because women also had more favorable survival among patients with tumors limited to one lobe—57 per cent against 45 per cent* for epidermoid tumors, and 65 per cent against 17 per cent for adenocarcinomas. Thus, part of the advantage in favor of women remains unexplained. Further investigation of factors associated with the variation in prognosis between men and women with lung cancer may not only help explain the sex difference more adequately, but may also provide additional biologic insight into cancer of the lung.

Prognostic Factors in Cancer of the Female Breast

Data collected by the End Results Group in a study designed to compare the staging schemes for cancer of the breast recommended by the Inter-

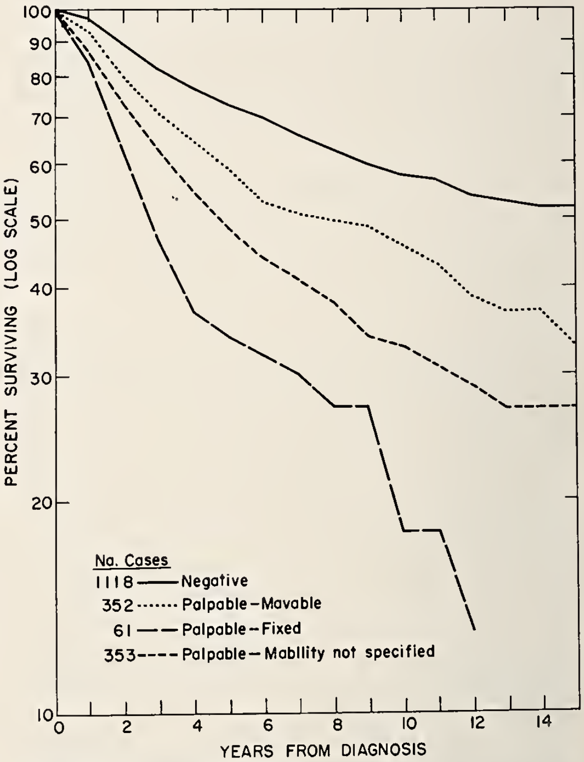


Chart 11.—Survival of breast cancer patients, by clinical evaluation of axillary node status.

---

*Five-year relative survival rates.

national Union Against Cancer and by the American Joint Committee on Cancer Staging and End Results Reporting provides an opportunity to explore the inter-relationship among a number of factors associated with variation in patient survival. Comparison of the two schemes, both of them based on the TNM system of classification (Tumors, Nodes, Metastasis), has been reported on by Zippin,[6] who was kind enough to let us have the punched cards from this study for further exploration.

Since both the International Union (U.I.C.C.) and the American Joint Committee set out to develop a clinical staging system, we considered a number of characteristics evaluated before initiation of therapy. Chart 11 shows the relationship of patient survival to axillary node status. Patients with no palpable nodes clearly had the most favorable experience, those with palpable nodes fixed to one another or to other structures the least favorable. There were 353 cases in which the records showed palpable nodes but gave no indication as to whether or not these nodes were moveable. The survival curve for these patients falls between the curves for patients with moveable nodes and patients with fixed nodes. This suggests that the group with incomplete information contains a sizable number of patients with fixed nodes.

Chart 12 shows the relationship of clinical evaluation of tumor size (the greater diameter) to patient survival.* It is noteworthy that the curves for the first four groups (up to 1 cm, 1.1-2.0 cm, 2.1-3.0 cm and 3.1-4.0 cm) are clearly separated. In contrast the curve for patients with tumors between 4 and 5 centimeters is virtually superimposed on the curve for patients with tumors between 5 and 7.5 centimeters. These findings suggest that the recommendations of both the International and the American Committees warrant reconsideration. The International scheme groups under Stage I all tumors of 5 centimeters or less (in the absence of other manifestations). The American scheme uses 2 centimeters as a point of demarcation between Stages Ia and Ib and between Stages IIa and IIb. The data at hand suggest that clinicians should be encouraged to record tumor size in centimeter units rather than in broad, arbitrary categories. Experience with such detailed data may in time lead to agreement on an acceptable set of summary categories.

TABLE 2.—*Survival Rates for Breast Cancer Patients by Clinical Evaluation of Axillary Node Status and Tumor Size*

Axillary Node Status and Tumor Size	*Per Cent Survived** *5 Years*	*Per Cent Survived** *10 Years*	*Average Annual Death Rate*
Negative Nodes			
<2.0 cm	86	77	.027
2.1 – 4.0 cm	75	59	.053
4.1 – 7.5-cm	55	32	.107
>7.5 cm	46	42	.114
Palpable Nodes, Moveable			
<2.0 cm	79	69	.042
2.1 – 4.0 cm	63	48	.075
4.1 – 7.5 cm	45	33	.126
>7.5 cm	32	26	.198
Palpable Nodes, Fixation Not Specified			
<2.0 cm	61	43	.082
2.1 – 4.0 cm	55	33	.111
4.1 – 7.5 cm	25	18	.214

*Relative survival rates; adjusted for normal mortality expectations.

The inter-relationship of axillary node status and tumor size to patient survival is shown in Table 2. The data indicate that survival varies inversely with tumor size within each node status category: "Negative," that is, not palpable; palpable, moveable; palpable, fixation not specified.* Within each

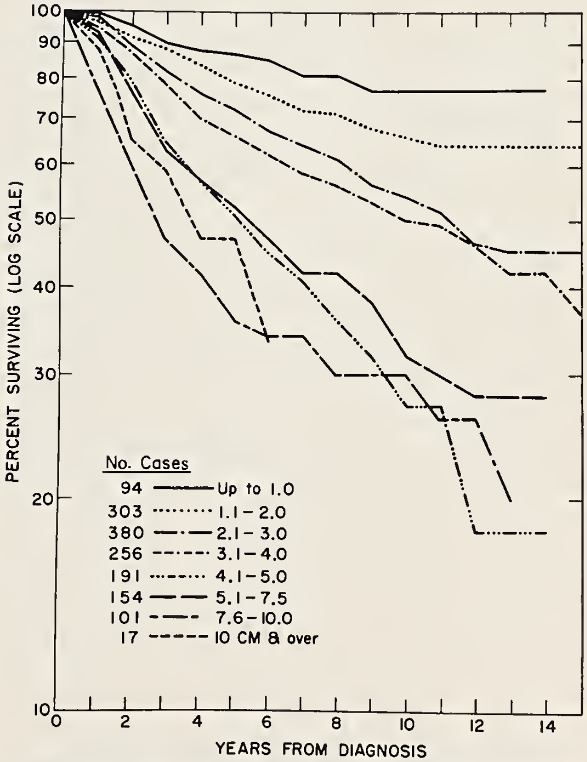


Chart 12.—Survival of breast cancer patients, by tumor size.

---

*One hundred and eighty-one cases in which tumor size was recorded as fruit and vegetable sizes were omitted from the data in Chart 12. These cases were included in Tables 2 and 3, in which tumor size was classified in less detail.

*The 61 cases in which nodes were specified as fixed were excluded from this analysis.

TABLE 3.—*Survival Rates for Breast Cancer Patients by Clinical Evaluation of Axillary Node Status, Skin Involvement and Tumor Size*

Axillary Node Status, Skin Involvement, Tumor Size	*Per Cent Survived**		*Average Annual Death Rate*
	5 Years	*10 Years*	
Negative Nodes			
No Skin Involvement			
<2.0 cm	87	89	.025
2.1 – 4.0 cm	77	64	.044
>4.0 cm	60	38	.089
Skin Involved			
<2.0 cm	87	70	.033
2.1 – 4.0 cm	71	51	.064
>4.0 cm	48	29	.125
Palpable Nodes			
No Skin Involvement			
<2.0 cm	76	60	.047
2.1 – 4.0 cm	63	43	.081
>4.0 cm	44	32	.134
Skin Involved			
<2.0 cm	54	40	.109
2.1 – 4.0 cm	54	39	.103
>4.0 cm	39	23	.157

*Relative survival rates, adjusted for normal mortality expectations.

tumor size category, survival is most favorable for patients with negative nodes. Similarly, among patients with palpable nodes, within each size category, survival is more favorable for patients with nodes that were specified as moveable.

The cases are further cross-classified with respect to skin involvement in Table 3. Skin involvement is associated with lower survival rates for each tumor size category among both patients with negative nodes and patients with palpable nodes.

Other clinical factors found to influence prognosis in cancer of the breast include ulceration of the skin, fixation to underlying structures, edema of the arm and involvement of supraclavicular and contralateral nodes. Information obtained from microscopic examination of the surgical specimen provides additional, important information on factors influencing prognosis in breast cancer.[3] Discussion of statistical methods for systematically assessing the inter-relationships among the factors known to influence prognosis in breast cancer is beyond the scope of this discussion. However, before closing I want to show an interesting relationship between clinical and microscopic evaluation of axillary node involvement. Chart 13 shows how the clinician's and the pathologist's evaluations of axillary node involvement relate to each other and to patient survival. Of the 1,118 patients with no palpable nodes, 435 or 39 per cent were found by the pathologist to have node metastasis. Of the 766 cases with palpable nodes, 233 or 30 per cent were classified by the pathologist as having negative nodes. Should we assume that only the pathologist's evaluation is of prognostic significance? The data at hand say no. Among patients with nodes "positive" histopathologically, survival was considerably lower for those with clinically palpable nodes than for those without palpable nodes. Although the difference was much smaller among patients with histopathologically negative nodes, the displacement was in the same direction.

Discussion

The effect of therapeutic policy and medical skill is reflected in the survival experience of the general run of patients with a specified form of cancer. This is why we are interested in cancer registration and patient follow-up programs, which make possible comparison of results obtained in different periods of time, in different geographic areas and in different institutions. However, it does not follow that differences in survival between patient populations are necessarily or even usually due to differences in therapeutic policy or medical skill. The data presented in the preceding sections suggest that certain clinical and histological characteristics probably influence patient survival more

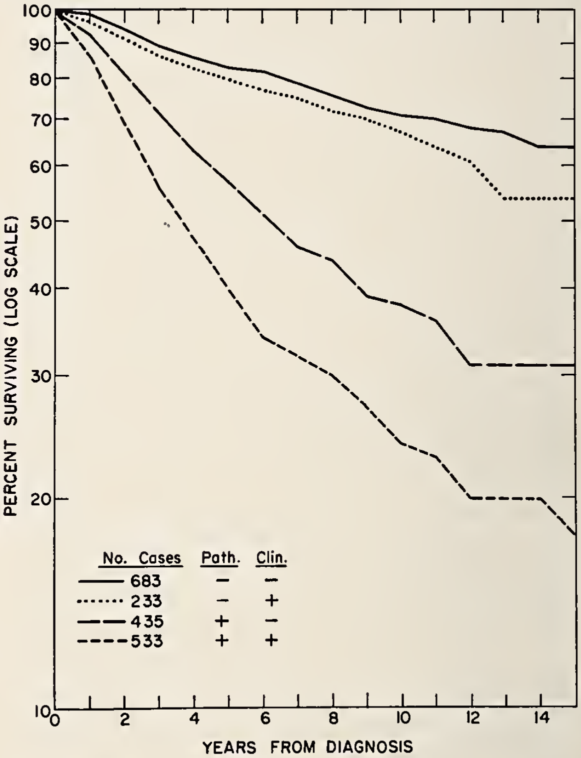


Chart 13.—Survival of breast cancer patients, according to cross-classification of clinical and histopathologic evaluation of axillary node status.

strongly than do variations in specific therapeutic policies and techniques. This has important implications for the planning of clinical trials and for the interpretation of clinical trial results.

The usual intent of a clinical trial is to compare results obtained in patient groups that are similar in all relevant characteristics except for the alternative treatment to be compared. By relevant characteristics we mean characteristics that are likely to influence the course of the disease or response to therapy. In clinical trials with cancer patients we do not hesitate to ignore a wide variety of characteristics such as eye and hair color, height, and weight. We ignore such characteristics for one of two reasons: (1) We see no logical relationship and see no need to verify the validity of our preconceptions, or (2) we have evidence from experience with the general run of patients that such factors are not associated with variation in end results. Conversely, we build certain characteristics into our experimental designs on the basis of unverified preconceptions as well as on the basis of objective experience.

I suggest that the evaluation of the relative importance of various patient and disease characteristics warrants more study. If an experimental design calls for stratification, patients should be stratified with respect to the most important prognostic factors. Furthermore, even though we depend on randomization procedures to take care of uncontrolled variation within strata, we must examine characteristics of the patient groups obtained through random selection to determine whether they are in fact comparable. We must select for inspection characteristics that are in fact of prognostic importance. Thus, analysis of the experience of the general run of patients is essential to provide guides for planning clinical trials and for interpreting their results.

Since clinical trial design aims at the selection of comparable groups of patients, it frequently leads to the selection of groups that fall within a restricted range of characteristics. For example, a trial pertaining to breast cancer may be restricted to patients below a certain age, with no evidence of lymph node fixation, skin involvement or extension to underlying structures. Furthermore, the qualifying characteristics specified in the protocol may be correlated with other factors of prognostic significance. For example, the restrictions specified above may result in the selection of patients with tumors of relatively low malignancy.

This kind of selection restricts the nature of the generalizations that may be made on the basis of the trial results. Consequently, no matter how dramatic the results of a clinical trial may be, they must be regarded as no more than tentative. The applicability of the study conclusions to different patient populations must be evaluated through actual experience. A highly technical therapeutic procedure that proved to be beneficial in the specialized institutions that usually participate in clinical trials may turn out to be of lesser or of no value when carried out by the average practitioner, or when used in the treatment of patients with characteristics that are different from those in the carefully planned clinical trial.

REFERENCES

1. Axtell, L. M., and Chiazze, L.: A note on the changing relative frequency of cancers of the colon and rectum in the United States, Cancer, publication pending.

2. Connelly, R. R., Cutler, S. J., and Baylis, P.: End results in cancer of the lung: Comparison of male and female patients, J. Natl. Cancer Inst., 36:277-287, 1966.

3. Cutler, S. J., Black, M. M., and Goldenberg, I. S.: Prognostic factors in cancer of the female breast, Cancer, 16:1589-1597, 1963.

4. Mersheimer, W. L., and Ederer, F.: End-results evaluation of cancer of the lung and the bronchus, Proceedings, Fourth National Cancer Conference, Minneapolis, September 1960, pp. 319-322, J. B. Lippincott Company.

5. Public Health Service Publication No. 1149: End Results in Cancer, Report No. 2, U.S. Government Printing Office, Washington, D.C. 20402, 122 pp., 1964.

6. Zippin, C.: Comparison of the international and American systems for the staging of breast cancer, J. Natl. Cancer Inst., 36:53-62, 1966.

Narcotic and Dangerous Drug Problems

Current Status of Legislation, Control and Rehabilitation

WILLIAM F. QUINN, M.D., *Los Angeles*

RECENT LEGISLATION reclassifies dangerous drugs into those in which there are problems of abuse and those in which there are no problems, which is a constructive step. Other legislation included but was not limited to stricter inventory controls on dangerous drugs. This legislation requires triplicate forms for Percodan®* prescriptions, forbids over-counter sale of cough syrup mixtures containing codeine and makes the rehabilitation facilities for addicts at Corona available to the noncriminal user who is in imminent danger of addiction although not necessarily yet an addict.

California is showing leadership in this field and the programs are being copied by other states. Federal legislation in this field also appears imminent.

Rehabilitation

It has been recognized that while the addict is indeed a sick person and is suffering from a disease, he also has brought the disease upon himself and, in general, seems desirous of transmitting the disease to others.

The treatment of his disease therefore does involve quarantine in a narcotic-free atmosphere rather than perpetuation of his addiction by making narcotics easily available to him.

The former Naval hospital at Corona has been acquired by the State of California as surplus property and its 2,300 beds have been made available for treatment and rehabilitation of narcotic addicts.

Most addicted persons in California have been criminals before they became addicted, and the addiction is merely another incident in a life of nonconformity. The characteristic addict is about 24 years of age, has been arrested for four or five other crimes such as forgery, theft of automobiles and robbing of liquor stores, before he became involved with narcotics. In other words, he was a criminal first and an addict second.

It is now possible for a judge to suggest to the addict at a criminal hearing that since his desire for narcotics was part of his criminal activities, he might be interested in volunteering for rehabilitation at Corona, and if he did volunteer, his criminal trial would be held in abeyance until it had been determined whether or not he could be rehabilitated. The general idea is that if he makes the grade at Corona, the criminal charges might be dismissed in the light of his rehabilitation.

The facility at Corona has had reasonable success with a program of rehabilitation which has departed from the revolving door philosophy wherein the criminal is off narcotics for a short time and then leaves the institution and promptly begins using them again.

This is accomplished by a change in legal philosophy; namely, that the situation is comparable to that associated with mental illness wherein the mentally ill person is under the jurisdiction of the courts until his illness has been corrected. Therefore, the time element involved is quite flexible. In this system, the addicted person is under the authority of the rehabilitation center until those involved feel that he has indeed been rehabilitated. The time involved may be months or it may be many years, but the attitude is to cure the patient, not to sentence him.

The rehabilitation program is a combination of in-patient and out-patient service in which vocational rehabilitation and group therapy are emphasized, with the addicts actually helping themselves

---

The author is chairman of the CMA Committee on Dangerous Drugs and of the California Narcotics Rehabilitation Advisory Council. The article was written in furtherance of a House of Delegates resolution that physicians be informed as to the current status of the problem.

Reprint requests to: 1401 South Hope Street, Los Angeles 90015.

*A mixture containing dihydrohydroxycodeinone.

to become aware of their fundamental problems and to create motivation to solve them. It is important that job training and efforts to create dignity and the sense of worthwhileness in achieving some stature in life are paramount in the program.

The parole officers who ordinarily have a case load of about 75 to 90 have a case load of only 30 and, therefore, can devote more time to the individual problems of each resident and establish a personal and friendly rapport which is extremely important in producing results.

Halfway Houses

The establishment of halfway houses wherein during the first six months to a year after release the ex-addict can return for help during this critical period is extremely important and worthwhile. Most of the people currently being committed to the program have been guilty of criminal activities and are, therefore, conceded to be hardcore addicts. Thus far, 1,200 persons who have been treated under the program have been narcotic-free for almost two years, are holding down jobs, and have not returned to crime. This represents a 30 per cent rehabilitation rate for those released for all reasons, including technical reasons, so that the rate is really somewhat better than this figure would indicate. This compares so well with the usual 2 to 5 per cent rehabilitation rate that restrained optimism seems warranted.

The general philosophy parallels the rehabilitation efforts which resulted in a surprisingly high rehabilitation rate in addicted physicians, as reported in an article published in CALIFORNIA MEDICINE in April 1961: A 10-year follow-up on 130 physicians revealed a 92 per cent rehabilitation rate. Naturally it will not be as easy to create motivation in the routine addict, but if the 30 per cent rate can be maintained, this would appear to be the most successful large-scale effort to date.

Better results may well be anticipated as there is an increase in the proportion of the population at Corona made up of those who have volunteered for the program when they are in imminent danger of addiction rather than long-time addicts. The Narcotic Rehabilitation Advisory Council appointed by the Governor reports on the progress of the program each year to the Governor and the legislature. The results today certainly justify restrained optimism, and in a few years we will undoubtedly have documentary evidence as to its justification or lack of it.

Voluntary commitment parallels the approach to mental health wherein the individual may be observed for a period of 72 hours if necessary, and then a determination made as to whether or not he should be committed to the program.

Marijuana

Categorically marijuana is not an opiate and an addicting drug, but this pattern is quite clear: Unstable persons under marijuana will often eventually become involved with heroin addiction, whereas had marijuana not been available to them, this probably would not have occurred. It therefore seems simple enough that since there is no medical indication for the use of marijuana, it should be classified as a dangerous drug and unsafe for self-medication, and the penalties for possessing or peddling it should be comparable to those for narcotic drugs.

Percodan®

As noted in a previous article, Percodan® has been placed on the triplicate form since evidence presented to the legislature suggested that its abuse might be curtailed thereby. The Endo Company previously opposed this legislation but in a constructive attitude did not oppose it at the most recent consideration in the legislature, feeling that this was a matter to be decided by physicians, law enforcement agencies and legislators.

It is hoped that the usefulness of this product will be in no way curtailed by the triplicate form, but on the other hand abuses would be largely eliminated.

Education of Physicians

Remarkable progress has been achieved in the education of medical students to the calculated risk of addiction among persons to whom narcotics are easily available. It is pointed out that once addicted such individuals are no different from any stumblebum on skid row, and that narcotics are not something which they can take or let alone.

In the past 12 years, panel discussions on this subject have brought about a 60 per cent decrease in the anticipated addiction rate among medical students who attended.

Emphasis has also been placed on the fact that it is the duty of the physician himself to explain to the unstable person that giving him narcotics to relieve all the minor aches and pains in life is not doing him a favor. With such persons it is better that the physician obtain their confidence and then explain that they must learn to live with their migraine, their dysmenorrhea, their backaches rather than expect complete relief when such relief may indeed create more problems than are solved.

Cough Syrups

Another interesting development has been the abuse of exempt codeine-containing cough syrup compounds. The exempt mixtures have been subject to much abuse by unstable juveniles, with criminal activity involving rapes, stabbings and fights as a result.

The original approach was that the mother with a child with a cough should be able to obtain these mixtures without the necessity of taking up the time of a physician in order to obtain these compounds.

The position of the California Medical Association was that if these were available by telephone authorization for which no charge would be made, then there would be no objection to making these compounds available only on prescription. The legislators agreed, and these compounds are now available on prescription only.

Abuse of Dangerous Drugs

The severe penalties involved in peddling narcotics have been such that many peddlers have turned to the dangerous drug group, namely, the amphetamines and barbiturates, as a source of livelihood.

Since no one has any sympathy for the peddler, it was not difficult to initiate and have passed legislation which makes the penalties for peddling of dangerous drugs comparable to those for peddling narcotics. Increased penalties for illegal possession are also now in effect.

Synanon

The Synanon Foundation is an in-patient program run by former addicts for addicts who voluntarily seek help. Its work has been widely reported by the communications media but is subject to considerable controversy. Evaluation of its success is extremely difficult.

The approach is somewhat comparable to the philosophy of Alcoholics Anonymous, and certainly any efforts in this direction should receive encouragement. Unfortunately, scientific evaluation of its results seems to be resented by those in charge of the program. Some of its original supporters are becoming a bit restrained in their enthusiasm although in others it remains undiminished. Time as well as documented statistics eventually will determine the validity of the claims for this fascinating experiment.

The "British System"

One facet of a California Medical Association House of Delegates Resolution was that of education of physicians. It might be well to point out that perennially the subject of the so-called British system of the approach to narcotics, that is, to eliminate the profit motive by providing narcotics at cost, comes up again and again.

Most knowledgeable persons who have pursued the subject—and this includes a group from New York whose representatives went to England and studied the question thoroughly—point out that the boons of the system are less than meets the eye, for even with over-counter sale, there just never was much of a narcotic problem in England. The system would indeed merely perpetuate addiction and even in England it doesn't seem to work anymore.

Gene Sherman, the Pulitzer prize winning Los Angeles *Times* correspondent in London, has recently written an article which states that the British themselves have become disillusioned with the system since the rate of addiction has increased 100 per cent or more in the last few years, and they recognize that making narcotics easily available will merely perpetuate rather than contain addiction.

An artificial paradise wherein all problems are alleviated or at least postponed is attractive enough to the unstable individual but certainly is no answer to the problem itself.

The high incidence of addiction in physicians, pharmacists and nurses with easy availability of narcotics, and without any profit motive at all being involved, should negate the philosophy that elimination of the profit motive might help solve the problem.

High Mortality Rate

At a recent meeting of the National Research Council evidence was presented on 2,743 addicts between the ages of 15 and 25. Their mortality rate was 26 per thousand compared with 0.99 per thousand of nonaddicted persons of that age in New York. In other words the addicts had 26 times the mortality rate of nonaddicts.

Methodon and Cyclazocine

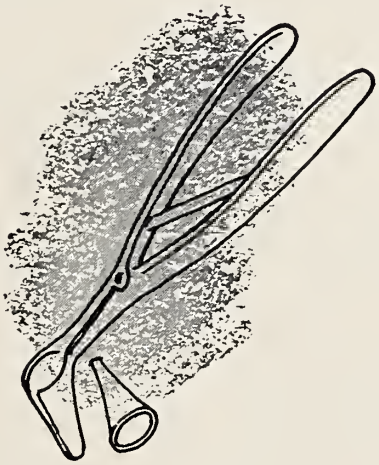
The results of several pilot programs wherein methodon and cyclazocine are used in maintenance dosage as substitutes by former heroin addicts will be awaited with much interest. While these substitutes are themselves addicting, they appear less addicting than heroin, and apparently they negate the desire for heroin euphoria. Presumably the hope is that during the period of maintenance on these substances, the deep-seated emotional problems leading to the original addiction can be understood and solved. Apparently at present few of those involved have been withdrawn from the substitute therapy. Those involved with the projects appear to be well informed and dedicated persons, although with a somewhat evangelical zeal for their point of view.

The final answer, of course, is to find a pain-relieving drug which does not also produce euphoria; and there is restrained optimism that this may not be too far off. This would then produce a situation wherein the United Nations might receive strong support in producing a climate of cooperation in which the need for the production of legitimate narcotics would no longer exist. More, the uncooperative nations such as Red China, India, Turkey, France and Mexico, would then not have the excuse that they merely produce a legitimate product for medicinal purposes, and it is not their fault if there are abuses.

In summary then the climate is one of flexibility with a constructive and realistic approach to these problems by the combined efforts of legislators, the medical and legal professions, researchers, persons engaged in law enforcement and rehabilitation work, and interested citizens.

GENERIC AND TRADE NAME OF DRUG

Percodan® (a mixture containing dihydrohydroxycodeinone).



Roentgen Findings in Occipital Pole Tumors

LARRY BILODEAU, M.D., AND WILLIAM HANAFEE, M.D., *Los Angeles*

■ *The arteriographic findings of occipital pole tumors consist of the following:*

1. *Anterior displacement and elevation of the middle cerebral group.*
2. *"Accordioning" of the anterior choroidal artery.*
3. *Anterior displacement of the lateral branch of the posterior choroidal artery.*
4. *Local stretching of vessels about the tumor.*
5. *"Onion skinning" of the pial vessels.*
6. *Anterior displacement of the vein of the occipital horn.*
7. *Greater displacement of the internal cerebral vein than of the anterior cerebral artery.*

Air studies require precise radiographic technique lest the subtle displacements of the occipital horn and trigone area be missed.

OCCIPITAL POLE TUMORS are relatively uncommon and frequently defy early roentgen diagnosis. The purpose of this communication is to review the criteria for diagnosis, using angiography and air contrast studies, as related to our experience.

Material

The material consists of 19 pathologically proven occipital pole lesions seen at the UCLA Center for the Health Sciences from the years 1955 through 1965. The lesions consisted of 10 gliomas, three metastatic tumors, two meningiomas, one ependymoma, one abscess, one epidermoid tumor and one arteriovenous malformation. For a tumor to remain localized to the occipital lobe is uncommon. Gliomas usually extend from the occipital lobe to invade the posterior temporal or posterior parietal lobes. More frequently the occipital lobe is involved secondarily as a result of extension of tumors originating in adjacent areas. Meningiomas arising from the torcular Herophili are well known.

Anatomy

The occipital lobe contains about 50 gm of tissue, about 8 per cent of the volume of the supratentorial portion of the brain.[1] The lobe lies in a well protected rigid sling with its medial and inferior surfaces against the tough fibrous tentorium and posterior falx, while its lateral surfaces are against the occipital portion of the calvarium. The topography of the occipital lobe presents three surfaces. The lateral surface is limited in front by the parieto-occipital fissure and by a line connect-

---

From the UCLA Center for Health Sciences, Los Angeles.

Presented before the Section on Radiology at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: Department of Radiology, University of California Center for the Health Sciences, Los Angeles 90024 (Bilodeau).

ing the lower end of this fissure to the pre-occipital notch. The medial surface of the occipital lobe extends anteriorly to the medial part of the parieto-occipital fissure. The calcarine fissure subdivides this surface into the cuneus superiorly and the lingual gyrus inferiorly. The tentorial surface is limited in front by an imaginary transverse line through the pre-occipital notch.[2]

Technique of Examination

Angiography in the early part of the series was conducted by the direct needle puncture of the common carotid artery. Later, examinations were performed by selective catheter technique with separate contrast injections into the internal and external carotid arteries. The catheter method has been previously described[5] and the only change in our technique has been the use of a smaller polyethylene catheter than that formerly used for carotid angiography—0.037 inch outside diameter against 0.045 previously. No change has been made in the size of the catheter used for vertebral angiography, whether by the transfemoral or the axillary approach.[4]

Pneumoencephalography was by the fractional technique. The air for ventriculography was instilled as a surgical procedure.

TABLE 1.—*Roentgen findings in 19 pathologically proven cases of occipital pole tumors at UCLA Center for the Health Sciences for the years 1955 through 1965.*

	Cases
ARTERIOGRAPHIC FINDINGS IN 14 STUDIES	
I. Arterial findings	
A. Anterior choroidal artery (accordioned and forward displacement)	10*
B. Middle cerebral group of vessels (forward displacement and elevation)	11
C. Lateral branch of posterior choroidal artery (forward displacement)	3
II. Late arterial and capillary findings	
A. Local stretching	11
B. "Onion skinning"	6
III. Venous findings	
A. Displaced veins of occipital horn	2†
B. Lateral shifting of internal cerebral vein	9
C. Displacement of the internal cerebral vein greater than anterior cerebral	4
D. Occlusion of the sagittal sinus	0
AIR CONTRAST IN SIX EXAMINATIONS	
I. Non-filling of occipital horn	6
II. Anterior shift of trigone	—

*In two cases the artery was not seen, in one case it was stretched and in one it was normal.

†In 12 cases non-visualized.

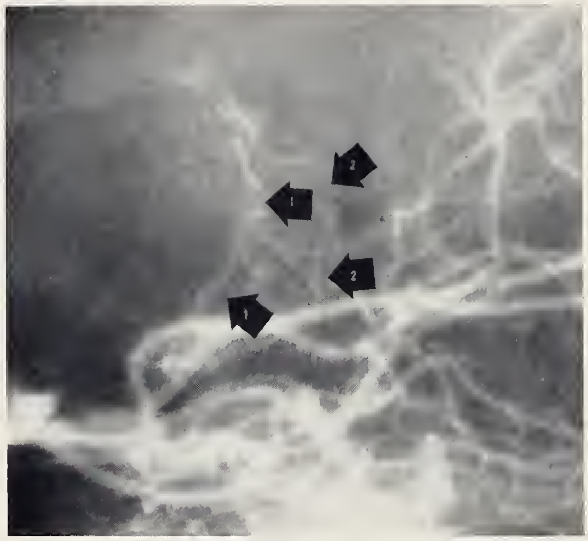


Figure 1.—The medial posterior choroidal artery (2→) and lateral posterior choroidal arteries (1→) are visualized on the lateral projection of a vertebral angiogram. The medial posterior choroidal, as it passes over the quadrigeminal plate to enter the roof of the third ventricle, is a midline structure and shifts very little. The lateral posterior choroidal artery passing around the pulvinar thalamus in the trigone is displaced forward by occipital tumors.

Analysis of Material

As noted in Table 1, most of the patients were investigated by arteriography rather than pneumography, which merely reflects a preference for arteriography in cases of suspected increased intracranial pressure. Vertebral angiography should be the examination of choice. More use of the method will undoubtedly be made in the future because of the ease of performing vertebral angiography by the catheter technique. Any method that fills the posterior cerebral artery will demonstrate the blood supply of the tumor.

The posterior choroidal artery has two branches, a medial branch which courses in the roof of the third ventricle and a lateral branch which passes posterior to the pulvinar thalamus. In the lateral view, the lateral branch will normally be projected posterior to the medial branch. As occipital pole tumors expand, the trigone area containing the lateral branch of the posterior choroidal artery is displaced forward (Figure 1). On the lateral projection the smooth curved sweep of the lateral branch of the posterior choroidal was shown in three of the patients to be displaced anterior to the medial branch. Vertebral angiography is necessary for demonstration of these small branches. If internal carotid angiography is used, the posterior cerebral artery may fill and contrast medium will enter the posterior choroidal arteries, but accurate delineation of these vessels is usually prevented by the overlying branches of the middle cerebral complex together with the anterior choroidal artery.

On internal carotid angiography the most common finding was forward displacement and elevation of the posterior portion of the sylvian group, middle cerebral artery branches (Figure 2A).[3,7] Almost as frequently seen was foreshortening (accordion sign)[10] of the anterior choroidal artery (Figure 2B). Both of these findings are related to the fact that the trigone area and posterior portion of the sylvian fissure are displaced forward by the occipital expanding process. Since the entire occipital lobe is at or below the level of the sylvian fissure in a vertical plane, any expansion in this area will cause upward as well as forward displacement of the middle cerebral group.[12] The forward and upward displacement of the middle cerebral group aids in a clearer delineation of the anterior choroidal artery, frequently without overlapping vessels (Figure 2B). Since the anterior choroidal artery usually terminates at the level of the glomus of the choroid plexus in the region of the trigone, it will be foreshortened and "accordioned" upon itself. This configuration is not specific for occipital pole tumors, however, since intraventricular tumors may produce a similar displacement of the anterior choroidal artery.

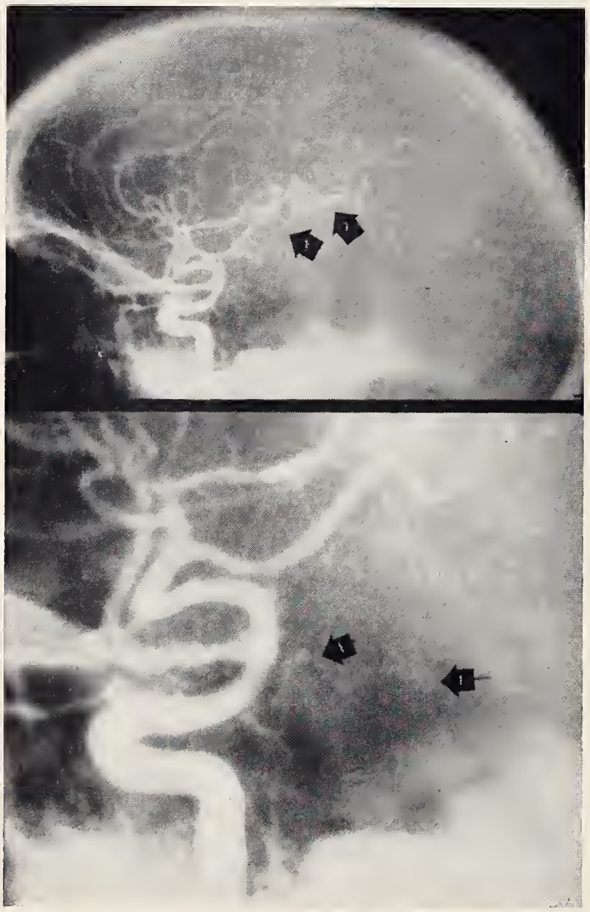


Figure 2.—*Above,* the occipital lobe is considerably caudad to the sylvian fissure. Expansivity in the region causes elevation, as well as forward displacement, of the branches of the middle cerebral artery. *Below* (same patient), the anterior choroidal artery normally terminates at the glomus of the choroid plexus in the trigone. This entire region becomes displaced forward, causing a fore-shortening of the artery. In addition, the elevation and forward displacement of the middle cerebral complex allows better visualization of the anterior choroidal artery.

In the late arterial and capillary phase, small localized expansions frequently are manifested. The best criteria of tumor localization is local stretching of the artery adjacent to the tumor. This was demonstrated in 11 of 14 arteriograms in the present series.

Six of the patients in the series showed "onion skinning" as described by Taveras,[12] a condition caused by edema in the surrounding brain adjacent to a tumor. With edema, the convolutions tend to become swollen and are compressed into a more parallel course. The small capillaries in the fissures will then line up in a curvilinear parallel fashion about the tumor much like the ribs of an onion skin.

The deep cerebral veins give considerably more information regarding the deep cerebral structures than the arterial phase of the arteriogram.[9] Lateral shifting of the internal cerebral vein occurred in nine of the patients, and in four cases the vein shift was greater than that of the anterior cerebral artery. The displacement definitely placed the lesion in the posterior half of the skull but was not specific for an occipital pole lesion. Anterior displacement of the veins of the occipital horn was described by Wolf[15] as a sign of occipital pole tumor. In two of the patients in the present series such a displacement was noted, but we were unable to identify the vein of the occipital horn in the 12 remaining arteriograms.

Occlusion of the sagittal sinus may occur with tumors invading the dura such as meningiomas. This not only has localizing value as to the extent of the tumor but also implications regarding surgical therapy, for if the tumor has totally interrupted the superior sagittal sinus and the patient is still functioning, it can be assumed that adequate collateral circulation is present and that the tumor may be removed in toto without fear of fatal occlusion of the sinus by edema in the postoperative period.

Subtraction technique is exceedingly helpful if not essential for a conclusive roentgen diagnosis of patency or occlusion of the sagittal sinus. We have only had this available in the last several years. In neither of the two cases of meningioma in the present series were we able to make a diagnosis of occluded sagittal sinus.

Air Contrast Studies

Ventriculography can be extremely difficult if not dangerous in patients with occipital pole tumors. Because of the distortion and displacement of the trigone area, the ventricles may be shifted considerably from their normal position.[9] Frequently at ventriculography, air can only be injected into the side not affected by tumor and then must be maneuvered through the intraventricular foramen to fill the side to be examined.

In six of the patients in the present series the occipital horn did not fill whether examination was by pneumoencephalogram or by ventriculography. Two patients showed anterior shift of the trigone, which at times may be exceedingly difficult to appreciate.[6,7] In one instance, the trigone area was massively shifted and constricted. Only a small amount of air could be trapped in the trigone on the prone views. On the translateral projection this small collection of gas was obscured due to its superposition on the third ventricle (Figures 3A and 3B.)

With the patient in the prone position, translateral projections are essential to show minor forward shift of the trigone area. Obtaining true lateral projections is sufficiently difficult that we have adopted a principle of always taking three translateral views at one sequence in occipital pole tumor suspects (Figure 4). The first view is an attempt at a true lateral. The second view is made with the x-ray tube angled 5 degrees toward the floor and the third with the tube angled 5 degrees toward the ceiling. These three views will provide at least one true lateral projection which with the other two views, may be used for stereoscopic vision.

The midline structures of the pneumoencephalogram will reflect the same displacements as the angiogram. The third ventricle will usually be shifted to a greater extent than the anterior portion of the septum pellucidum due to its proximity to the tumor.[6]

Discussion

At times angiography and pneumoencephalography not only locate the tumor but may give valuable clues to the histological diagnosis.

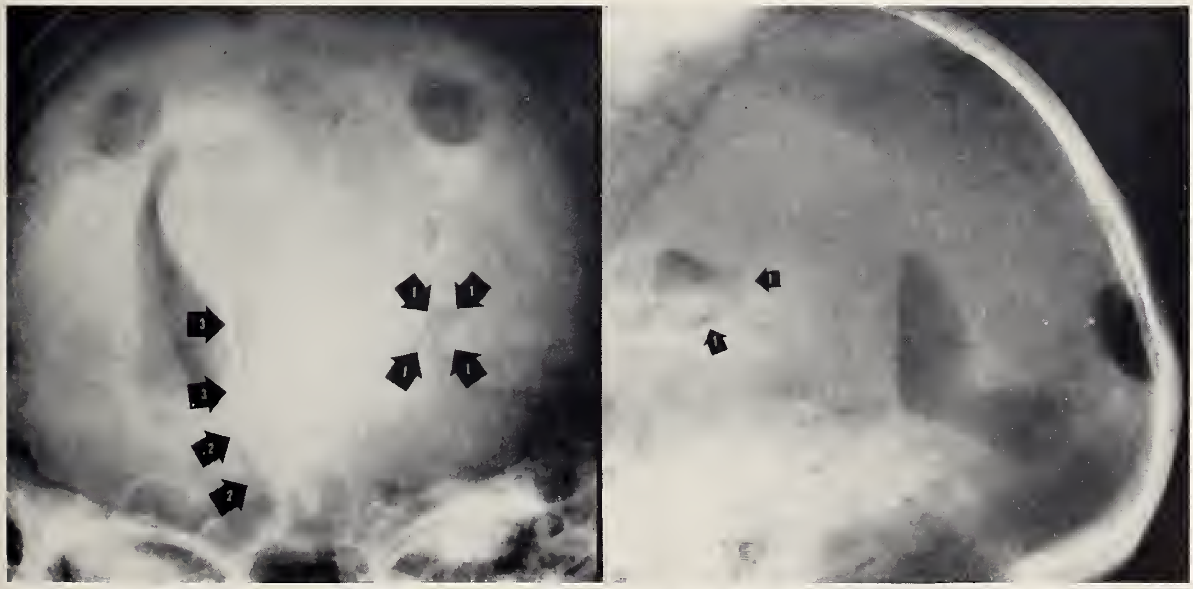


Figure 3.—Ventriculograms demonstrating displacement of the third ventricle (2→) to a greater extent than the septum pellucidum (3→). The trigone (1→) of the lateral ventricle has a diffuse appearance on the posteroanterior projection and is displaced decidedly forward on the lateral view.

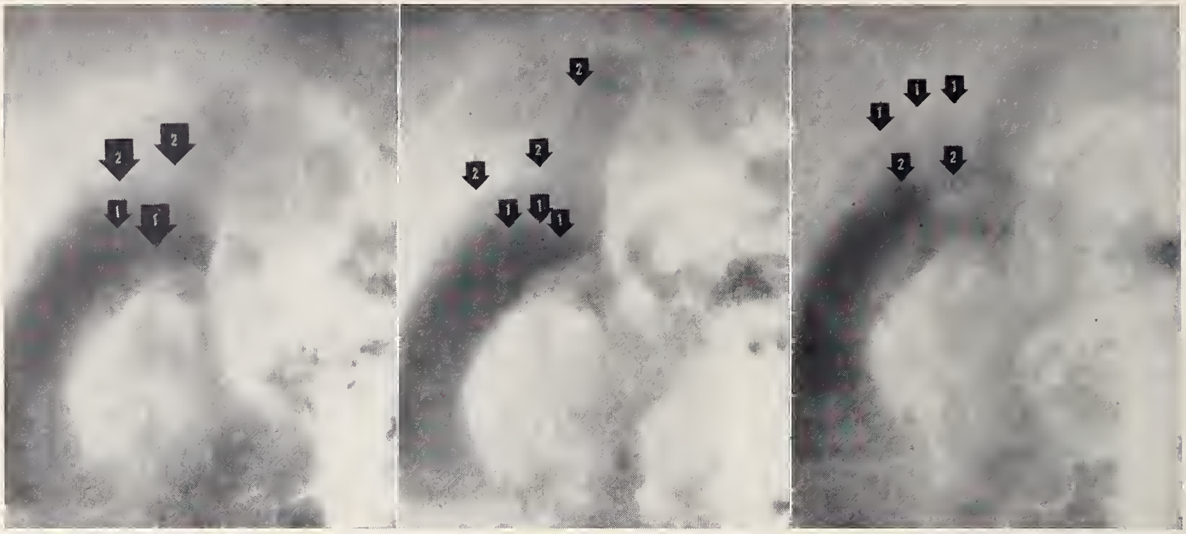


Figure 4.—A method for obtaining true translateral views when occipital pole tumor is suspected. Occipital horn and trigone areas are identified by arrows (normal 2→) and abnormal (2→). The film at left is true lateral; the x-ray tube is not angled. Center film is with lateral bow down; the x-ray tube is angled 5° toward the floor. At right, lateral bow down; the tube is angled 5° toward the ceiling.

With the three views, an indentation on the medial-posterior wall of the trigone that might otherwise be missed can be appreciated.

Malignant tumors of the occipital lobe characteristically derive their blood supply from the leptomeningeal vessels, that is, the posterior cerebral artery or the posterior temporal and angular branches of the middle cerebral artery.

During the late arterial phase and capillary phase of the angiogram, one or more of the following signs of malignant growth may be found[13]:

1. Vessels of irregular lumens and bizarre branching;
2. Puddling of contrast material in irregular venous channels;
3. Arteriovenous shunting with early filling of deep cerebral veins.

For benign lesions, such as meningiomas, one should look for hypertrophy of the dural branches of the internal carotid artery[8,11,14] or enlargement of the dural branches of the vertebral artery as they ascend from the vertebral artery at the level of the foramen magnum to supply the falx cerebelli and posterior portions of the tentorium.

All of these roentgen signs of etiological diagnosis are relative and should be considered in the light of the clinical findings. Often malignant tumors may cause no roentgenographically observable change in surrounding vessels; and sometimes with benign meningiomas there may be diffuse staining of the kind associated with metastatic tumors.

REFERENCES

1. Ecker, A., and Riemenschneider, P. A.: Angiographic Localization of Intracranial Masses, Charles C Thomas, Springfield, Illinois, 1955, p. 292.

2. Gray, H., and Goss, G. M.: Anatomy of the Human Body, Lea & Febiger, Philadelphia, Pa., p. 845.

3. Greitz, T., and Lindrgen, E.: Supratentorial Expanding Lesions from Angiography. Edited by H. H. Abrams, Little, Brown and Co., Boston, 1961, p. 107.

4. Hanafee, William: Anillary artery approach to carotid, vertebral, abdominal aorta, and coronary angiography, Radiology, 81:559-567, October 1963.

5. Hanafee, W., and Weidner, W.: Selective carotid angiography, Radiology, 81:589-591, October 1963.

6. Robertson, E. G.: Pneumoencephalography, Charles C Thomas, Springfield, Illinois, 1957, pp. 189, 272.

7. Schinz, H. R., Baensch, W. E., Friedl, E., and Vehlinger, E.: Roentgen Diagnostics, Vol. II, Grune and Stratton, Inc., New York, 1952, pp. 1661, 1728.

8. Schniirer, L. B., and Stattin, S.: Vascular supply of intracranial dura from internal carotid artery with special reference to its angiographic significance, Acta Radiol., Diagnosis, 1963, 1:441-450.

9. Shanks, S. C., and Kerley, P.: A Textbook of X-Ray Diagnosis, Third Edition, Vol. 1, W. B. Saunders Company, Philadelphia and London, 1957, pp. 118, 197.

10. Sjogren, E. S.: The anterior choroidal artery, Acta Radiologica, 46: July-December 1956.

11. Stattin, S.: Meningeal vessels of internal carotid artery and their angiographic significance, Acta Radiol., 55:329-336, 1961.

12. Taveras, J. M., and Wood, E. H.: Diagnostic Neuroradiology, Williams and Wilkins Co., Baltimore, 1964, pp. 1615-1618.

13. Wichigom, I.: Angiography of carotid artery, Acta Radiol., 1948, Supplement 72.

14. Wilson, G., Weidner, W., and Hanafee, W.: The demonstration and diagnosis of meningiomas by selective carotid angiography, Am. J. Roent., 95: December 1965.

15. Wolf, Bernard: Personal communication.

Methods of Training in Community Mental Health

JOHN S. VISHER, M.D., *San Francisco*

ONE OF THE MOST EXCITING and important developments in psychiatry in many years is the concept of comprehensive community mental health as discussed by Harris[5] in a recent issue of CALIFORNIA MEDICINE. Treatment of acute psychiatric illness is shifting from the isolated mental hospital into the community, and is of shorter duration. Outpatient programs are more effective, and a variety of alternatives to staying in hospital the clock around are now available. New techniques of prevention are being developed, utilizing mental health education and consultation to agencies in the community providing caretaker services to the emotionally disturbed.

History of Community Mental Health In California

Community mental health in California received its greatest impetus only nine years ago with passage of the Short-Doyle Act[2] which provided partial financial support for a variety of mental health services under local administration and control. Subsequent state and federal legislation has further assisted local mental health services. The Community Mental Health Centers Act of 1963[3] authorized federal financial support for construction of physical facilities for mental health centers meeting certain criteria. Two years later, federal support was given for staffing of community mental health centers for demonstration purposes.[4] Most counties in California now have more or less elaborate mental health services. The demand for trained mental health professionals is far exceeding the supply.[6]

Training of Mental Health Professionals

To assist in meeting this need for trained professionals, in 1959 the California Department of Mental Hygiene through the Langley Porter Neuropsychiatric Institute Outpatient Department began to offer some experience in community organization and consultation to third-year psychiatric residents. By 1963 a full-time training staff was approved: A psychologist, a psychiatric social worker, a mental health nurse consultant and a psychiatrist. A part-time psychiatrist heads the program, and also engaged in it are a research sociologist and a psychoanalyst who are staff members. The staff, in addition to the author, consist of: M. Robert Harris, M.D., director; Fariborz Amini, M.D.; Egon Bittner, Ph.D.; W. Reed Brockbank, M.D.; Rosalie M. Jones, R.N., M.S.; Betty L. Kalis, Ph.D.; Helen Swick Perry, A.B.; Lida Schneider, A.M.; and J. M. Stubblebine, M.D. Important teaching contributions are also made to the program by unsalaried clinical faculty members and a variety of agency and program representatives. A grant from the National Institute of Mental Health made possible an advanced program for fourth-year and fifth-year fellows who are interested in careers in community mental health.

Who Is Trained

The advanced fellows may be either psychiatrists who have finished their training in psychiatric residencies or post-doctoral clinical psychologists, and they are in the program for one-year periods, full-time. Psychiatric residents in the third year of their residency may elect training half-time for six months. Social work students in the second year

---

From the Department of Psychiatry, University of California School of Medicine and Langley Porter Neuropsychiatric Institute.

Submitted 16 June 1966.

Reprint requests to: Langley Porter Neuropsychiatric Institute, 401 Parnassus Avenue, San Francisco 94122.

of the Master's Degree social work program at the School of Social Welfare, University of California, Berkeley, receive training one-third time for three to six months, and limited practical experience in the community is occasionally offered to medical and criminology students.

Methods of Training

Seminars

Seminars consist of presentations and discussion by staff, by students and by visiting mental health professionals. A suggested bibliography is usually prepared in advance so that those participating can become familiar with the pertinent literature. Many theoretical and practical topics are considered: Mental health consultation, crisis theory, epidemiology of mental illness, research and program evaluation, poverty, social class and mental illness, community organization, mental health and the law and many others. Trainees also present reports of their experiences to the group.

Community Experience

In addition to attending seminars, each trainee receives a varied experience in practical community organization and its problems. This comes through his assignment to a "Geographic Focus," a limited area where his community experiences may be concentrated. He studies middle class, segregated or slum areas through meeting with concerned and involved professional and lay people. He talks to public health officials and accompanies public health nurses on their calls, and he regularly attends the meetings of the planning councils and agencies. He offers consultation services to one or more agencies or organized groups. For example, the staff of a senior citizen group may request consultation about problems of their clients, or a recreational agency that offers services for the physically and emotionally handicapped may wish help in understanding its clients, its staff relationships or the need for new services to the group which it serves.

Staff Involvement in the Community

Involvement with the community is arranged on an individual basis for each trainee by a staff member who maintains a close working relationship with the staff of scores of agencies cooperating in the training program. One psychiatrist staff member, for example, is a consultant to the Bureau of Social Work, an agency offering aftercare services to patients on leave from state hospitals of the Department of Mental Hygiene. Another staff member, a clinical psychologist, maintains contact with the San Francisco Department of Education and arranges opportunities for trainees to act as consultants in schools, where they become familiar with the problems of school guidance. Attention is also given to political problems, and staff members and trainees often attend meetings of important boards and political bodies. Since one possible role for a psychiatrist who is a trained community mental health professional might be as a staff member or administrator, it is especially important for trainees who may be interested in this professional career to become familiar with one or more local mental health service programs and the role of the program chief.

Consultation with Trainees

Each trainee consults with staff members of each professional discipline, but his major consultation is with a staff member of his own profession. Thus a psychiatric resident trainee meets with a psychiatrist staff member once each week for an hour and with staff representatives of other disciplines less intensively. We believe that this method is helpful in assisting the identification of the trainee with a professional person of his own discipline, while at the same time offering some direct exposure to the varied viewpoints of other disciplines.[1] A psychiatric resident realizes that his identity as a psychiatrist is not lost in community mental health, and that his knowledge of dynamics, psychopathology and treatment is useful to him in his community role.

Special Studies

Finally, each trainee is assigned a special study project at the beginning of his training experience, and special opportunities are created for him to become familiar with agencies concerned with his particular subject. A trainee studying the mental health problems of the unwed mother, for example, might consult or observe at a home for unwed mothers, participate in the program of the YWCA, which sponsors group activities for unwed mothers living at home, and visit pregnant girls and their families with a district public health nurse. At the end of the period of training, the trainee writes a project report on his topic and presents it to his fellow students and the training staff.

Summary of Experience

During his time in the program, each trainee explores at least one area of San Francisco, and becomes familiar with the caretaking agencies in his area. Each trainee also participates as a mental health consultant and discusses his consultation with one or several staff members. He also studies in depth a special field of interest. He becomes acquainted with some of the literature of community mental health and participates in seminar discussions of current theory and practice in this developing new discipline.

REFERENCES

1. Brockbank, R.: Problems of self-identity in mental health and mental illness, from Mental Health in a Changing Community, Brockbank, R., and Dorothy Westby-Gibson (eds.), Grune & Stratton, New York, 1966.

2. California Department of Mental Hygiene: California's Community Mental Health Services Act: The Short-Doyle Act, May 1962.

3. Community Mental Health Centers Act of 1963, Title II, Public Law 88-64, U.S. Government Printing Office, 1965.

4. Community Mental Health Centers Act, Amendments of 1965, Public Law 89-105, U.S. Department of Health, Education and Welfare, Public Health Service.

5. Harris, M. R.: Community Mental Health, Calif. Med., 104:454-457, June 1966.

6. Langsley, D. G., and Harris, M. R.: Community Mental Health Activities of Psychiatrists, The Psych. Quart. Suppl., 34:314-325, 1960.

Bladder Tumor Diagnosis

Improved Excretory Cystograms

ARJAN D. AMAR, M.B.B.S., *Walnut Creek*

■ *The correct precystoscopic diagnosis of bladder tumor was made in 20 of 23 patients with this disease, among more than 1,000 persons studied by double-dose excretory urography. There was no increase in the incidence of untoward effects. Double-dose excretory urography with delayed bladder films is recommended as the primary urographic procedure in all patients with gross or microscopic hematuria in whom bladder tumor is suspected.*

ROUTINE EXCRETORY UROGRAPHY with the standard-dose of contrast medium usually gives little or no indication of bladder tumor. Double-dose excretory urograms with delayed films of the bladder[1,2] led to the correct precystoscopic diagnosis in 20 of 23 patients with such lesions seen in our clinic during the last two years.

This high rate of prediction is worthwhile. Advance knowledge that a tumor is probably present minimizes the dismay of discovery, and the lack of preparation for it, at the time of cystoscopic examination. With foreknowledge, the surgeon can make adequate arrangements for general anesthesia, biopsy, fulguration and transurethral resection concurrently with endoscopy. When bladder tumor has been suspected because of signs, symptoms or previous history, this method may provide an advance estimate of the probable size and site of the lesion, and even the type. If bladder tumor is associated with other disease that has been believed to offer adequate explanation for the clinical manifestations, warning that a bladder tumor is also present has led to improved treatment of both conditions. Additional tumors in the upper urinary tract have been delineated as clearly as by retrograde urography. Excellent information about other lesions that must be differentiated from bladder tumor is obtainable by this method. In patients with good renal function, the concentration of contrast medium, which approaches 10 per cent, gives clear radiographic visualization of the entire urinary tract. The improved cystographic quality makes retrograde cystography unnecessary.

In more than a thousand patients studied by double-dose excretory urography in our clinic, there has been no increase in the incidence of untoward reactions. This simple modification of excretory urography, which makes available so much additional information about the total urinary tract at no extra risk or inconvenience, is recommended as the primary urographic procedure in all patients with gross or microscopic hematuria.

Method and Material

The patient is prepared as for routine intravenous urography and is asked to empty the bladder. A scout film of the abdomen is then made. A double-dose (adult, 60 ml) of 50 per cent diatrizoate (Hypaque®) is injected intravenously, and anterioposterior films of the abdomen are made at five, 10 and 15 minutes. Upright anteroposterior and right and left oblique films of the abdomen are also obtained at this time. Additional anteroposterior views are taken at one and two hours. The patient then again voids, and an anteroposterior

---

From the Departments of Urology, The Permanente Medical Group, Kaiser Foundation Hospital, Walnut Creek, and Presbyterian Medical Center, San Francisco.

Presented before the Section on Urology at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: 1425 South Main Street, Walnut Creek 94596.

postvoiding film is made. Films may also be exposed during voiding, and cineradiographic control may be employed during this phase. Since it is easier to detect fine detail in the early phase, before the picture is obscured by the complete filling of the bladder with dye, the films made up to 30 minutes are scrutinized with special care, and, if necessary, additional views are obtained. Both supine and prone positions are used where indicated (as Elkin[4] has emphasized, lesions of the anterior wall are best delineated with the patient prone).

Double-dose excretory urography has been used in more than a thousand patients in our clinic during the past five years. In the last 500 cases, delayed films up to two hours were made.

Results

The presence of bladder tumor was proved by cystoscopic examination and biopsy in 23 of the 500 patients studied with delayed films. In 20 of the 23 patients, the existence of such a neoplasm was indicated on the double-dose excretory uro-

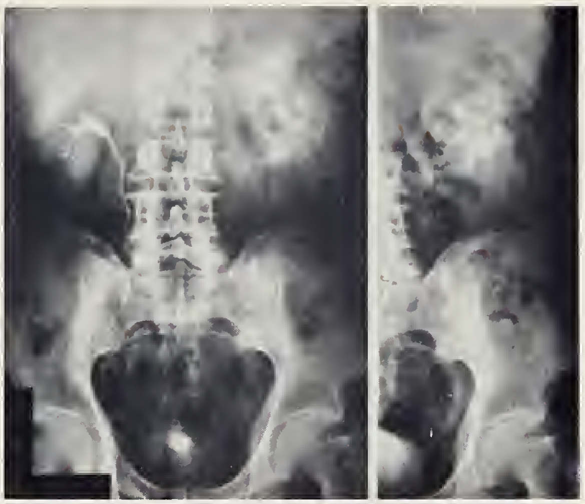


Figure 2.—Double-dose excretory urogram of a 63-year-old man with irritated bladder symptoms including urinary frequency and microscopic hematuria. *Left*, on the 10-minute film there is delayed excretion of contrast medium in the left kidney. *Right*, one-hour film shows left hydronephrosis and hydroureter, a filling defect on the left side of the bladder wall (representing tumor) and increased thickness of the vesical wall. The lower end of the ureter is obstructed by the tumor.

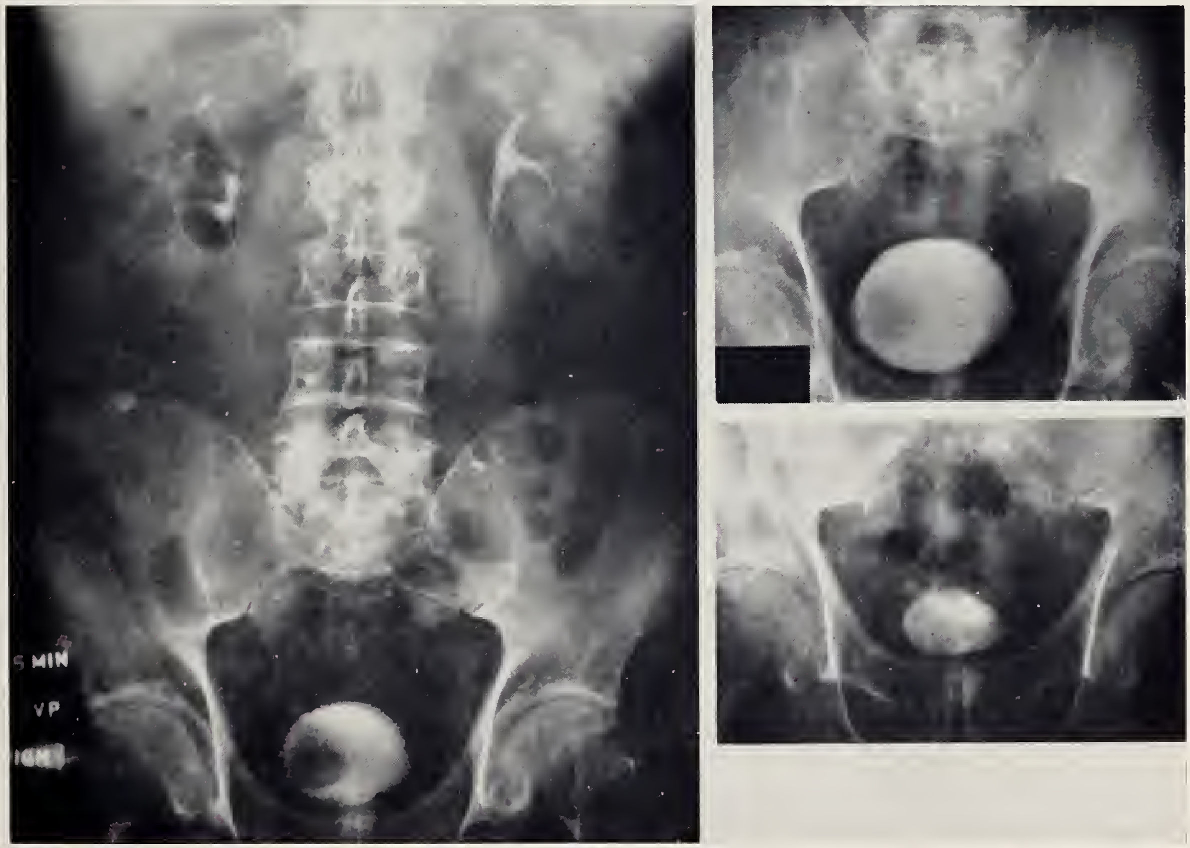


Figure 1.—Double-dose excretory urogram of a 60-year-old man with hematuria. *Left*, 15-minute film shows a filling defect on the right; *upper right*, one-hour film reveals persistence of the filling defect, later proved caused by a bladder tumor. *Lower right*, postvoiding film demonstrates the bladder tumor and a diverticulum in the right side of the bladder wall.

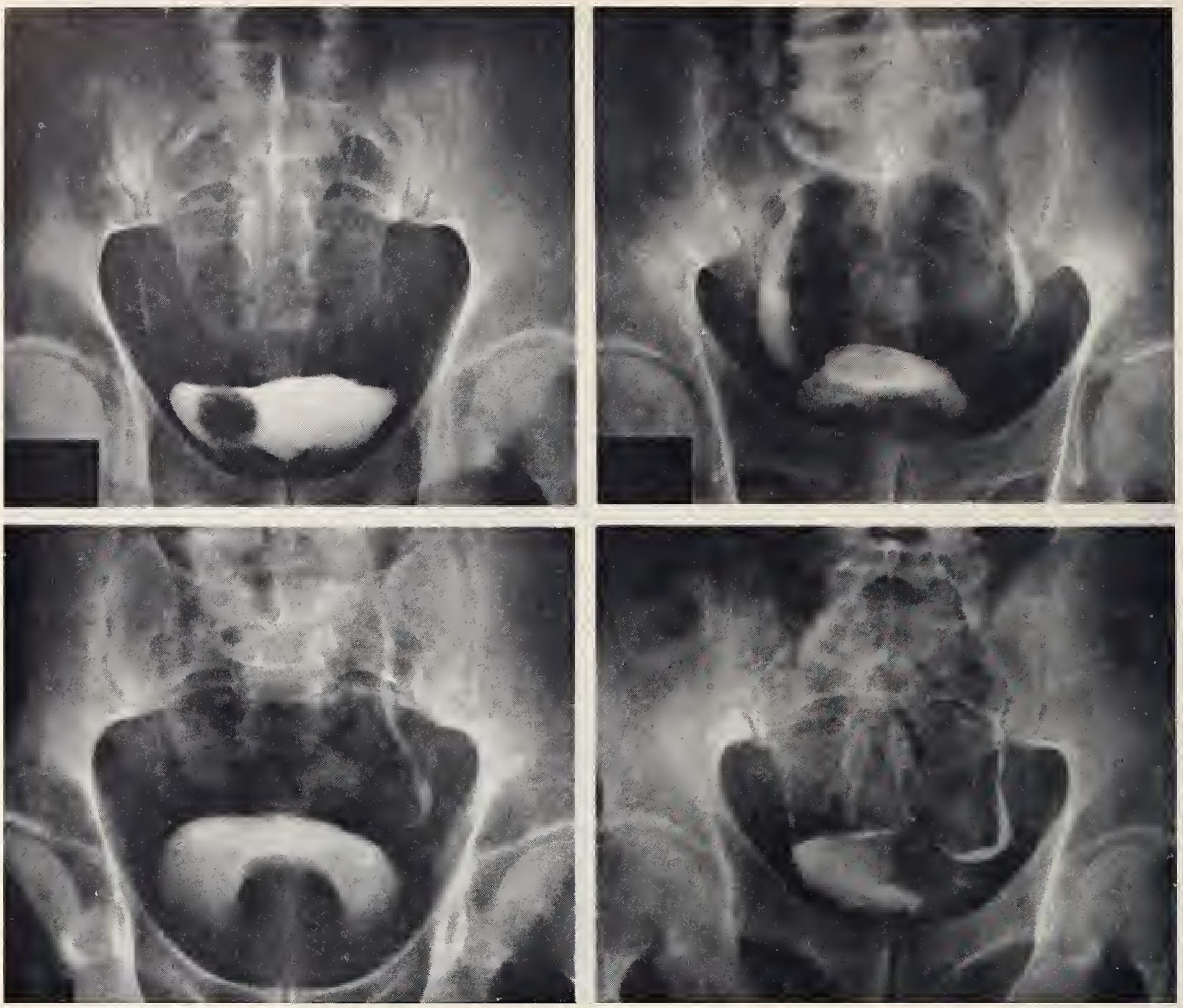


Figure 3.—Some points in differential urographic diagnosis. *Upper left,* bladder tumor. May occur in any part of the bladder; usually above the trigone, but sometimes involves it, and occasionally obstructs the ureter. Vesical wall thickness may be increased. *Upper right,* radiolucent bladder calculi. Usually float on surface of the urine, hence appear above the trigone on recumbent film, but often lie on the trigone in the postvoiding upright view. *Lower left,* benign prostatic hypertrophy. Always below the ureteral openings; usually in the midline. *Lower right,* prostatic abscess. Attached to the prostate, close to the trigone, below the ureteral orifices, usually unilateral.

gram by a filling defect, by increased thickness of the bladder wall, or both, with or without ureteral obstruction (Figures 1 and 2). In three patients excretory cystography did not adequately define the tumor, either because the lesion was too small or because its location was such that it did not cause a filling defect.

In five of the 23 patients, symptoms of other urinary tract disease had prompted excretory urography: In two, a urographic diagnosis of ureteral stone was proved by passage of the stone followed by relief of obstruction; in three, bladder neck obstruction was due to benign prostatic hypertrophy and until urography there was no indication of the coexistence of a bladder tumor.

Discussion

The high proportion of accurate precystoscopic diagnosis obtained by means of double-dose excretory urography is noteworthy in view of the lower percentages reported by careful workers using well-planned techniques with the standard-dose of contrast medium. Thus, only 260 (36.8 per cent) of the 737 bladder tumors reported by Braband[3] caused a filling defect. Smith,[5] reviewing 137 bladder tumors, noted that only 58 per cent of carcinomas and 33 per cent of papillomas had caused a filling defect on the standard-dose cystogram.

All urographic methods are supplementary diagnostic techniques and they do not replace cystoscopic examination and other time-tested diagnostic methods; but the double-dose of contrast medium does increase the predictive value of a complementary procedure long relied upon. Excretory urography is essential to the investigation of most urologic disease because of the information it yields regarding the morphologic features and function of the total urinary tract; in addition, it is safer than retrograde study. The simple modification suggested here is worthy of consideration because it provides distinctly superior visualization of the total urinary tract without increased inconvenience or risk.

The type of tumor is an important factor affecting the probability of demonstration by any urographic method. The majority of bladder tumors are papillary transitional cell epitheliomas. Tumors of this type are usually exophytic; infiltration of the bladder wall is infrequent and is not likely to be extensive; ureteral obstruction is seldom encountered. Because such a tumor protrudes into the bladder lumen, it may well cause a filling defect on the urogram, but if it is less than 1 cm in diameter, even a papillary tumor usually escapes detection by all roentgenographic methods. A large proportion of infiltrating tumors cause increased thickness of the bladder wall, visible urographically as widening of the distance between the edge of the opacity representing intraluminal contrast medium and the perivesical fat (Figure 2). I have often observed that a double-dose of contrast medium produces a clearer definition of the vesical wall—comparable with that obtained in retrograde cystography—and thus yields a more accurate estimate of bladder wall thickness than could be achieved with the standard-dose.

As Wesselhoeft and his colleagues[6] noted, cystography—whether excretory or retrograde—is of impressive value in demonstrating tumors inaccessible to endoscopic detection because they are within bladder diverticula. In one of the cases I observed, the double-dose modification was especially useful in detecting such a lesion.

This method gives excellent information about associated or alternative lesions that must be differentiated from bladder tumors (Figure 3), including lower ureteral obstruction due to calculus, tumor or ureterocele; bladder diverticula; radiolucent calculi; intravesical blood clots or foreign bodies; bullous edema of the bladder; bladder neck obstruction; prostatic hyperplasia, cyst or abscess. Lesions of other pelvic organs that compress or displace the bladder or lower ureters must at times be differentiated from bladder tumors and are usually delineated more clearly with the double-dose than with the standard-dose of contrast medium.

In view of our experience, we recommend double-dose excretory urography as a primary procedure in all patients with gross or microscopic hematuria, to investigate the possibility that a bladder tumor may be its cause, to provide necessary evaluation of the total urinary tract and to facilitate adequate planning for treatment if a neoplasm is found. After treatment for bladder tumor, double-dose excretory urography with delayed films is very helpful in the detection of recurrence, of ureteral obstruction by a neoplasm or of extension of neoplastic growth into the bladder wall and beyond. It is also useful in the assessment of the status of the kidneys and ureters.

REFERENCES

1. Amar, A. D.: Double-dose of contrast medium in excretory urography, Surg., Gynec. & Obst., 118:1083-1087, May 1964.

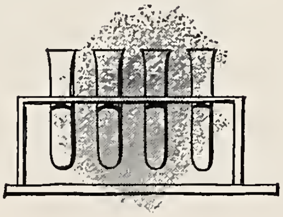
2. Amar, A. D.: Improved excretory cystograms with double-dose of contrast medium, Urol. Digest, 4:21-29, September 1965.

3. Braband, H.: The incidence of urographic findings in tumors of the urinary bladder, Brit. J. Radiol., 34: 625-629, October 1961.

4. Elkin, M.: Supine and prone positions in intravenous urography for diagnosis of bladder lesions, Radiology, 78:904-913, June 1962.

5. Smith, H. W.: The reliability of excretion urography in the diagnosis of bladder tumor, J. Fac. Radiologists, 6:49-54, July 1954.

6. Wesselhoeft, C. W., Jr., Perlmutter, A. D., Berg, S., Unger, L., Gherardi, G. J., Harrison, J. H., and Clarke, B. G.: Pathogenesis and surgical treatment of diverticulum of the urinary bladder, Surg., Gynec. & Obst., 116: 719-725, June 1963.



CASE REPORTS

Cure of Intracranial Aneurysm Without Use of Blood Transfusion

JACK POSNIKOFF, M.D., *Orange*

THERE ARE PEOPLE still present in our society who are willing to accept death rather than violate religious principles. To the surgeon this presents a most distressing professional and medico-legal frustration when blood transfusion is considered and the patient refuses because of religious tenets.

In the case of minors the law aids the surgeon in circumventing this restriction. An adult, however, is generally legally able to decline the use of blood in his own therapy.

Under circumstances of refusal by adults before elective operation, the surgeon may reject the case entirely or he may accept the restrictions, promising to adhere to them at operation regardless of the consequences. In the dire circumstances of mandatory blood transfusion and the latter alternative, the surgeon would be faced with distressing conflict between legal impediments and ethical responsibility.

In the case presented herein decision to carry out operation despite absolute rejection of transfusion of blood or blood components was made only after extensive deliberation. The necessity for more extensive sociological and medico-legal study and definitive assistance in this regard was evident.

The purpose of the presentation is to emphasize features which indicated that the surgical hazards were less than they were thought to be and to interest medical societies in exploring even further the medical and legal ramifications of this fortunately rare but taxing religious encumbrance.

Report of a Case

A 41-year-old woman had been examined by a number of experienced neurosurgeons who recommended operation but refused to operate because of the patient's steadfast rejection of blood transfusion in any circumstances.

Several years previously she had begun to have episodes of nausea and dizziness associated with transient periods of "greying" of vision. Soon thereafter she began to have transient numbness of the left extremities and occasionally of the left facial region. Concomitantly she had feelings of fullness in the head and faintness. In the year before admission to hospital, the incidence of these episodes increased to six to seven times weekly. In addition, she had been having twitching of the left extremities and occasionally of the face for about a year, and often at night she wakened suddenly in fright. During the few months before admission there was an increase in the sensation of fullness of the head associated with generalized headache and discomfort over the right side of the cranium. In the three weeks before she entered the hospital she had become increasingly more lethargic and headache increased.

Inquiry disclosed a tendency to left handedness in the patient, and three of her children showed mixed dominance. She had had a minor head injury without loss of consciousness 14 years previously.

From the Department of Neurosurgery, Orange County General Hospital, Orange.

Submitted 7 July 1966.

Reprint requests to: Department of Neurosurgery, Orange County General Hospital, 101 Manchester Avenue, Orange 92668.

On physical examination, moderate lethargy was noted, with no dysphasia. There were no tremors or twitching. Carotid pulsations were equal without cranial or cervical bruits and there was no papilledema. There was slight left lower facial weakness with definite incoordination of the left foot and slight incoordination of the left hand. This was accompanied by moderate dorsiflexion weakness of the left foot but with preservation of heel walking. Slight weakness of left hand grip was noted. Hypesthesia was present and involved the entire left side but was most pronounced in the foot. Objectively there was no gross error in two point discrimination but the patient said she noted some difficulty on the left side. The left biceps jerk was more active than the right and there was a definite Babinski sign on the left.

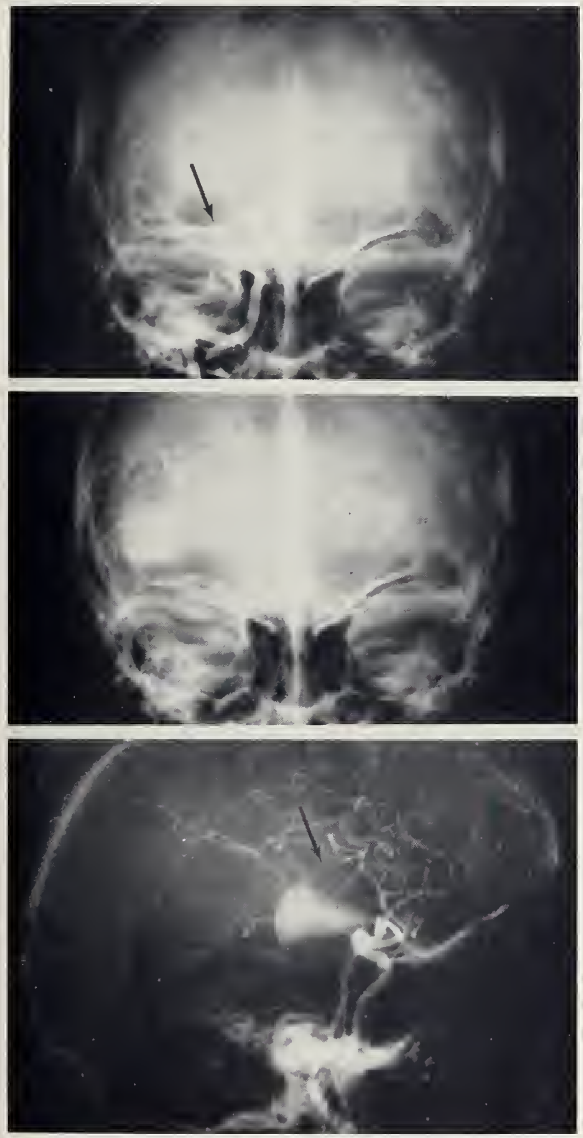


Figure 1.—Arteriogram: *At top,* film in an early arterial phase reveals a sessile trifurcation area aneurysm with bowing of the middle cerebral branches and bloc displacement of the anterior cerebral complex. *Middle,* a late venous phase demonstrates protracted filling of the large aneurysm which acted as a mass lesion. There is displacement of the internal cerebral vein to the left. *Bottom,* there is upward displacement of the middle cerebral artery complex. The pseudopod aneurysmal projection and the discrete linear calcification were considered indicative of intraluminal clotting in an aneurysm more extensive than depicted by the contrast material.

Plain x-ray films of the skull showed linear calcification in the right central sylvian area. An angiographic study demonstrated two aneurysms involving the right middle cerebral artery complex. A sessile aneurysm 0.5 cm in diameter was situated in the trifurcation region and a much larger aneurysm was present distal to the trifurcation in the sylvian area that produced a mass effect with bowing of the middle cerebral branches and upward displacement of the sylvian vascular complex (Figure 1). In addition there was displacement of the anterior cerebral artery complex and of the internal cerebral vein to the left side. The large aneurysm did not fill in the earliest arterial period but showed tardy filling extending well into the venous phase (Figure 2). Exact anatomical delineation of the vessel supplying the area was difficult but the aneurysm was definitely originating from one of the branches distal to the trifurcation. The aneurysm was irregular in outline and did not extend as far as the calcifications mentioned previously. This indicated a larger aneurysm partially filled with clot, while the tardiness in filling was in keeping with origin in a branch rather than a main vessel.

An electroencephalogram was diffusely abnormal and demonstrated a mild degree of slowing with some right hemisphere predominance. There were no discrete focal abnormalities.

Operation was carried out in two stages. The first operation was under local anesthesia and sedation with elevation of a large free temporocentral bone flap. The dura was not opened at this time. The bone was rewired into position and the soft tissues closed. Four days later, after normal convalescence with supportive anticonvulsants, a second operation was done with general anesthesia and again without blood transfusion. The craniotomy was performed with minimum loss of blood. The moderately tense dura was opened and the temporal lobe resected back 6 centimeters. This produced adequate exposure of the inferior aspect of the large aneurysm. The medially situated feeding vessel, a branch of the middle cerebral, was identified, clipped and divided. Two efferent smaller vessels were identified more posteriorly and were dealt with similarly. The aneurysm was then gently retracted from its compressed bed. This allowed visualization of the more medial aspect of the cerebral branches as well as the trifurcation area where the small sessile aneurysm that had been seen radiographically was observed. It was not disturbed for fear of inducing vascular spasm that might complicate a potential ischemic situation based on the sacrifice of a major sylvian branch. The bed of cerebral tissue at the site of the large aneurysm showed yellowish and brownish discoloration in keeping with probable previous bleeding. After hemostasis, bone and soft-tissue closure was carried out. The dimensions of the dissected and shrunken aneurysm are shown in Figure 2.

The immediate postoperative course was uneventful, with rapid remission of objective physical findings. Several weeks after discharge, however, the patient began to have increasing incidence of episodes of lightheadedness and aberrant behavior associated with hyperactivity. Electroencephalography demonstrated paroxysmal sharp wave and theta activity over the right frontal, central and parietal regions, accompanied by repetitive low to moderate voltage spike discharges in the right posterior temporal and right central areas. The substitution of mysoline medication for dilantin and phenobarbital resulted in resumption of normal behavior and amelioration of the lightheadedness. Postoperative angiograms demonstrated again the trifurcation sessile aneurysm but with complete absence of the original large aneurysm (Figure 3

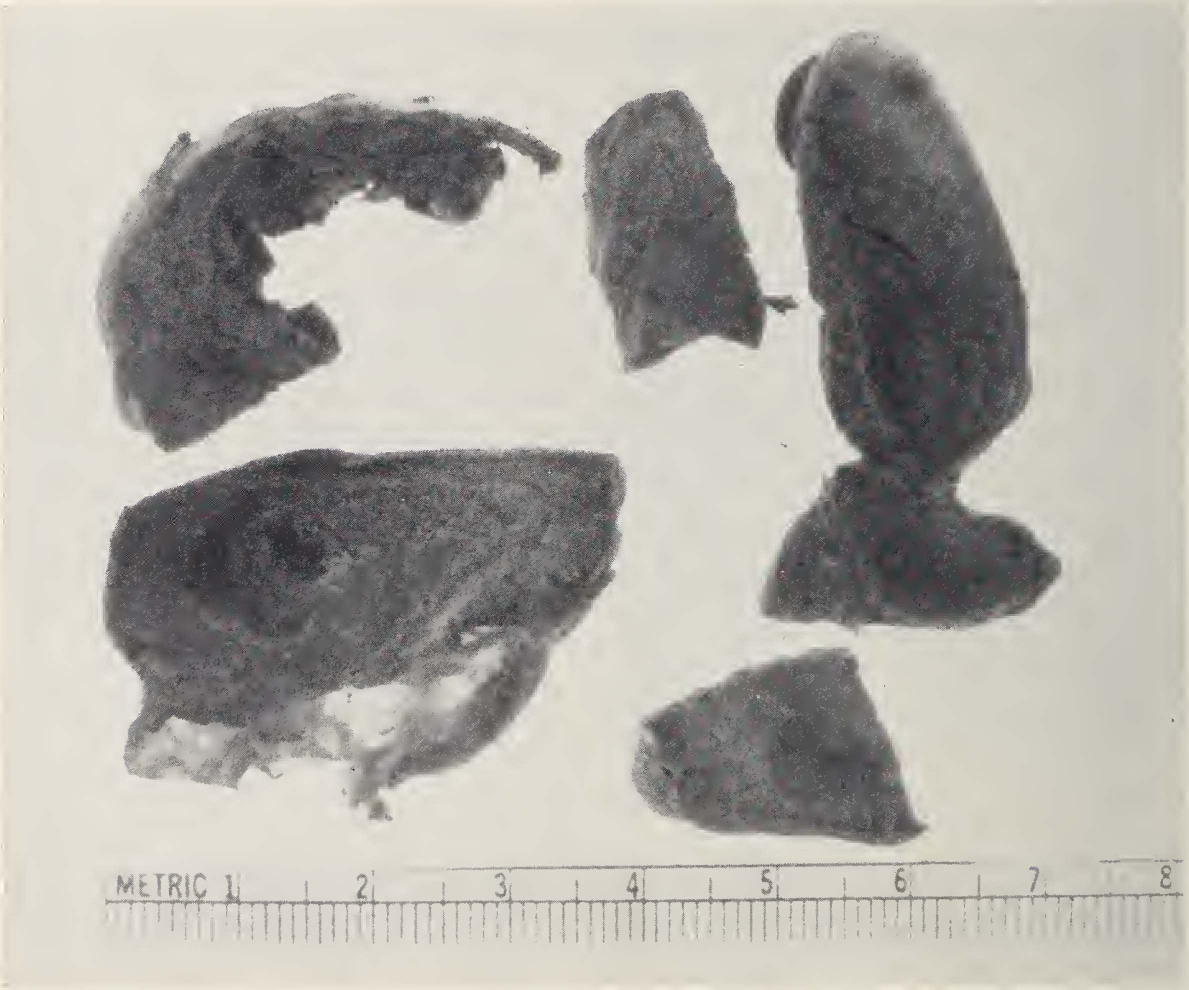


Figure 2.—In spite of the pathological dissection the dimensions of the now shrunken aneurysm are still evident. Partially organized clot occupies a considerable portion of the lumen.

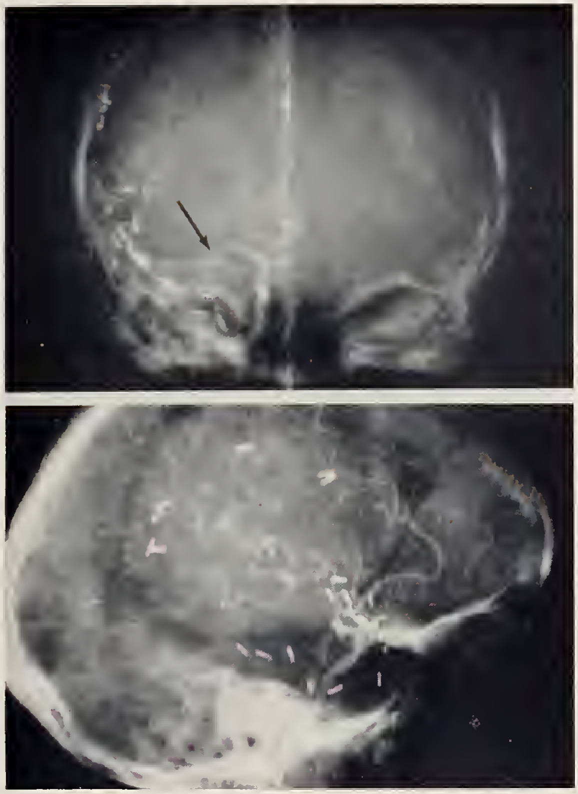


Figure 3.—Postoperative arteriograms. *At top,* a late arterial, early arteriolar phase demonstrates return of the anterior cerebral complex to the midline with absence of the large aneurysm but persistence of the trifurcation sessile dilatation. *Below,* the large aneurysm is no longer present and the middle cerebral artery complex is more normally situated.

a and b). The anterior cerebral complex and internal cerebral vein were back in normal position. Recommendation of another craniotomy for plastic reinforcement of the trifurcation region was rejected.

Discussion

The indication for operation was therapeutic removal of a mass lesion and possible elimination of potential hemorrhage. The two-stage program was carried out in order to preserve blood at the second operation when aneurysmal isolation might have resulted in extensive blood loss. On the other hand, it was considered that the tardy filling of the large aneurysm, along with evidence of intrasaccular clot, was indicative that the lesion was in a minor vessel rather than the middle cerebral artery. As a consequence it was the author's opinion that danger of uncontrollable hemorrhage was unlikely. Although there was mixed dominance it was felt that actual sacrifice of tissue would be at a minimum following the temporal lobectomy. This proved to be the case and there was no postoperative dysphasia or motor dysfunction.

It is the current opinion of most neurosurgeons that transfusion of blood is absolutely essential in operations for intracranial aneurysms. This case demonstrates, however, that each aneurysm should be considered individually. It therefore behooves us not to routinely deny major operation to those who may be in desperate need but cannot morally accept blood transfusion.

Summary

A case of successful excision of an extremely large intracranial aneurysm was carried out without blood transfusion, which the patient forbade on religious grounds. The case reemphasizes the two-stage technique often used by earlier neurosurgeons who lacked the blood bank facilities of today and calls attention to a need for further sociological and medicolegal exploration of the question of religious restriction of indicated therapeutic procedures.

Insidious Splenic Rupture Due To Infectious Mononucleosis Presenting as Acute Glomerulonephritis

MURRAY SHEVICK, M.D., ROBERT ZWEIG, M.D.
AND LAWRENCE BRASLOW, M.D., *Arlington*

THE BIZARRE MANIFESTATIONS of infectious mononucleosis present a particular problem in differential diagnosis of acute illness in young persons. The incidence of ruptured spleen has been reported as from 0.5 per cent[5] to 0.1 per cent[14] in treated infectious mononucleosis. The mortality rate associated with this complication is reported between 30 per cent[13] and 55 per cent.[10] While hepatitis,[3] encephalitis,[8] splenomegaly and neuritis[2] are widely

---

From the Department of Medicine and Surgery, Parkview Memorial Hospital, Arlington.

Submitted 13 May 1966.

Reprint requests to: 3838 Jackson Street, Arlington 92503 (Dr. Shevick).

known sequelae of mononucleosis, renal involvement or clinical suspicion of renal involvement is rare.[1,7,9,11]

Report of a Case

The patient, a 29-year-old white student, noted malaise, fatigue and flatulence about three weeks before admission. He began running as an exercise to improve his physical condition. On questioning, he could think of no physical trauma. His left side and back began to ache about the time that he felt feverish, and sore throat developed. One week before admission, puffiness was noted about the face. Oral temperature at that time was 101°F (38.3°C) and a physician found him to have pharyngitis with cervical adenitis. Penicillin was administered. Pleuritic pain low in the left side of the chest was not lessened by a pleurisy belt. The spleen was not palpable.

The following day the patient became nauseated and was relieved by self-induced vomiting. Pallor was noted by the physician, and results of laboratory studies were: Hemoglobin 12.0 gm per 100 ml; leukocytes 7,000 per cu mm with cell differential within normal range; negative heterophile; urinalysis, specific gravity of 1.032, with no albumin or bile. Thereafter, the patient noticed scanty, dark urine in spite of increased fluid intake. Two days before admission to hospital his lips appeared very pale and the skin seemed to have a sallow tint. A rectal suppository was inserted and mild diarrhea ensued. The patient consulted another physician, who noted periorbital edema, pallor and edema of the hands and face. Hemoglobin at that time was 9.7 gm per 100 ml; and on urinalysis there was a 2-plus reaction for albumin. The urine contained 10 to 15 erythrocytes, eight leukocytes and many cellular casts per field. The patient was put in hospital with initial impression of acute glomerulonephritis.

On admission the patient was edematous, pale, sallow and lethargic, yet responsive. The temperature was 38.0°C (100.4°F), the pulse rate 100 and blood pressure 110/50 mm of mercury. A minimal erythematous rash was noted over the hard and soft palate. There was no adenopathy. A few fine crackling rales were audible at both lung bases. Sinus tachycardia (130 beats per minute) with a gallop rhythm and a faint apical ejection systolic murmur were noted. The abdomen was slightly protuberant but organs were not palpable. There was generalized muscle guarding and diffuse minimal tenderness throughout the abdomen. Bowel sounds were diminished. No abnormalities were noted on rectal examination. There was no pitting edema of the legs, although there was edema of the face and hands. Hemoglobin was 9.7 gm per 100 ml on admission, and next day was 7.0 gm. Reticulocytes made up 7.6 per cent of the erythrocytes and there were 6,800 leukocytes per cu mm —4 per cent stab cells, 45 per cent segmented forms and 51 per cent lymphocytes, many of them abnormal-appearing. Febrile agglutinins were absent and heterophile titer was 1:28. Urinalysis showed specific gravity of 1.021 with a trace of albumin. On microscopic examination of the urine there were four to eight white cells and 20 to 25 red cells per high dry field, with 12 to 18 coarsely granular and white cell casts per low power field, and amorphous crystals. Blood urea nitrogen was within normal limits. The serum protein was 6 gm per 100 ml with normal ratio of albumin to globulin. A culture of material from the throat showed normal flora, and antistreptolysin. A titer of the blood was 50 Todd units. No abnormalities were noted in x-ray studies of the chest and abdomen. A gastrographin survey of the upper gastrointestinal tract showed medial shift of the gastric fundus, suggesting a moderate enlargement of the spleen or pseudocyst of the pancreas. Equivocal medial displacement of the left ureter was noted on an intravenous pyelogram.

The following day, the abdomen became decidedly distended and was tense with muscle guarding, but there was no rebound tenderness. Bowel sounds were diminished. Maximal tenderness was noted along both lower sides of the abdomen, and on palpation there seemed to be a mass in the left upper quadrant. After four units of blood had raised the hemoglobin to 10.7 gm per 100 ml, abdominal paracentesis was performed and blood that did not clot was withdrawn from the peritoneal space.

At laparotomy the spleen was found to be three times enlarged, with a laceration across the midportion. It weighed 650 grams. Blood with masses of clots—1,950 ml in all—were removed from the abdominal cavity. The patient received a total of six units of blood. Four days after operation the pathologist reported abnormal lymphocytes in the spleen, suggesting infectious mononucleosis.

The night he was admitted, the patient had received intermittent intravenous tetracycline and the temperature had decreased to normal the day the diagnostic procedures were performed. Following operation the rectal temperature spiked to 39.4°C (103°F). Bone marrow aspirate and blood for culture and malarial smears was obtained and the administration of penicillin, streptomycin and chloramphenicol was begun. The temperature fell rapidly to normal in 24 hours, but rose again a day later to 38.9°C (102°F). Again the blood cultures were negative for pathogenic organisms. Thereafter, the temperature subsided to normal with only symptomatic therapy. Symptomatically, the patient was dramatically improved following operation and had little if any subjective discomfort during the postoperative febrile course.

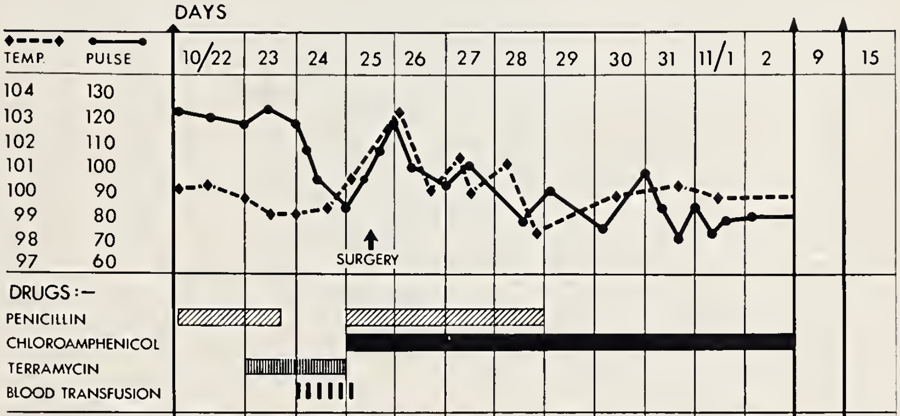
A heterophile test was reported positive at 1:448 with a differential absorption test positive at 1:225. A test for mononucleosis[6] was also positive. Results of bone marrow examination, blood cultures and malarial smears were reported negative. Pertinent laboratory data are shown in Chart 1.

The patient made an uneventful recovery without complication and returned to his studies.

Discussion

The patient's condition when he was examined by the first physician was certainly that of a person with pharyngitis and mild pleurisy, and there was no indication of any serious complication. The second physician, seeing him as pale, edematous and febrile with a history of sore throat (probably streptococcal), dark, scanty urine with hematuria, proteinuria and anemia, suspected acute glomerulonephritis.

The third physician excluded acute glomerulonephritis when he saw the patient with hypotension and blood urea nitrogen within normal limits. The progressive unexplained anemia, vomiting, abdominal distention and diarrhea without roentegenographic evidence of intestinal involvement suggested acute hemorrhagic pancreatitis. Abdominal paracentesis of blood which did not clot led to a preoperative diagnosis of spontaneous ruptured spleen, due to either lymphoma or infectious mononucleosis, even though the atypical lymphocytes were not classic Downey cells.



	10/22	23	24	25	26	27	28	29	30	31	11/1	2	9	15
LABORATORY :—														
HEMOGLOBIN	12.7	9.7	9.1	7.4	10.7	11.2		10.3	10.7		11.2			14.2
W.B.C.	7.1	8.8	16.1	9.6	17.8	16.2		19.4	21.8		10.5			7.9
LYMPHOCYTES — %	43	51	37	55	33	58		58	69		77			70
" % ABNORMAL		90%		90%	75%	90%		90%						1:56
HETEROPHILE				1:28		1:448		1:448				1:224		
URINALYSIS :—														
PROTEIN	+++	++	TR	TR	+		0					TR	+	
RBC	0	10	20-25	25	MANY		70					OCC	OCC	
WBC	20	15	4-8	8	OCC		8					5	0	
CASTS	4	8	12-18	8			0					0	0	
SGOT								128						
MONO TEST								+++						
METHYLENE. BLUE DYE TEST								0						
CREATININE CLEARANCE TEST											81.6 ml./min.			

Chart 1.—Laboratory and clinical data in case of patient with splenic rupture caused by infectious mononucleosis.

In retrospect, his pallor, polydipsia, weakness, urinary findings and even fluid retention may have been owing to chronic but profound hemorrhagic shock which bore an amazing similarity to acute glomerulonephritis.[7] The insidious nature of the splenic rupture, the slowly progressing anemia over a six-day period, the absence of jaundice, hepatomegaly or adenopathy or of early absolute lymphocytosis or positive heterophile, made early diagnosis difficult.

Since rupture of the spleen usually occurs within the first three weeks of illness[12] and the heterophile titer peaks between the third and fourth week, nondiagnostic heterophile titers may be encountered in spontaneous splenic rupture.

As stated by Smith and Custer,[10] splenic rupture may be followed by a reversal of the leukocyte count from relative lymphocytosis to neutrophilic leukocytosis. In the present case an intermediate response was demonstrated.

Freeman[4] stressed that the fever accompanying the postoperative course in cases of splenic rupture associated with mononucleosis might not be from superimposed infection or surgical complication, but from mononucleosis itself.

Although the patient was in a typical age group and setting for this disease, this case is unique in several respects. The complication of splenic rupture, which occurs in perhaps one per thousand cases, was so insidious as to escape the attention of two physicians. The first symptoms were like those of a completely unrelated disease, acute glomerulonephritis. The abnormal urinary findings may have been due to the decreased renal blood flow of hemorrhagic shock, yet could also represent primary involvement of the kidney with infectious mononucleosis. Rapid recovery of the urinary sediment in infectious mononucleosis suggested a point of differential diagnosis from acute glomerulonephritis in a case reported by Lindsey.[7] However, only by throat culture and antistreptolysin titer can a simultaneous streptococcal infection with infectious mononucleosis be established.

The postoperative fever was due to infectious mononucleosis, proved only after intensive investigation including negative blood cultures, bone marrow aspiration and culture, malarial smears and benign clinical course. Vigorous antibiotic therapy was probably unwarranted.

REFERENCES

1. Castleman, B., and McNeely, B. U.: Weekly clinicopathological exercise, New Engl. J. Med., 272:37-41, 7 January 1965.
2. Chang, P., and Wurm, M.: Infectious mononucleosis—With high heterophile and neurological manifestations, Calif. Med., 103:435-438, December 1965.
3. Erwin, W., Weber, R. W., and Manning, R. T.: Complications of infectious mononucleosis, Amer. J. Med. Sci., 238:699-711, December 1959.
4. Freeman, A. R. M.: Rupture of spleen in infectious mononucleosis, Bin. J., Vol. 2, 14 July 1962.
5. Hoagland, R. M.: The clinical manifestations of infectious mononucleosis: A report of two hundred cases, Amer. J. Med. Sci., 240:21-29, July 1960.
6. Hoff, G., and Bauer, S.: A new rapid slide test for infectious mononucleosis, J.A.M.A., 194:351-353, 25 October 1965.
7. Lindsey, D. C., and Chrisman, W. P., Jr.: Gross hematuria as the presenting symptom of infectious mononucleosis, J.A.M.A., 157:1406, 16 April 1955.
8. Nichols, W. W., and Athreya, B.: Encephalitis probably due to infectious mononucleosis, Am. J. Dis. Child., 103:104-108, February 1962.
9. Schumacher, H. R., and Barcay, S. J.: Hemorrhagic phenomena in infectious mononucleosis, Amer. J. Med. Sci., 243:175-182, January-June 1962.
10. Smith, E. B., and Custer, R. P.: Rupture of the spleen in infectious mononucleosis: A clinico-pathologic report of seven cases, Blood, 1:317, July 1946.
11. Taub, E. A.: Renal lesions, gross hematuria, and marrow granulomas in infectious mononucleosis, J.A.M.A., 195:175-177, 28 March 1966.
12. Walton, T. T., Jr.: Splenic rupture in infectious mononucleosis, Texas State J. Med., 59:333, April 1963.
13. Wetherill, J. H., and Oldfield, M. C.: Rupture of the spleen and thrombocytopenia in glandular fever, The Lancet UIC, 1:636, 23 March 1963.
14. York, W. H.: Spontaneous rupture of the spleen: Report of a case secondary to infectious mononucleosis, J.A.M.A., 179:178-179, 13 January 1962.

- It has been possible to hold the subscription rates without any increase during the 12-year period, added costs and expenses having been absorbed by the continuously increasing volume.

- 1966 was the year of greatest growth—16 per cent over the preceding year.

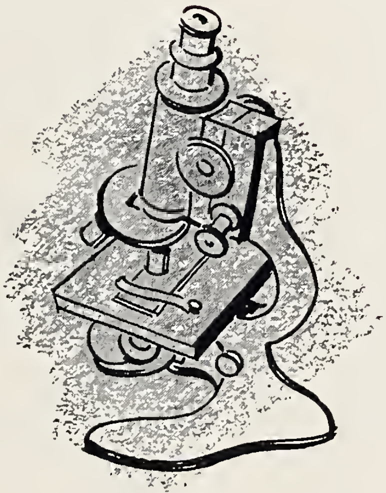
- The annual subscription renewal rate is high and getting higher—77 per cent in at the time of report, against 68 per cent a year earlier.

- The number of subscribers now is about 13,000; and, since often the tapes are listened to by groups—hospital staffs, discussion clubs, military personnel particularly in outpost service—the total number of listeners is estimated at 50,000.

- Contributions to medical education in 1966 amounted to $110,000—$55,000 each to the American Medical Education and Research Foundation and the California Medical Education and Research Foundation. In all, Audio-Digest has contributed $309,750 to medical education.

These accomplishments in the thoughtfully planned, dynamically prosecuted growth of Audio-Digest are pleasant to contemplate: Medical education is broadened as the number of subscribers increases, and the more subscribers the more revenues, and the more revenues the greater the cash contributions in support of medical education.

Lo! A benign cycle.



Council Meeting Minutes

Tentative Draft: Minutes of the 526th Meeting of the Council, San Francisco, Hilton Inn, 22 November 1966.

The meeting was called to order by Chairman Anderson at the Hilton Inn near San Francisco on Tuesday, 22 November 1966, at 7:00 p.m.

A quorum present and acting (full roll call, including names of invited guests, appears at the end of these minutes).

Purpose of this special meeting of the Council, as described by Doctor Anderson, pertained to the implications of changes in the legislative and executive branches of State government resulting from the election of 8 November 1966.

After due consideration of many aspects of the change-over in State government, the Council:

***ACTION:* Voted to support the concept of a single administrative agency for the Medi-Cal program in California, namely the Health and Welfare Agency.**

***ACTION:* Voted approval of a joint search committee composed of members of the California Medical Association and the California Hospital Association. Purposes of the search committee include development of criteria for positions in the executive branch in which medicine and hospitals may have an interest, development of a list of suggested nominees for these various positions and contacts to be made with the new State administration. (Included on the joint search committee from CMA are: President MacLaggan, President-Elect Morrison, Legal Counsel Hassard, and Mr. Ben Read of the Public Health League. From the California Hospital Association: President Jackson, President-Elect Cumming, Mr. Emery Dowell, and Legal Counsel Ludlam.) Consultation from CMA Councilors and other interested persons will be invited by the search committee.**

***ACTION:* Voted for the search committee to use a joint CMA-CHA letterhead as the need arises, provided the letters are signed by the presidents of both organizations.**

***ACTION:* Voted that the Council express officially its willingness to assist the new administration of Governor-Elect Reagan on matters of health care.**

***ACTION:* Voted to offer to the California Health and Welfare Agency the good offices of the CMA in trying to correct abuses in nursing home-boarding home utilization.**

Roll Call

Present were President MacLaggan, President-Elect Morrison, Speaker Quinn, Vice-Speaker Telford, Secretary Weyrauch, and Councilors Isen-

JAMES C. MacLAGGAN, M.D.	President
JOHN G. MORRISON, M.D.	President-Elect
WILLIAM F. QUINN, M.D.	Speaker
JOSEPH W. TELFORD, M.D.	Vice-Speaker
CARL E. ANDERSON, M.D.	Chairman of the Council
ALBERT G. MILLER, M.D.	Vice-Chairman of the Council
HELEN B. WEYRAUCH, M.D.	Secretary
DWIGHT L. WILBUR, M.D.	Editor
HOWARD HASSARD	Executive Director

General Office, 693 Sutter St., San Francisco 94102 • 415 776-9400

JACK CURLEY . . . . Southern California Office
1515 N. Vermont Ave., Los Angeles 90027 • 213 663-8071

RICHARD W. LEMOS . . . . Sacramento Office
1127 11th St., Sacramento 95814 • 916 444-7496

hour, Eastman, Todd, Gooel, Bullock, O'Connor, Ham, Rogers, Crum, Maguire, Burnett, Richard S. Wilbur, Miller, Watts, Fenlon, Kay, Kaiser, Anderson, Yant, Grunigen, and Immediate Past President Teall. Absent for cause were Editor Dwight L. Wilbur and Councilors Melone, Taw, Boyle and Longmire.

Present by invitation were CMA staff members Bowman, Clark, J. Collins, Curley, Eberlein, Goldman, Hetland, Lemos, Miller, Thomas and Whelan; Messrs. Hassard and Huber, legal counsel; Messrs. Read, Brown and Putnam of the Public Health League; and Doctors Kilroy and Sherman.

Adjournment

The meeting was adjourned at 10:15 p.m.

Carl E. Anderson, M.D., *Chairman*
Helen B. Weyrauch, M.D., *Secretary*

527th Meeting

Tentative Draft: Minutes of the 527th Meeting of the Council, San Francisco, Hyatt House, 10 December 1966.

The meeting was called to order by Chairman Anderson at the Hyatt House near San Francisco on Saturday, 10 December 1966, at 11:15 a.m.

A quorum present and acting (full roll call, including names of invited guests, appears in Item 38).

1. *Minutes for Approval*

The minutes of the 525th meeting of the Council, held 29 October 1966, were approved with the following changes:

(a) Addition of representatives of component societies which maintain medical libraries to the ad hoc Committee on Medical Libraries (Item 8).

(b) Correction of the name of a new Associate member—Thomas H. Hindle, III of Imperial County (Item 42).

The minutes of the 526th meeting of the Council, held 22 November 1966, were approved as submitted.

2. *Report of the President*

President MacLaggan informed the Council that the next meeting of the Health Manpower Council will be held the first weekend in February. He asked that a monetary contribution of $25,000 be made to the Health Manpower Council this year, with a commitment that, if necessary, the same amount be contributed for two more years.

ACTION: Voted to approve an appropriation of $25,000 to the Health Manpower Council for this year, with a commitment for a contribution of the same amount for two more years if necessary.

Doctor MacLaggan also reported on activities of the "joint search committee" composed of members of the CMA and the California Hospital Association. At the 22 November 1966 meeting of the Council, the joint committee was authorized to proceed in helping the new State administration in locating qualified persons to fill governmental posts in which medicine and hospitals may have an interest. Since that time, Doctor MacLaggan stated, the joint committee has contacted the four regional groups advising Governor-Elect Reagan on appointments and has sent a joint CMA-CHA letter offering assistance.

3. *Report of the President-Elect*

President-Elect Morrison stated that he had attended interim hearings on drug costs in Sacramento as the CMA representative, but had not found it necessary to testify. He said that the Committee on Committees had no report but would be meeting at an all-day session sometime in the near future.

4. *State Health and Welfare Agency*

Mr. Paul Ward, administrator of the Health and Welfare Agency, reviewed recent events involving the Medi-Cal Program. He spoke on two areas that have had major impact on the program: The unanticipated increase in utilization of nursing homes beds, and the Attorney General's interpretation of the Veneman amendment. There was discussion on certain portions of the regulations and their impending modification, with special emphasis on the definition of x-ray and laboratory services, the definition of "provider of service" as it is interpreted in the Federal and State laws, and the direct billing concept under Medi-Cal.

Mr. Ward thanked the Council for its cooperation. The Council, in turn, expressed its appreciation to Mr. Ward, giving him a standing ovation.

ACTION: Voted to commend Mr. Paul Ward for his outstanding efforts as administrator of the Health and Welfare Agency.

5. *State Department of Public Health*

In the absence of Doctor Lester Breslow, Doctor Robert Day, Deputy Director of the State Department of Public Health, reported to the Council. Doctor Day introduced members of his staff who were present, and gave a brief review of a newly issued report on communicable disease statistics for 1964-65. The report pointed out that the number of deaths remained quite constant at 1,200 per year, and half of these deaths were due to tuberculosis. However, Doctor Day stated that the rate of new cases of tuberculosis was decreasing. The report also revealed that there was some decrease in venereal disease in 1965, and that polio had shown a marked and rapid decrease.

6. *Stanford University School of Medicine*

Dean Glaser introduced Mr. Robert Lindee, assistant dean of administration at Stanford. He also informed the Council of a lecture series for the lay public sponsored by Stanford School of Medicine. The weekly series, called "Progress in Medicine," is open to all interested persons. Dean Glaser said that the attendance at the lectures has been over 200. It was also announced by Doctor Glaser that Doctor David Hogness, Professor of Biochemistry at Stanford, had been awarded the American Association for Advancement of Sciences Newcomb-Cleveland prize for the best presentation at the Association's meeting last year.

7. *University of California, San Diego, School of Medicine*

Acting Dean Robert Tschirgi reported to the Council that the Medical School at San Diego would open in the fall of 1968, and the first building was under construction. He also informed the Council that the medical school at San Diego had taken over the responsibility for operation of the San Diego County Hospital—now called the San Diego County University Hospital.

8. *University of California, Davis, School of Medicine*

Although Dean Tupper was unable to attend, Chairman Anderson reported that a letter had been received from Doctor Tupper announcing that Doctor Julian Youmans, currently Professor of Neurosurgery and Chairman of that Section at the University of South Carolina, would join the faculty at Davis on 1 July.

9. *UCLA School of Medicine*

Doctor John Dillon, recently appointed assistant dean in charge of professional society relations at UCLA School of Medicine, informed the Council that the medical school is expanding, adding seven stories to its hospital, and would admit 128 freshman medical students next fall.

10. *California Hospital Association*

Mr. Gordon Cumming, president-elect of the CHA, told the Council that the Association had recently employed Arthur Young and Company to carry out a cost study on hospitals to try to uncover some of the elements involved in the federal cost formula approach under the Medicare law. He also reported that CHA was seeking a new approach to the collection of data on a statewide basis that might be useful to both CHA and CMA. In addition, Mr. Cumming discussed some of the feelings of county government regarding the Veneman amendment.

11. *California Nurses Association*

Speaking for the Association, Mrs. Helen Hancock conveyed to the Council the interest of CNA in the Health Manpower Council and reported that at its recent Board meeting the California Nurses Association had appointed a steering committee to develop task forces on specific areas relating to health manpower. Mrs. Hancock also informed the Council that the Board had gone on record in support of the CMA bill on therapeutic abortion, to be introduced into the coming session of the legislature.

12. *California Medical Assistants Association*

In the absence of Mrs. Dorothy Fitzgerald, Mrs. June Florey, first vice-president of the CMAA, announced to the Council that the Association is working on its program for the CMA Annual Meeting. The program will focus on efficiency in office management, particularly in regard to the new physician entering into practice. Mrs. Florey stated that the CMAA favored the proposed merger of the CMA Liaison Committee to CMAA and the CMAA Advisory Committee.

13. *1967 Component Society Officers Conference*

Doctor Dale Ritter, chairman of the program committee for the 1967 Conference, reported to the Council that the format would consist of major presentations in the morning and afternoon, followed by discussion groups. He said that each society is invited to send five representatives to the meeting, which hopefully would include the society president, president-elect and a delegate.

14. *1967 Annual Session*

Speaker William Quinn informed the Council that at a planning meeting held the previous night, several new thoughts concerning the upcoming annual meeting had been presented. Among them were the suggestions that seconding speeches following nominations be limited to 30 seconds and that elections be placed later on the agenda. Doctor Quinn stated that these suggestions were under consideration.

15. *California Delegation to the AMA*

Chairman Davis reported that the CMA Delegation had been very successful in conveying CMA positions by means of five resolutions submitted for consideration at the Clinical Session in Las Vegas. Doctor Davis stated that the delegation continued its preparations for the campaign and election of Doctor Dwight L. Wilbur to the office of President-Elect of the AMA. A caucus of the delegation evoked the decision to select a candidate to fill Doctor Wilbur's unexpired term on the AMA Board of Trustees when he is elected to the AMA office. The candidate selected for nomination to the Board of Trustees was Doctor Burt L. Davis.

16. *Finance Committee*

Chairman Kay expressed the thanks of the Finance Committee to Mr. Lytton Hetland for his great assistance to the Committee in preparing the first draft of the 1967-68 CMA budget. He also asked Council direction on several suggestions for restructuring some CMA commissions and committees, and it was suggested that the recommendations of the Finance Committee be submitted to the Committee on Committees for review and recommendation. Doctor Kay asked the Council authorization for the Finance Committee to meet with representatives of the AMA delegation to analyze costs incurred by the delegation.

ACTION: Voted to authorize the Finance Committee to meet with representatives of the AMA delegation regarding delegation costs.

Doctor Kay also informed the Council that a proposed CMA budget for 1967-68 would be available to the Councilors at the January meeting.

17. *Cigarette Smoking and Health*

Doctor Longmire, Chairman of the Scientific Board, presented to the Council a position paper on smoking and health which had received Board approval. As amended by the Council, the report embodied the following: That CMA reaffirm the concept that cigarette smoking is harmful to health and that most physicians and their societies support this point of view; that the resources of CMA preclude massive public educational campaigns but the Association can support those agencies that do have such resources; that CMA reinforce the need for physicians to instruct and advise patients and others not to smoke and that the giving of such advice for the protection of health is a medical responsibility; that CMA should play a leading and active role in the Inter-Agency Council on Cigarette Smoking and Health; that CMA should assist in whatever ways are possible in research and local studies of more effective measures and that physicians convinced of the dangers of cigarette smoking set an example by abstention.

It was recommended that the report act as a guide to the CMA representatives on the Inter-Agency Council on Cigarette Smoking and Health in carrying out their activities.

ACTION: Voted to approve the CMA position paper on smoking and health as amended.

ACTION: Voted to approve a budgetary allocation of $3,000 to the California Inter-Agency Council on Cigarette Smoking and Health for next year.

18. *Aplastic Anemia Study*

Doctor Longmire also presented to the Council for approval a report on deaths from aplastic anemia associated with the use of the drug, chloramphenicol. The study, prepared for submission to the legislature, was carried out jointly by the State Department of Public Health, the California Pharmaceutical Association and the CMA.

It concluded that there is no need for: Special legislation to restrict the use of the drug, chloramphenicol, by licensed physicians in California; restriction of the use of the drug for in-hospital patients; special legislation requiring blood or bone marrow examinations when the drug is used; special legislation governing the refilling of prescriptions for this drug, to single it out from others; or a special identifying label. It further stated that the facilities now being developed to carry out studies on adverse drug reactions should be supported and that the study of aplastic anemia deaths be continued by CMA Committee on Adverse Drug Reactions.

ACTION: Voted to approve the report on aplastic anemia deaths for submission to the State Legislature.

19. *Role of the Health Educator Within CMA*

Doctor Gerald Jampolsky presented some ideas to the Council regarding the role of a health educator within the CMA organizational structure. A presentation also was given by Doctor Henry Bruyn of the University of California Student Health Service. Discussion followed on the activities involved in the health education functions and the role a health educator would play in the CMA staff organization.

ACTION: Voted to approve the area of health education as a reasonable function of the CMA.

ACTION: Voted to refer the implementation of the function of health education to the Finance Committee and the Executive Director for study and report to the Council at its next meeting.

20. *Tuberculosis Eradication*

Doctor H. Corwin Hinshaw, CMA representative on the Interagency Council on Tuberculosis, presented to the Council for its approval, a proposed resolution to be introduced concurrently into both the Senate and the Assembly in the coming state legislative session. There was discussion of the proposal, and its was recommended that, consistent with its policy regarding proposed legislation, CMA approve the proposal in principle and refer it to the Legislative Committee with the request that it be reviewed and a report given to the Council at the next meeting.

ACTION: Voted that CMA go on record as being in favor of taking "strong affirmative steps" to eradicate tuberculosis and to refer the specific proposals to the Legislative Committee with the request that the Committee review them and report back to the Council at the next meeting.

21. *Asilomar Conference on Nursing*

Doctor Gerald Scarborough presented to the Council a summary of recommendations from the Asilomar Conference on the Shortage of Nursing Personnel in California, which emphasized the need for better utilization of nursing service; realignment of nursing education; increased and more efficient production of nursing personnel; recruitmet, retention and functioning of nurse manpower; continuation of the committee that produced these recommendations and the formation of a voluntary group to carry out the recommendations of the conference.

It was reported that the Commission on Allied Health Professions and Services concurred with the recommendations of the Committee on Other Professions, and Council approval of the report was requested.

ACTION: Voted to approve the recommendations of the Asilomar Conference as presented by the Committee on Other Professions for use as a guide to CMA representatives on the newly-formed Health Manpower Council.

22. *Joint Statement on Intravenous Administration of Fluids*

Doctor Scarborough presented an amended joint statement (California Hospital Association, California Nurses Association and CMA) regarding the intravenous administration of fluids by registered nurses licensed to practice in California. The statement, as amended to include extended care facilities, was reviewed.

ACTION: Voted to approve as amended the joint statement on the intravenous administration of fluids by nurses licensed to practice in California.

23. *Resolution No. 19-66, Utilization of Paramedical Personnel*

Chairman Grunigen reported that the Commission on Allied Health Professions and Services recommended that the resolution be studied in greater detail by another appropriate group before a formal recommendation was made to the Council. The report of the Commission was accepted and it was suggested that an appropriate group for further study of the Resolution be designated by CMA staff.

ACTION: Voted that Resolution No. 19-66 be given further study by an appropriate group to be designated by CMA staff (to be brought to the Council for formal referral).

24. *Liaison Committee to CMAA*

Doctor Grunigen presented to the Council a recommendation for the merger of the Liaison

Committee to the CMAA with the CMAA Advisory Committee. The single committee would consist of eight physicians, four of whom would be appointed by the CMA Council and four to be designated by the CMAA Board.

ACTION: Voted to adopt the proposal and refer it to the Committee on Committees for report back to the Council.

25. *Conference on Paramedical Groups*

Chairman Grunigen informed the Council that the Commission on Allied Health Professions and Services recommended holding a conference on paramedical groups and Council approval was sought. In the discussion that followed, it was decided that because the newly-created Health Manpower Council was to be involved in this type of activity, it might be better to wait until the activities of the Health Manpower Council were more clearly defined, to see if there would be a need for such a conference.

26. *Medical Executives Conference*

Speaking for the Chairman of the Conference, Mr. Norman A. Brown, Executive Secretary of Sonoma County Medical Society, reported to the Council on the 1 and 2 December meeting of the ad hoc Committee on Medical Executives Conference format. Mr. Brown informed the Council that the committee had suggested one hundred items for consideration. Those designated for early implementation or consideration included: Continuation of the 10:00 a.m. meeting of voting members of the Medical Executives Conference; encouragement of more consultation between CMA representatives and executive secretaries at the county level; and invitation of executive staff representatives of California medical specialty groups to the afternoon session of the Conference. Mr. Brown also reported that the Medical Executives Conference wished to offer to the Council the services of individual executive secretaries as resource personnel to appropriate CMA committees and ad hoc groups and it was requested that this information be transmitted to the Committee on Committees for its information. Upon Doctor Morrison's request, Mr. Brown said that an outline of the particular areas of knowledge of individual executive secretaries would be provided to the Committee on Committees.

27. *Utilization Review in Extended Care Facilities*

Mr. Boyd Thompson, executive secretary of the San Joaquin County Medical Society, initiated the discussion on utilization review in extended care facilities by describing a meeting on the subject recently attended by executives from 12 county medical societies. Mr. Thompson asked the Council's assistance in clarifying for county societies CMA's position on payment for physicians for *administrative functions* involved in utilization review of extended care facilities.

Chairman Anderson called on Councilor Albert Miller, chairman of the Commission on Public Agencies, to comment, on behalf of the Commission, on Mr. Thompson's request and other items relating to the general subject of utilization review in extended care facilities. Councilor Miller reported that his commission had held a meeting the previous evening on the subject and presented two recommendations relating to it. Discussion followed.

ACTION: Voted (1) to encourage county medical societies to become more active in utilization review in extended care facilities, and (2) to authorize dissemination to county societies all available background information on the subject.

ACTION: Voted to authorize the Commission on Public Agencies to establish a committee for the purpose of establishing guidelines to assist in utilization review of extended care facilities.

On the question of reimbursement of physicians for utilization review in extended care facilities, Doctors Miller, Anderson and Teall each expressed the opinion that Council action of 17 September did not preclude an institution's compensating a physician for *administrative functions* associated with utilization review. Chairman Anderson suggested that CMA policy on the subject might be spelled out more clearly in the written report of the Commission on Public Agencies.

28. *Statement on Certification of Nursing Homes*

Mr. William Whelan of the CMA staff announced that a statement had just been released by the Office of Health Care Services which requires that all nursing homes, if they are to continue to receive payments under Medi-Cal as of January 1967, must either (a) be qualified to participate as an extended care facility under Title 18 of the Social Security Act; or (b) state their intention to meet the requirements for participation under Title 18 by 1 January 1968.

29. *Status Report on 1966 House of Delegates Resolutions*

Chairman Anderson announced that the Council is required to submit to the delegation 90 days in advance of the Annual Session a status report on the resolutions and actions of the previous House of Delegates meeting. The proposed status report was distributed; action prior to distribution will be taken up at the January meeting of the Council.

30. *Blue Shield-California Physicians' Service*

Board Chairman Richard Wilbur reported that under the Medi-Cal program, Blue Shield had paid out over $100,000,000 for more than eight million claims (50 per cent of this money to physicians). Under the Medicare program, about 388,000 claims had been processed, and $6,715,000 had been paid out. However, 258,000 of the Medicare claims were "no pay" statements because the $50 deductible had not yet been met. Doctor Wilbur also informed the Council that Blue Shield has revised its system for claims handling and that eligibility determination is one of the first things to be done under this new system. The claims, he said, are first cleared through the computer; those that do not clear are checked manually and if still not cleared, are returned to the physician for clarification.

Councilor Bullock said that he had been requested by the Los Angeles County Medical Association Council to request that Blue Shield send out information on the various over-65 plans available to those persons who had been cancelled out with the advent of Medicare.

ACTION: Voted to request Blue Shield-CPS to publicize the various over-65 programs available to those persons whose policies were cancelled out with the advent of Medicare.

31. *Malpractice Insurance*

Councilor Yant reported on the recommendations of the Commission on Professional Welfare and its Medical Review and Advisory Committee on the subject of availability of malpractice insurance. It was recommended that a questionnaire survey be conducted on malpractice and other types of physician insurance.

ACTION: Voted to authorize MRAC to prepare a questionnaire to be used for the purpose of surveying physician problems and requirements in the area of malpractice insurance and related matters (to be submitted to the Council for approval before initiating the survey).

32. *Treatment of Workmen in Student Health Centers*

Councilor Maguire inquired on progress being made by the Committee on Occupational Health and Rehabilitation in fulfilling its assignment of looking into the problem of student health centers providing and charging for treatment of workmen employed by contractors on various campuses of the University of California. Chairman Anderson asked that the committee explore the situation further and report back to the Council.

33. *Billing Under Amended Military Dependents Medical Care Act*

President MacLaggan briefly discussed problems encountered under the complicated method of billing to be used by physicians providing care under the amended Military Dependents Medical Care Act. He stated that the problems were aptly described in a letter sent by Doctor Milo Youel to General Peatfield.

ACTION: Voted to refer the problem to the Committee on Federal Medical Care Programs (Doctor Youel's letter to be transmitted to the committee).

34. *Report of the Executive Director*

Mr. Howard Hassard reported on the position of Executive Director. He stated that during his eight years of tenure, the scope and complexity of the organization had increased to the extent where, he felt the association would be better served if the position were full-time.

It was Mr. Hassard's recommendation that a search be instituted to find a person suitable to fill a full-time executive directorship. He stated that he wishes, if his recommendation is accepted, to return to the practice of law and continue as legal counsel of CMA.

ACTION: Voted to accept the recommendation of the Executive Director to institute a search for a full-time Executive Director.

Dr. Teall recalled the circumstances of the appointment of the Executive Director and Mr. Hassard's selection for the position. He commended Mr. Hassard for his excellent performance and expressed appreciation for the superb job he had done for CMA as Executive Director.

ACTION: The Council voted deep appreciation to Mr. Hassard for his service to CMA as Executive Director.

Mr. K. L. Hamman was asked to discuss the methods of selecting executive personnel and how to develop the criteria for such selection.

Mr. Hassard asked for assistance in his search for a new Executive Director. The Council discussed the means of implementation.

ACTION: Voted to request the Committee for Emergency Action to cooperate with Mr. Hassard in the search for a full-time Executive Director.

35. *Feasibility Study To Evaluate the Quality of Medical Care*

The feasibility study to evaluate the quality of medical care in individual medical practice was presented to the Council by the Bureau of Research and Planning. The Council directed the California Medical Education and Research Foundation to submit the document to the supportive agency—the U.S. Public Health Service, according to the contract, with appropriate modifications.

ACTION: Voted to direct CMERF to submit the feasibility document to the supportive agency with the appropriate modifications.

ACTION: Voted to direct the Bureau of Research and Planning to defer its Socio-Economic report on the subject at this time.

36. *Council for Health Plan Alternatives*

Councilor Roberta Fenlon discussed the work being done by the University of California "University-wide Committee" designated by UC President Clark Kerr to explore areas of possible cooperative research with the California Council for Health Plan Alternatives. Doctor Fenlon is a member of the UC Committee and requested possible aid from the CMA Bureau of Research and Planning in developing appropriate information for the committee's use.

ACTION: Voted to request the Bureau of Research and Planning to develop appropriate information in cooperation with Doctor Fenlon, as necessary.

37. *Membership*

Seven applicants were voted election to Associate Membership. These were: Seawright W. Anderson, Orange County; Jean E. Hayward, Maylene Wong, San Francisco County; Barksdale E. Fortson, Jean Haber Green, George J. Magid, Frederick Paul Schnell, Santa Clara County.

Five members were voted election to Retired Membership. These were: Helen M. Koke, Alameda-Contra Costa County; Jasper M. Humphreys, Butte-Glenn County; Charles Bothamley, Forty First Medical Society; Nathan Barbour, San Joaquin County; Paul T. Martin, Santa Clara County.

Reduction of dues was voted for 10 members for reasons of prolonged illness or postgraduate education.

38. *Roll Call*

Present were President MacLaggan, President-Elect Morrison, Speaker Quinn, Vice-Speaker Telford, Secretary Weyrauch, and Councilors Isenhour, Eastman, Melone, Todd, Gooel, Bullock, O'Connor, Rogers, Crum, Boyle, Maguire, Burnett, Richard S. Wilbur, Miller, Watts, Fenlon, Kay, Kaiser, Yant, Grunigen, Longmire, and Immediate Past President Teall. Absent for cause were Editor Dwight L. Wilbur and Councilors Taw and Ham.

Present by invitation were CMA staff members Becker, Belser, Bowman, Clark, E. Collins, J. Collins, Eberlein, Edwards, Goldman, Griffith, Hetland, Klutch, Miller, Price, Redfern, Thomas and Whelan; Messrs. Hassard and Huber, legal counsel; component society executives Scheuber of Alameda-Contra Costa, Rideout of Butte-Glenn, Lingerfelt of Fresno, Geisert of Kern, Brock of Imperial, Johnson of Los Angeles, Sower of Marin, Colvin of Monterey, Blough of Orange, Walters of Riverside, Dochterman of Sacramento, Donmyer of San Bernardino, Nute of San Diego, Neick of San Francisco, Thompson of San Joaquin, Wood of San Mateo, Marvin of Santa Barbara, and Brown of Sonoma; Messrs. Paton, Babb, Heller, Koch, Light and Potloff of Blue Shield-California Physicians' Service; Messrs. Read, Brown and Putnam of the Public Health League; Doctor Glaser of Stanford School of Medicine; Doctor Bostick of UC-California College of Medicine; and Doctor Tschirgi of UC-San Diego; Mr. Ward and Doctor Rosen of the Health and Welfare Agency, Doctor Day of the State Department of Public Health, Doctor Skelly of the State Department of Social Welfare, Doctor Young of the State Department of Rehabilitation; Mr. Gordon Cumming of the California Hospital Association, Mrs. Florey of the California Medical Assistants Association, Mrs. Hancock of the California Nurses' Association; Mr. Gould of the American Medical Association; Doctors Davis, Herzog, Hill, Hinshaw, Kilroy, Ritter, Scarborough and others.

Adjournment

The meeting was adjourned at 5:10 p.m.

CARL E. ANDERSON, M.D., *Chairman*
HELEN B. WEYRAUCH, M.D., *Secretary*

CALIFORNIA MEDICAL ASSOCIATION

1967 ANNUAL SCIENTIFIC ASSEMBLY

Biltmore Hotel • Los Angeles • April 15-19

Plan NOW to Attend

Three General Sessions on

ADVANCES IN DIAGNOSTIC TECHNIQUE

(See Program Following Page 158)

DISTINGUISHED SPEAKERS INCLUDE:

- ★ William A. Altemeier, M.D., Chief, Department of Surgery, University of Cincinnati College of Medicine, Cincinnati
- ★ Irvine H. Page, M.D., Director, Research Center, Cleveland Clinic, Cleveland
- ★ Herbert M. Stauffer, M.D., Professor and Chairman, Diagnostic Section, Department of Radiology, Temple University Science Center, Philadelphia
- ★ Jon V. Straumfjord, Jr., M.D., Professor and Chairman, Department of Clinical Pathology, Medical College, University of Alabama, Birmingham
- ★ George Zur Williams, M.D., Chief, Clinical Center, National Institutes of Health, Bethesda

SPECIAL CONFERENCES ON:

Cancer of the Colon and Rectum

Research Advances in Veterinary Medicine

The Impact of Medicare—Titles 18 and 19

Medicine and Religion—Moral Aspects of New Procedures

PLUS . . .

EIGHTEEN SCIENTIFIC SECTION PROGRAMS

SCIENTIFIC AND TECHNICAL EXHIBITS

COLOR TV AND FILM SYMPOSIA

application for HOTEL ACCOMMODATIONS

NINETY-SIXTH *Annual Session*

CALIFORNIA MEDICAL ASSOCIATION • APRIL 15-19, 1967

BILTMORE HOTEL, LOS ANGELES

House of Delegates Opening Session Saturday evening, April 15; Scientific Programs begin Sunday morning, April 16.

1. Fill in the form below **completely** for room accommodations at the CMA's 1967 Annual Session. There is only a limited number of rooms available. Your choice of accommodations will be better if your request is for rooms to be occupied by two or more persons.
2. Your reservation request should include the definite date and hour of your arrival and departure.
3. All reservations must be made through the CMA Housing Bureau, 693 Sutter Street, San Francisco, California 94102.
4. **DEADLINE** for housing: April 1, 1967.

HOTEL ROOM RATES*

	Single	Double and Twins	Suites
BILTMORE HOTEL			
515 So. Olive Street	10.50-17.50	15.50-21.50	30.00-55.00
MAYFLOWER HOTEL			Triples
535 So. Grand Avenue	8.50-13.50	12.00-17.50	22.00
STATLER-HILTON			Suites
930 Wilshire Boulevard	11.00-17.50	16.00-22.50	30.00-65.00

*Rates subject to change

Send to: California Medical Association—*Housing Bureau*
693 Sutter Street, San Francisco, California 94102

Please reserve the following accommodations for the CMA's 1967 Annual Session in Los Angeles. April 16-19, 1967. The first meeting of the House of Delegates begins Saturday evening, April 15; Scientific Programs begin April 16.

Single Bedroom $........ Twin-Bedded Room $........ Double Bedroom $........

Small Suite $........ Large Suite $........ Other $........

First Choice Hotel........ Second........

Arrival (date) .... Hours........ a.m. p.m. Departure (date) .... Hour........ a.m. p.m.

THE NAME OF EACH HOTEL GUEST MUST BE LISTED. Include the names and addresses of both persons for each twin-bedded or double room, and names and addresses of all other persons for whom you are requesting reservations.

—PLEASE PRINT—

Your Name .... Officer? .... Delegate? .... Alternate? .... Speaker? ....

Address .... County ....

City and State........ Zip No. ....

In Memoriam

Bigotti, Richard Thomas, San Jose. Died 21 December 1966, in San Jose, aged 58. Graduate of Creighton University School of Medicine, Omaha, Nebraska, 1936. Licensed in California in 1937. Doctor Bigotti was a member of the Santa Clara County Medical Society.

◆

Bruck, Edwin L., San Francisco. Died 5 January 1967, in Ross, aged 73. Graduate of the University of California School of Medicine, Berkeley-San Francisco, 1920. Licensed in California in 1920. Doctor Bruck was a member of the San Francisco Medical Society.

◆

Dahlgren, Roland Walfred, Fresno. Died 4 October 1966, in Fresno, aged 70, of cirrhosis of the liver and arteriosclerotic heart disease. Graduate of the University of Nebraska College of Medicine, Omaha, 1923. Licensed in California in 1924. Doctor Dahlgren was a member of the Fresno County Medical Society.

◆

Godett, Michael Robert, San Marino. Died 24 December 1966, in Los Angeles, aged 62, of cancer of the lung, metastatic. Graduate of Stanford University School of Medicine, Palo Alto-San Francisco, 1933. Licensed in California in 1933. Doctor Godett was a member of the Los Angeles County Medical Association.

◆

Hand, Delbert Wilfred, San Francisco. Died 2 January 1967, in San Francisco, aged 62. Graduate of Creighton University School of Medicine, Omaha, Nebraska, 1929. Licensed in California in 1929. Doctor Hand was a member of the San Francisco Medical Society.

◆

Jones, F. Burton, Vallejo. Died 2 January 1967, in San Francisco, aged 75. Graduate of George Washington University School of Medicine, Washington, D.C., 1914. Licensed in California in 1915. Doctor Jones was a member of the Solano County Medical Society.

◆

Kohlhase, Lawrence H., Los Angeles. Died 4 May 1966, in Los Angeles, aged 82, of heart disease. Graduate of Northwestern University Medical School, Chicago, Illinois, 1915. Licensed in California in 1920. Doctor Kohlhase was a member of the Los Angeles County Medical Association.

◆

Lusby, Fletcher H., Inglewood. Died 13 September 1966, aged 62, of diabetes mellitus and pneumonia. Graduate of the College of Osteopathic Physicians and Surgeons, 1934. Licensed in California in 1934. M.D. degree from California College of Medicine, 1962. Doctor Lusby was a member of the Los Angeles County Medical Association.

◆

Manson, Guy, Fresno. Died 27 December 1966, in Fresno, aged 81. Graduate of Cooper Medical College, San Francisco, 1910. Licensed in California in 1910. Doctor Manson was a member of the Fresno County Medical Society, a life member of the California Medical Association, and a member of the American Medical Association.

◆

Pottenger, Francis Marion, Jr., Monrovia. Died 4 January 1967, in Azusa, aged 65. Graduate of the University of Cincinnati College of Medicine, Ohio, 1930. Licensed in California in 1930. Doctor Pottenger was a member of the Los Angeles County Medical Association.

Raitt, G. Emmett, Santa Ana. Died 30 December 1966, aged 72, of cerebral hemorrhage. Graduate of the University of California School of Medicine, Berkeley-San Francisco, 1926. Licensed in California in 1927. Doctor Raitt was a member of the Orange County Medical Association.

◆

Schinagel, Geza, Burbank. Died 4 January 1967, in Los Angeles, aged 73. Graduate of Orvosi Fakultas Tudomanyegyetem, Budapest, Hungary, 1919. Licensed in California in 1942. Doctor Schinagel was an associate member of the Ventura County Medical Society.

◆

Spirtos, George Nicholas, Woodland Hills. Died 8 August 1966, in Kauai, Hawaii, aged 45, of coronary thrombosis. Graduate of Des Moines Still College of Osteopathy, Des Moines, Iowa, 1957. Licensed in California in 1961. M.D. degree from California College of Medicine, 1962. Doctor Spirtos was a member of the Forty First Medical Society.

◆

Stone, Robert Spencer, San Francisco. Died 18 December 1966, in San Francisco, aged 71, of cancer. Graduate of the University of Toronto Faculty of Medicine, Ontario, Canada, 1924. Licensed in California in 1928. Doctor Stone was a member of the San Francisco Medical Society.

◆

Taylor, Merrel H., San Diego. Died 8 October 1966, aged 85. Graduate of Denver and Gross College of Medicine, Denver, Colorado, 1905. Licensed in California in 1942. Doctor Taylor was a retired member of the San Diego County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

◆

Welch, Sylvester H., Glendale. Died 5 December 1966, in Glendale, aged 83. Graduate of Vanderbilt University School of Medicine, Nashville, Tennessee, 1905. Licensed in California in 1924. Doctor Welch was a member of the Los Angeles County Medical Association.

◆

Wharton, George Murdock, Los Angeles. Died 21 November 1966, in Los Angeles, aged 48, of coronary occlusion. Graduate of Howard University College of Medicine, Washington, D.C., 1950. Licensed in California in 1955. Doctor Wharton was a member of the Los Angeles County Medical Association.

◆

Willson, Lamont Roy, Fresno. Died 17 December 1966, in Fresno, aged 88. Graduate of the University of Illinois College of Medicine, Chicago, 1903. Licensed in California in 1903. Doctor Willson was a member of the Fresno County Medical Society, a life member of the California Medical Association, and a member of the American Medical Association.

◆

Wuerthele, Herman William, La Jolla. Died 5 January 1967, in La Jolla, aged 83. Graduate of the University of Pittsburgh School of Medicine, Pennsylvania, 1906. Licensed in California in 1953. Doctor Wuerthele was a retired member of the San Diego County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

Continuing Medical Education

Toward Improving Postgraduate Medical Education

PHIL R. MANNING, M.D.
Professor of Medicine
Associate Dean for Postgraduate Medical Education
School of Medicine
University of Southern California

and

JAMES C. MAC LAGGAN, M.D.
President, California Medical Association

"POSTGRADUATE MEDICAL EDUCATION for the practicing physician is inadequate, inefficient, and ineffective." This statement would probably receive supporters from physicians in all walks of life, whether they be in practice, academic medicine, or medical administration. The concern of the practicing physician that medical schools do not provide many postgraduate experiences which are helpful to them in daily practice, coupled with the concern of the faculty member that the practicing physician is not really interested in academic medicine, creates a frustrating and difficult situation.

Teaching methodology in postgraduate medical education also has received its share of complaints. For the most part, the complaints are centered about the inadequacies of the teacher and his presentation and the fact that presentations are usually based on passive techniques. In addition the subject matter itself is often not relevant to the problems of the practicing physician. It is somewhat surprising that despite the dissatisfaction with the current approach to postgraduate education for the practicing physician, medical school faculties and the practicing community spend tremendous amounts of time, energy, and money in striving to utilize postgraduate medical education to help physicians provide patients with the best medical care possible.

The desire of physicians in the California Medical Association and the faculties of the various schools of medicine within the State to seek better ways in postgraduate education has been guiding the California Medical Association to take steps to work with the medical schools in improving postgraduate medical experiences. To this end, several important studies in the field of postgraduate medical education are being undertaken. It is hoped that these studies will ultimately provide the medical school faculties with an understanding of the subject matter that will be most beneficial to the physician in his quest to offer the finest in medical care. In addition, it may be hoped that the studies will provide medical school faculties information that will allow them to develop improved techniques in teaching the practicing physician.

In order to begin to evaluate various teaching techniques it will, of course, be necessary to enlist the active support of the members of the California Medical Association. The chief support that will be required will be participation in some of the experiments which will test teaching techniques. This might mean participating in a postgraduate session in which the effectiveness of one type of teaching technique would be compared with another. Testing for information-gains in these experiments will be aimed at evaluating the teaching technique and not the physician himself.

It is hoped that participation in experiments in medical education will be well received by members of the California Medical Association. It is perhaps not too much to expect that the participation will be interesting and even exciting. In all events, the cooperation of the members of the CMA and medical school faculties should lead to providing physicians with significantly better postgraduate programs than are now available.

Physicians representing the practicing community of the West Coast counties have already indicated interest in this project by agreeing to have a portion of the CMA Institute, to be held March 2 and 3, 1967 at Pebble Beach, devoted to an experiment in teaching techniques. It is hoped that all of the physicians present will participate actively in this project which could be a step toward improving the educational experience available to the physician in the State.

17th annual regional postgraduate institute

DEL MONTE LODGE

Pebble Beach

March 2-3, 1967

WEST COAST COUNTIES

Presented cooperatively by West Coast Counties Medical Societies, University of Southern California School of Medicine, Los Angeles, and the Committee on Continuing Medical Education, California Medical Association. A 12 hour, 45 minute course.

HOST: Monterey County Medical Society
***Regional Chairman:* Donald M. Scanlon, M.D.**

INSTITUTE FEE: $15.00. For additional information contact Continuing Medical Education office, California Medical Association, 693 Sutter Street, San Francisco 94102. All California Medical Association members and their families are cordially invited to attend.

GUEST SPEAKERS: Virginia E. Johnson, Research Associate, Reproductive Biology Research Foundation, St. Louis, Missouri, **William H. Masters, M.D.**, Director, Reproductive Biology Research Foundation, St. Louis, Missouri.

EVENING SPEAKER: Eric Berne, M.D., Author of "Games People Play."

PROGRAM THEMES:

LIVER DISEASE...COMPLICATIONS OF PREGNANCY... HUMAN SEXUAL RESPONSE...GASTROINTESTINAL DISEASES...THE MEDICAL INTERVIEW...EXPERIMENT IN MEDICAL EDUCATION

THURSDAY, MARCH 2

Morning Session

PLENARY SESSION (attendance limited to physicians registered in course)

9:00—**Female Sexual Physiology**—Virginia E. Johnson and William H. Masters, M.D.

10:00—**Coffee Break**

10:15—CONCURRENT WORKSHOPS (you may go to one of your choice)

A. Workshop in Liver Disease—Part I—William P. Mikkelsen, M.D., Telfer B. Reynolds, M.D., and Allan G. Redeker, M.D.

B. Workshop in Medical Complications of Pregnancy— Gail V. Anderson, M.D., and Phil R. Manning, M.D.

C. Workshop in Gastrointestinal Diseases—Recent Developments in Peptic Ulcer Disease —Bernard J. Haverback, M.D.

D. The Medical Interview—Allen J. Enelow, M.D.

Afternoon Session

PLENARY SESSION

1:30—**Contraceptive Physiology**—Virginia E. Johnson and William H. Masters, M.D.

2:15—CONCURRENT WORKSHOPS

A. Workshop in Liver Disease—Part II—William P. Mikkelsen, M.D., Telfer B. Reynolds, M.D., and Allan G. Redeker, M.D.

B. Workshop in Complications of Pregnancy—Maternal Mortality Due to Hemorrhage—Gail V. Anderson, M.D., and Lester T. Hibbard, M.D.

C. Workshop in Gastrointestinal Diseases—Recent Developments of Pancreatic Diseases—Bernard J. Haverback, M.D.

D. The Medical Interview—Allen J. Enelow, M.D.

E. Experiment in Medical Education—James V. Dooley, M.D., Irwin Hoffman, M.D., and Julien H. Isaacs, M.D.

Thursday Evening

8:00—**Dinner**—Presiding Joseph E. Turner, President, Monterey County Medical Society
Guest Speaker: Eric Berne, M.D., "Games Played During Medical Interviews"

FRIDAY, MARCH 3

Morning Session

PLENARY SESSION

8:45—**Male Sexual Physiology**—Virginia E. Johnson and William H. Masters, M.D.

9:15—CONCURRENT WORKSHOPS

A. Experiment in Medical Education—James V. Dooley, M.D., Irwin Hoffman, M.D., and Julien H. Isaacs, M.D.
B. Workshop in Liver Disease—Part II (for those who attended Experiment in Medical Education in previous day)—William P. Mikkelsen, M.D., Telfer B. Reynolds, M.D., and Allan G. Redeker, M.D.

Afternoon Session

PLENARY SESSION

1:30—**Human Sexual Inadequacy**—Virginia E. Johnson and William H. Masters, M.D.

3:00—**Coffee Break**

3:15—CONCURRENT WORKSHOPS

A. Workshop in Liver Disease—Part III—William P. Mikkelsen, M.D., Telfer B. Reynolds, M.D., and Allan G. Redeker, M.D.
B. Workshop in Maternal Mortality—Maternal Mortality Due to Obstetrical Infections—Gail V. Anderson, M.D., and Lester T. Hibbard, M.D.
C. Workshop in Gastrointestinal Diseases—Recent Developments of Small Intestine Diseases—Bernard J. Haverback, M.D.
D. The Medical Interview—Allen J. Enelow, M.D.

PUBLIC HEALTH REPORT

Lester Breslow, M.D., M.P.H.
Director, State Department of Public Health

MORE THAN 300 MENTALLY retarded persons and their families received assistance during the first 10 months of operation of the state's two regional centers. Ninety-three per cent of the families who requested center care have continued with the program.

The regional center program was established during the 1965 session of the State legislature. The State Department of Public Health in December of that year awarded the first two contracts for center operation to Aid Retarded Children, Inc., San Francisco, and to Los Angeles Childrens Hospital.

The centers began operation in February 1966. Problems of recruitment of skilled personnel limited operations at first, but by the end of June both centers were fully staffed and caseloads then increased considerably.

Services of the two centers include diagnosis, counseling on a continuing basis to the mentally retarded person and his family, special medical services, nursery and preschool training, recreation, vocational rehabilitation and residential facilities.

During the first year of operation priority for assistance was given to persons who had been accepted for care in a state hospital but for whom beds were not currently available.

In the first two months of operation 40 per cent of the families requesting assistance had already placed their retarded children outside their home. By late autumn only 20 per cent of the families requesting assistance had resorted to this alternative.

Over half of the assistance provided by the centers has been counseling activities. Because of this, and the decrease in the proportion of crisis cases, the State Health Department has arranged for increased counseling staff at both centers.

Eighty per cent of the mentally retarded persons receiving assistance were born in California, 19 per cent in other states and 1 per cent outside the United States. The age range is from less than one year to 52 years. Twenty-one per cent are under five years of age, 72 per cent between the ages of five and 21, and 7 per cent are 21 years of age or older.

Envisaged is a statewide network of regional centers to help in their own communities the thousands of mentally retarded persons who do not need to be relegated to lifetime confinement in a state institution. At the same time the program will make institutional care available to those who really need it.

The 1966 Population Study Commission has completed its assignment and its findings and recommendations have been published in a report prepared by the State Department of Public Health.

Twenty-six recommendations were made, dealing with such issues as broader dissemination of family planning information and services, therapeutic abortion legislation, family life education and extending Medi-Cal benefits to all low income families.

Single copies of the report may be obtained upon request to Theodore A. Montgomery, M.D., Chief, Division of Preventive Medical Services, State Department of Public Health, 2151 Berkeley Way, Berkeley.

WOMAN'S AUXILIARY

to the California Medical Association

American Medical Association Education Research Foundation

THE WOMAN'S AUXILIARY to the California Medical Association is working diligently to promote the program of the American Medical Association Education Research Foundation.

The Auxiliary has been asked to actively support three important projects:

FUNDS FOR MEDICAL SCHOOLS

The Foundation distributed $1,133,585 to 108 medical schools in the United States and Canada this past year. This sum represented contributions from physicians and from women's auxiliaries to the AMA-ERF Fund for Medical Schools program.

Since 1951, the Fund has distributed $16,600,000 to medical schools. Medical School deans may use the funds as they see fit. Typically, the money goes toward faculty salaries, improvement of laboratories and libraries, student assistance programs and general operating expenses.

Medical societies of states in which the schools are located are distributing the funds for AMA-ERF.

The distribution in California this past year was as follows:

Loma Linda	$ 41,237.72
Stanford	33,566.94
California (San Francisco)	4,467.44
Southern California	38,163.97
California (Los Angeles)	5,687.11
California College of Medicine	93,816.89
California (San Diego)	35.00
TOTAL	$216,975.07

Funds distributed in California far exceed those of any other state. Private support is an absolute necessity if medical education is to remain at its high level.

LOAN GUARANTEE FUND

Perhaps, one of the most rewarding of the AMA-ERF projects is the Loan Guarantee Fund, since it provides funds for deserving persons so they may continue their medical education.

This past year the Loan Fund extended an average of 685 loans per month to medical students, interns and residents, for a total of 8,213 loans worth $9,573,050 in principal amount. This represented an 11 per cent increase over the previous year.

Californians were granted 912 loans in amount of $1,103,500. This consisted of 11 per cent of the entire program. More loans were made to trainees in this state than in any other. This has been true every year since the program began.

We can make two observations:

1. The AMA-ERF Loan Program is still expanding, with considerably increased borrowing by interns and residents. The government does not make loans to interns and residents.
2. Even though the government is offering medical students loans at a reduced rate of interest, the effect of that program so far appears to be in the direction of increasing total borrowing among the students rather than displacing the AMA-ERF program.

During the past year the availability of money for loans of all types has declined sharply. The consensus of many economists is that the economy cannot absorb the combined impact of a large domestic welfare program and a war. The current "tight money" conditions emphasize that money is desperately needed for the Loan Fund.

THE INSTITUTE OF BIOMEDICAL RESEARCH

What is it?

The Institute is a place where creative scientists have an unequalled opportunity to explore the mysteries of life, free from restrictions of administrative, publishing and teaching duties.

Why is it so important?

The Institute is devoted to a single purpose—basic investigation of the living cell, with the hope that what is learned will lead to discoveries that will provide better health for all.

Who will staff it?

The Institute has attracted top scientists from around the world, each working on an individual project in the field of basic biomedical research.

Where is it?

The Institute is housed within the new addition to the AMA Headquarters building.

How can you help?

Supported solely by contributions, the Institute needs our help to raise funds for continued research.

The money we contribute helps:

- Pay salaries of scientists and laboratory assistants;
- Buy laboratory equipment and supplies;
- Create an atmosphere where scientists are free from the concern of support for their projects.

The Biomedical Research Program is new to our Auxiliary.

This year, the Auxiliary has been asked by the president of the national organization to nearly double the last year's AMA-ERF contribution. This is a challenge we readily accept as we realize that medical education and biomedical research is the foundation of the practice of medicine.

Many projects have been developed by AMA-ERF to assist us in raising these necessary funds. Each county Auxiliary is actively participating in some phase of the program.

We encourage physicians making contributions to medical schools to do so through our Auxiliary. Checks should be made payable to "AMA-ERF Auxiliary Fund," and the school that is to receive the funds should be designated. The names of individual State, County and District groups making contributions should also be included so that proper credit can be given. The check can then be mailed direct to AMA-ERF, 535 North Dearborn Street, Chicago, Ill.

Enthusiastically working together, we can keep California on top and at the same time enjoy the satisfaction that comes through work and acomplishment for a most worthy project.

Mrs. Virgil S. Crawley
AMA-ERF Chairman, WACMA

NEWS & NOTES

NATIONAL • STATE • COUNTY

ORANGE

Orange County Medical Center has announced that it has been approved for three years' **orthopedic training** in adult orthopedics, fractures and children's orthopedics. Applications for residency are now being accepted. A year of residency in general surgery is prerequisite to application. The center also has available for orthopedic residents from other programs a six months' residency in children's orthopedics, fully approved.

Applications should be sent to S. H. Anzel, M.D., Chief of orthopedic surgery, Orange County Medical Center, Orange, California.

SAN DIEGO

Dr. Francis E. West was installed as president of the American Academy of Orthopaedic Surgeons at the thirty-fourth annual meeting of the organization held last month in San Francisco. Dr. West is a former president of the California Medical Association and is a member of the California State Board of Medical Examiners.

SAN FRANCISCO

The University of California San Francisco Medical Center will **telecast programs to hospitals** throughout northern and central California with the aid of $553,400 three-year grant from the U.S. Public Health Service, according to Chancellor Willard C. Fleming, who said that the purpose of the grant is to establish the value of such programs in hospital in-training and postgraduate education.

Up to 126 one-hour programs will be telecast over closed circuits during the next three years over KQED, San Francisco; KVIE, Sacramento, and KTEH, San Jose, the announcement said. Included will be actual telecasts of operating **techniques, patient interviews, grand rounds,** as well as several programs of interest to nurses, x-ray technicians and other health professionals such as physical therapists and social workers. Program content will be decided in cooperation with the staffs of the participating hospitals. The telecasts will be available to more than 150 hospitals in the northern and central portion of the state.

SANTA CLARA

Dr. Herbert L. Abrams, professor and head of the Division of Diagnostic Radiology at Stanford University School of Medicine, has been appointed the Philip H. Cook Professor and chairman of the Department of Radiology at Harvard Medical School. He will serve simultaneously as radiologist-in-chief at the Peter Bent Brigham Hospital in Boston. He will assume his new duties in late summer of this year.

* * *

Two Stanford medical alumni have been named to administrative posts at the Stanford University School of Medicine, Dr. Robert J. Glaser, vice president for medical affairs and dean of the Medical School, announced.

Dr. Thomas A. Gonda, 45, professor of psychiatry, has been named associate dean, and **Dr. Gunther W. Nagel,** 72, of San Francisco, emeritus clinical professor of surgery, will serve as director of alumni relations.

Dr. Gonda will have a major responsibility for clinical affairs, including relationships between the Medical School and its affiliated hospitals. He will also be concerned with graduate and postgraduate programs.

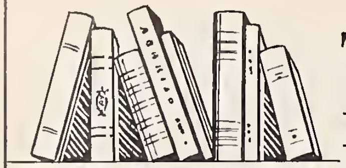
Dr. Nagel, first occupant of a newly created post, will work part-time with the Medical School administration to aid in the continuing development of medical school alumni relationships.

YOLO

Dr. Julian R. Youmans of the Medical College of South Carolina has been named chairman of the neurosurgery department at the **University of California, Davis, School of Medicine.** Dean C. John Tupper of the UCD medical school said that Dr. Youmans will assume his new post 1 July.

GENERAL

Mr. Paul Ward, former administrator of the State Health and Welfare Agency, has been named executive director of the California Committee on Regional Medical Programs. The committee, which was formed to assist medical schools and hospitals in the coordination and development of cooperative medical programs under federal legislation (P. L. 89-239), has received a grant of $223,400 to plan activities for improving diagnosis and treatment of heart disease, cancer, stroke and related diseases. California Medical Education and Research Foundation will act as fiscal agent for the funds. The coordinating committee office will be at 655 Sutter, Suite 302, San Francisco 94102.



The Physician's BOOKSHELF

TREATMENT OF FRACTURES—Supplementary Volume to the 5th English Edition—By Prof. Dr. Lorenz Bohler, Director of the Accident Hospital, Vienna XX; Professor of Traumatology, University of Vienna; and Prof. Dr. Jorg Bohler, Director of the Accident Hospital, Linz; Professor of Traumatology, University of Vienna. Translated from the first German Edition by Alfred Wallner, M.D., Kalispell, Montana. Grune & Stratton, Inc., New York, N.Y., 1966. 402 pages, $28.75.

Any new publication by the doctors Bohler must be received with great interest by any serious student of traumatology. The immense volume of material with which the two men are in direct personal control lends to their words tremendous authority when they speak on the subject of the treatment of fractures. The present volume, the supplementary volume to the 5th English Edition of the *Treatment of Fractures,* is such a work. Unfortunately it suffers from the discontinuity of any supplementary type volume which sends the reader back again and again to the original volume to see just what has been deleted and what added.

In the general section of the volume, dealing with principles of bone healing, the importance of avoidance of distraction of the fracture fragments is stressed. It is the opinion of the authors that too much traction is the most common cause of delayed and/or non-union. In addition to pseud-arthrosis and delayed union the authors ascribe Sudeck's dystrophy as being caused by excessive traction. Another cause of Sudeck's atrophy is thought to be physical therapy. Following the general section of approximately 50 pages there comes a new section on head injuries written by Joerg Bohler which gives an excellent description of the mechanism of the injuries, the clinical findings and the differential diagnosis of the various types of intracranial trauma. Treatment and postoperative care and post injury evaluation is also given in great detail.

Another special section deals with closed intramedulary nailing of tibial fractures. This technique, utilizing biplanar image amplifier fluoroscopy, serves to show how much further advanced is the instrumentation in Austria than in most major centers in this country. The remainder of the book describes the techniques of treating various injuries of the upper extremity as well as dislocations of the hip. For the beginning student the check list which is given at the end of each section, outlining the various steps which must be taken, will be very helpful. The admonition that the plaster cast must be split to the very last thread is given in each section to insure against circulatory disturbances, and is an admonition to which attention is justly given.

The volume is an excellent addition to the lore of orthopedic surgery, and should be included in the library of every serious orthopedist, along with the 5th English Edition to which the present volume is a supplement.

JEROME C. BEATIE, M.D.

PSYCHOTHERAPY: MYTH AND METHOD—An Integrative Approach—By Jan Ehrenwald, M.D., Visiting Consulting Psychiatrist, The Roosevelt Hospital, New York City. Grune & Stratton, Inc., New York and London, 1966. 212 pages, $7.75.

Dr. Ehrenwald has written a very scholarly text on the complex subject of psychotherapy. The title of the book was a bit misleading to me, as it brought to mind immediately the more polemical book, "Myth and Mental Illness" by Dr. Thomas Scaz. There is no similarity between the books, despite the common word in the title. This book follows the history of psychotherapy starting with the early myths of treatment in ancient times and it continues to explore the force of myths in modern day therapeutic approaches. The author examines in historical sequence the continuum of primitive magical healing to current psychoanalytic, behavioral and existential treatment approaches. He applies the elements of this historical schema to the analysis of each current treatment approach in a fruitful way. The polarities of myth and cold rationality in each system are always clearly explicated, and in fact, it is this service to the reader that is to my mind the most important contribution of the book.

The form of the book is quite an enigma; its strengths are also weaknesses. The comprehensiveness of the book is astounding and the author is obviously well-informed and suited to write such a book. Yet each therapeutic system is so succinctly included and fits so smoothly into the developing thesis that one can't help but feel that only those elements of the system that fit are emphasized. Where obvious discordant material appears, it is minimized. When case material is presented, the explanatory statements within the case narrative are so eclectic (from existential shift to corrective emotional experience to interpersonal expectations to cultural value systems to psychoanalytic id psychology) that conceptual jumps are confusing. Although on the surface the conceptual jumps are quite acceptable, further reflection shows that not enough data is available to apprehend meaningfully the conceptual evaluation.

These same considerations which are obviously thought above to be weaknesses are also the strengths of the book, which is subtitled, "An Integrative Approach." The case narratives present data and concepts which bridge the multiple therapeutic networks now taught in most training centers. The comprehensiveness lends a feeling of logical completeness to a usually boundaryless subject.

The book is well worth reading for physicians who are already familiar in depth with various psychiatric theories. I think the book would be misleadingly simple to a novice in the field, unless he were constantly aware that counter arguments to the main thesis are not strongly presented. This is not the book to judge the dissimilarities of various theories of therapy, but Dr. Ehrenwald certainly presents the similarities in a cogent and comprehensive manner.

ROGER L. GOULD, M.D.

SURGICAL DISEASES OF THE CHEST—Second Edition—Edited by Brian Blades, M.D., Lewis Saltz, Professor of Surgery and Chairman, Department of Surgery, The George Washington University School of Medicine, Washington, D.C. The C. V. Mosby Company, St. Louis, Mo., 1966. 687 pages, $25.00.

Intended primarily as a guide to surgically amenable disease of the thorax and its contents, this second edition fulfills its prime goals. Lesions of the chest wall, lung, esophagus, heart, great vessels and associated unique diagnostic and surgical techniques are reviewed. As in any text utilizing many contributors, the thoroughness of each chapter varies with the individual author and his own interests. Trivial discussion is often given to the etiology, embryology, and pathophysiology of thoracic lesions, while pediatric problems are dealt with in a scanty or superficial manner. Individual approaches to acquired cardiac diseases are followed with little thought given to controversial or disputed issues of diagnosis therapy, or postoperative care. This type of editorial approach in a text mars a book that generally offers much for the medical student, thoracic surgical resident, or physician interested in quickly reviewing the general therapy involved in thoracic and cardiovascular problems. Recent advances in the treatment of coronary artery disease and the use of hyperbaric oxygenation are well thought out and profusely documented.

* * *

PSYCHIATRY IN THE PRACTICE OF MEDICINE—By Allen J. Enelow, M.D., and Murray Wexler, Ph.D., University of Southern California School of Medicine. Oxford University Press, Inc., New York, 1966. 355 pages, $6.50.

This book is the outgrowth of the accumulated experience of the authors in a six-year program at the University of Southern California School of Medicine teaching psychiatry and psychiatric principles to practicing physicians. The book is primarily designed for practicing physicians, not only in the handling of psychiatric problems, which are present increasingly in every physician's practice, but also for a more meaningful understanding of the physician-patient relationship and the usefulness of psychiatric interview techniques for the evaluation and treatment of *every* patient. The book, however, would also be quite useful to the medical student, presenting an integrated approach to psychiatry in general medical practice, as well as a basic textbook and overview of the field of psychiatry itself.

The authors start with an excellent chapter on communication, its importance in the development of personality and in maintaining health, the disruptions of communication that occur at times of illness, and examples of more typical behavioral responses to illness—how they can be understood and how they can be dealt with. There are two chapters on diagnostic technique in an interview situation. The first is concerned with the analysis of what goes on in an interview, both in terms of process and content. It also outlines general principles of how the physician can most effectively guide the interview. The authors give their own classification of the nature and function of various kinds of interventions made by the interviewer which are made more meaningful in the following chapter, where a variety of clinical examples of interviews is presented and then discussed in terms of the techniques and the analysis.

The authors then present, in brief, a general psychiatric textbook. The chapter on the diagnosis of psychiatric disorders covers the whole nosology of psychiatric conditions with excellent brief descriptions and clinical examples. This is followed by a chapter on psychotherapy, presenting two techniques, i.e., process-oriented psychotherapy and supportive therapy, which the authors feel could be used efficaciously by nonpsychiatric physicians. It is not made clear, however, when the physician should attempt to handle a psychiatric disorder by himself and when he should seek outside consultation. I felt there was an implication that any psychiatric disorder could be handled by the general physician if he so desired without a critical evaluation of the limitations and dangers in a given diagnostic situation.

Included is an excellent review of the psychopharmacological agents now in use in psychiatric practice and their usefulness in different diagnostic categories, with a number of excellent case examples. The authors warn of the dangers of using drugs to treat a given symptom complex without concomitant understanding of the psychological basis of the illness and without appropriate psychotherapeutic intervention. Again it is not made clear which illnesses can be safely handled by the nonpsychiatric physician and which are best referred to the psychiatrist.

There is an excellent chapter discussing psychiatric emergencies, which are often first seen by the general physician, with advice on how these are best handled. This chapter covers the suicidal patient, the acute anxiety reaction, the acute psychotic reaction, the acute postpartum reaction and operations. The book ends with a chapter on contemporary psychotherapies which can orient the general physician as to the various schools of psychiatric thought at present.

In summary, this is an excellent book which combines a psychiatric approach, both in theory and technique, to the doctor-patient relationship and the psychiatric conditions that often first appear in the medical office, with a basic textbook of psychiatry. In its enthusiam to encourage the nonpsychiatric physician to handle psychiatric problems in his own office, it does not adequately delineate those situations which are best referred to the psychiatrist.

Robert L. Marvin, M.D.

* * *

CHILDREN OF TIME AND SPACE, OF ACTION AND IMPULSE—Clinical Studies on the Psychoanalytic Treatment of Severely Disturbed Children— By Rudolf Ekstein, Ph.D., Director, Project: Childhood Psychosis, Reiss-Davis Child Study Center, Los Angeles, Calif.; with contributions by Elaine Caruth, Ph.D.; Seymour W. Friedman, M.D.; Arthur Mandelbaum, M.S.W.; Judith Wallerstein, M.S.W., and Dorothy G. Wright, M.D. Appleton-Century-Crofts (A Division of Meredith Publishing Company), New York, N.Y., 1966. 466 pages, $8.75.

This is an excellent book, absorbing to read. The hard-won data concerning severe psychological illness in children are presented in many different ways. The children themselves, their impairment of functions, their harassing illnesses and their treatment are described vividly, in detail, macroscopically and microscopically . . . and over time. Many issues of clinical importance are discussed: Some of these are the complexity and use of diagnostic hours; the effects of the psychotherapist's interventions and interpretations; the effect of the patient on the people around him; the successes and failures of treatment interventions; the delineation of fluctuating states of personality functions in these children. Some of the issues of theoretical importance discussed are: The evolution of psychological functions in sick and well individuals; the nature of action, thought, play, speech; the child's capacity to use objects—both people and inanimate objects—in the creation of his inner world.

Physicians with psychological interests will find this work of great value. I fear that some physicians will be discouraged by sections which use technical psychological and psychoanalytic concepts. It would be very much worth their efforts, though, and the clinical descriptions help and carry the reader forward. Most of the 24 chapters had been published previously as individual articles and each chapter stands as a contribution in its own right. At times, this lends a quality of discontinuity to the book. However, there are two things which more than compensate. I believe the book accurately portrays the manner in which questions arose to the author and his colleagues. The second compensatory or corrective is illustrated by a quote from chapter 23, factor "Psychotherapeutic work with severely disturbed children can only be learned and taught against a background of honest experimentation, scientific curiosity and earnest dedication." This spirit gives the success to the book and firmly welds the total work together.

JOSEPH AFTERMAN, M.D.

* * *

EPILEPSY AND THE LAW—A Report on Legal Reform in the Light of Medical Progress—Revised Second Edition—By Roscoe L. Barrow, Wald Professor of Law and former Dean, University of Cincinnati College of Law; and Howard D. Fabing, M.D., Past-President, American Academy of Neurology, Chairman, Legislation Committee, American Epilepsy Society. Hoeber Medical Division, Harper & Row Publishers, Incorporated, 49 East 33rd St., New York, N.Y. 10016, 1966. Paperback, 174 pages, $5.95.

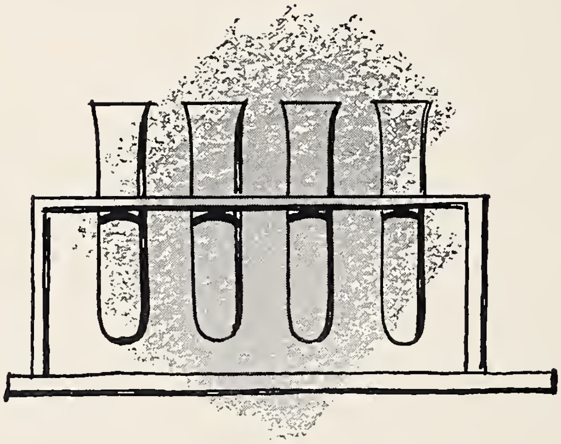
This is a happy second edition. The 10 years since the first edition have seen remarkable changes in the laws regarding epileptics and much of the credit can be taken by the authors and their original report. The Honorable Earl Warren has commented, *"Epilepsy and the Law* probably caused more legislatures to amend more laws in a shorter period of time than any similar research project of the past two decades."

The book itself was planned as a "legislative draftsman's handbook." The new edition should continue to serve this purpose well. It documents the legal changes that have come about in the past 10 years, summarizes the present laws and persuasively makes a case against those that are outmoded, unjust or detrimental to the epileptic. It lacks the impact of the first edition as the remaining unjust or undesirable laws are not nearly as flagrant or appalling as those changed in the past 10 years.

Two new chapters, one on epilepsy itself and the other on the epileptic's family, serve well to bring this into perspective for the layman but hold little for the physician.

The remainder of the chapters cover the various categories of law dealing with or affecting the epileptic. The back of the book contains appendices that give model statutes for driver's license laws (Wisconsin and Ohio), employment of the handicapped (Ohio), and examples of recommendations regarding epileptics. All physicians (in truth, all of society) should be familiar with it. Though some findings have changed because of new laws, there is no better capsule view of the epileptic's problems with society than in these pages.

Of interest to Californians is the argument against mandatory physician reporting of epileptics. This requirement fails to accomplish its purpose and results in driving the epileptic "underground."



96th annual • session program

BILTMORE HOTEL
LOS ANGELES
APRIL 15 TO 19, 1967

SCIENTIFIC SESSIONS • TECHNICAL EXHIBITS
MOTION PICTURE SYMPOSIA • VIDEO TAPE TV PROGRAMS
MEETINGS OF THE HOUSE OF DELEGATES

california medical association

C.M.A. ANNUAL MEETING—1967

SCIENTIFIC SESSIONS

LOCATION	Saturday, April 15		Sunday, April 16		Monday, April 17		Tuesday, April 18		Wednesday, April 19	
BILTMORE HOTEL	A.M.	P.M.	A.M.	P.M.	A.M.	P.M.	A.M.	P.M.	A.M.	P.M.
Biltmore Bowl	**6:00 p.m.** **House of Delegates Opening Session**							**3:00 p.m.** **House of Delegates**	**9:00 a.m.** **House of Delegates**	
Ballroom	**9:30 a.m.** Special Conference Comparative Medicine	**2:00 p.m.**	**9:00 a.m.** General Meeting Diagnastic Techniques	**2:00 p.m.** **L. Henry Garland Memorial Lecture**	**9:00 a.m.** Internal Medicine Calif. Coll. Med. (Gastroenterology)	**2:00 p.m.** General Meeting Advances in Radiolagy	**9:30 a.m.** General Meeting Laboratory Medicine			
Renaissance Room	**9:30 a.m.** Preconventian Radiology Conference	**1:30 p.m.**				**2:00 p.m.** General Surgery		**2:00 p.m.** Generol Surgery	**9:00 a.m.** Internal Medicine/Psychiatry Impact of Public Medicine	**1:30 p.m.**
Galeria Room	**9:00 a.m.** Council of the CMA		**9:00 a.m.** Pathology Cancer Conference	**2:00 p.m.** **8:00 p.m.** Medicine ond Religion	**9:00 a.m.** General Practice Urology	**2:00 p.m.** Gen. Practice Obst. & Gynec.		**2:00 p.m.** Internal Medicine Am. College Cardial.		
Rendezvous Room			**9:00 a.m.** Pediotrics	**12:30 p.m.** Pediatric Lunch **2:00 p.m.** Business meeting						
Music Room		**2:00 p.m.** Symposium on Cancer		**2:00 p.m.** **Film Symposium** Surgery	**9:00 a.m.** **Television**	**2:00 p.m.** **Film Symposia** Cardiology **3:30 p.m.** Pediatrics	**9:00 a.m.** **Television**	**2:00 p.m.** **Film Symposium** Obst. & Gynec.	**9:00 a.m.** **Television**	**2:00 p.m.** **Film Symposium** Medicine
Conference Room 1			**9:30 a.m.** Anesthesiolagy	**2:00 p.m.**	**9:00 a.m.** Disaster Medical Care			**2:00 p.m.** Pediatrics Urology		
Conference Room 2	**9:00 a.m.** Occupational Health Conf.		**9:30 a.m.** Radiology				**9:30 a.m.** Physical Medicine	**2:00 p.m.**		
Conference Room 4	**9:00 a.m.** Pathology		**9:30 a.m.** Allergy **12 Noon** Allergy Lunch	**2:30 p.m.** Allergy		**1:30 p.m.** Preventive Medicine			**9:00 a.m.** Industrial Medicine and Surgery	**2:00 p.m.**
Conference Room 8			**9:30 a.m.** Otolaryngology							
Conference Room 9			**9:30 a.m.** Office Management							
Embassy Auditorium 847 So. Grand Ave.		**Convention Hall**	**9:00 a.m.** Dermatology	**1:30 p.m.**						
		Assembly Hall	**9:15 a.m.** Orthopedics	**2:00 p.m.** Ophthalmology						

TECHNICAL AND SCIENTIFIC EXHIBITS

Rex Room

Biltmore Hotel

9:00 a.m. to 5:00 p.m.
Sunday through Tuesday
Wednesday: 9:00 a.m. to 3:00 p.m.

Scientific Program

CALIFORNIA MEDICAL ASSOCIATION

Ninety-Sixth Annual Session

Biltmore Hotel

LOS ANGELES
April 15 to 19
1967

House of Delegates
Opening Meeting
Saturday, April 15
6:00 p.m.

TABLE OF CONTENTS

Chart of Meeting Times and Places 2
Information 4
Other Meetings 5
Guest Speakers and Participants from States Other than California 7
Index to Participants 8

Special Conferences:

- Occupational Health 11
- Comparative Medicine (California Veterinary Medical Association) 11
- Pre-Convention Radiology 12
- Symposium on Cancer 12
- Pre-Convention Pathology 12
- Disaster Medical Care 13

General Meetings 14

Scientific Assemblies:

- Internal Medicine 16
- General Surgery 18
- General Practice 19
- Allergy 20
- Anesthesiology 21
- Dermatology and Syphilology 22
- Industrial Medicine and Surgery 24
- Obstetrics and Gynecology 25
- Ophthalmology 26
- Orthopedics 27
- Otolaryngology 28
- Pathology and Bacteriology 29
- Pediatrics 30
- Physical Medicine 32
- Preventive Medicine and Public Health 33
- Psychiatry and Neurology 34
- Radiology 35
- Urology 36

Motion Picture Symposia 37
Television Video-Tape Programs 39
Scientific and Organizational Exhibits 40
Woman's Auxiliary 42
Technical Exhibits 43

CMA Organizational Section:

- Photographs of Officers 50
- Members of House of Delegates 51
- Agenda 54
- Proposed Constitutional Amendments 56
- Annual Reports 58

Information

BADGES. It is important that badges be worn at all times. Admission to scientific meetings is by badge only.

COUNCIL. The first meeting of the Council will be held at the Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Friday, April 14 at 4:00 p.m., reconvening Saturday, April 15, 9:00 a.m. to 4:30 p.m., Galeria Room.

DELEGATES. For list of delegates, meeting times, places and agenda, see pages 51 to 56.

EMERGENCY CALLS AND MESSAGES. Convention Emergency Call Number: 213 629-6416—8:30 a.m. to 4:30 p.m., Saturday, April 15 through Wednesday, April 19.

MESSAGE CENTER (213 629-6416)—*Provided through the courtesy of the Pacific Telephone and Telegraph Company*—Registration Desk, Galeria—Open 8:30 a.m. to 4:30 p.m.—The Association will *attempt* to transmit messages to the individual physician. Each physician should notify his own office of the exact times and meetings he plans to attend, and the convention number.

TECHNICAL EXHIBITS—Rex Room. See pages 43 to 49.

SCIENTIFIC EXHIBITS—Rex Room and South Galeria. See pages 40 and 41.

ORGANIZATIONAL EXHIBITS—South Galeria. See page 41.

MOTION PICTURE SYMPOSIA will be shown in the Music Room. See program pages 37 and 38.

TELEVISION VIDEO-TAPE PROGRAMS will be shown in the Music Room. See program page 39.

You are urged to visit and attend all exhibits.

MEETING TIMES AND PLACES. See chart on page 2 for exact times and places of general and section meetings.

REGISTRATION. Registration and information desks are located in the Galeria. *All members, guests and visitors are requested to register immediately on arrival.* There is no charge for registration. Registration desks are open Saturday through Wednesday. *Admission to the general and section sessions and exhibit area is by badge only.* **Members wishing to vote in specialty sections must indicate appropriate section when registering; voting in other sections will not be allowed.**

QUALIFICATIONS/REQUIREMENTS FOR REGISTRATION. (a) All M.D.'s with credentials showing that they hold valid license to practice medicine. (Membership card in C.M.A.; county medical society/association or A.M.A. membership card.) (b) Medical students will be admitted upon presentation of credentials from their medical schools identifying them as medical students. (A membership card of the Student American Medical Association or letter from their dean's office.) (c) Medical assistants will be admitted upon presentation of a letter from the physician-employer or C.M.A.A. membership card. (d) Military paramedical personnel will be admitted upon presentation of a letter requesting their admittance, written by their commanding officer. (e) Dentists (D.D.S.), doctors of veterinary medicine (D.V.M.), registered nurses (R.N.), student nurses, x-ray technicians, laboratory technicians, allied public health personnel, and others will be admitted provided they have proper identification. (f) *All questions on admission will be passed upon by a member of the Committee on Registration who will be present at the desk.*

ACKNOWLEDGMENT

IN KEEPING with the charge made by the California Medical Association to the Annual Scientific Assembly in improving the programs and to provide meaningful information to the members of the Association, the theme of this year's session is *Diagnosis.*

It was the belief of the Committee that the subject of diagnosis is one of common interest of all members of the Association and one which, because of the rapid strides being made in technology, was worthy of a full program.

There has been considerable enthusiasm by the various sections as well as by groups sponsoring a variety of special programs.

We are indeed pleased to make the L. Henry Garland Memorial Lecture a part of future programs of the Association's annual meeting.

The Committee on Scientific Assemblies appreciates greatly the cooperation and assistance of all those who have been asked to help and of the many who have volunteered their assistance. The Committee always welcomes comments and suggestions on additional ways to improve the Assemblies.

Committee on Scientific Assemblies
of the Scientific Board
JOHN B. DILLON, M.D., *Chairman*

Other Meetings and Entertainment

- **SATURDAY, APRIL 15**

CMA HOUSE OF DELEGATES OPENING SESSION—Biltmore Bowl, 6:00 p.m.

CALPAC Meeting—Biltmore Bowl, immediately following the Opening Session of the CMA House of Delegates.

CMA and California Veterinary Medical Association—Symposium on Comparative Medicine—Ballroom, 9:30 a.m. and 2:00 p.m.

California College of Chest Physicians—Conference Room 1, 8:00 a.m. to 5:00 p.m.

California Society of Anesthesiologists—Conference Room 8, 8:00 a.m. to 5:00 p.m.

Conference on Occupational Health—Conference Room 2, 9:00 a.m.

Conference on Occupational Health Luncheon—Place to be announced, 12:15 p.m.

Los Angeles and Metropolitan Dermatological Societies—Good Hope Clinic, 1241 Shatto, Los Angeles, 2:00 p.m.

Los Angeles Branch, American Cancer Society—Symposium on Cancer—Music Room, 2:00 p.m.

Pre-Convention Radiology Conference—Renaissance Room, 9:30 a.m. to 3:30 p.m.

Reference Committee Chairmen Luncheon and Meeting—Conference Room 5, 12:00 noon.

- **SUNDAY, APRIL 16**

CMA PRESIDENTS' RECEPTION honoring the Presidents of the California Medical Association and the Woman's Auxiliary, Renaissance Room, 6:30 p.m.

CMA Section on Allergy and California Society of Allergy Luncheon—Conference Room 4, 12:00 noon.

CMA Section on Pediatrics Luncheon—Rendezvous Room, 12:30 p.m.

California Medical Assistants Association—Conference on Office Management—Conference Room 9, 9:30 a.m.

L. Henry Garland Memorial Lecture—Ballroom, 2:00 p.m.

Medicine and Religion Conference—Galeria Room, 8:00 p.m.

Tumor Tissue Registry/Pathology Conference—Galeria Room, 9:00 a.m. to 5:00 p.m.

- **MONDAY, APRIL 17**

The California Academy of Preventive Medicine Dinner Meeting—Conference Room 2, 6:00 p.m. Advance registration is required; price, $6.75. For information write: Erwin H. Braff, M.D., 101 Grove Street, San Francisco 94102.

California College of Medicine and CMA Section on Internal Medicine—Advances in Gastroenterology—Ballroom, 9:00 a.m.

Yale Alumni Reception—Conference Room 1, 5:30 p.m.

- **TUESDAY, APRIL 18**

American College of Cardiology and CMA Section on Internal Medicine—Galeria Room, 2:00 p.m.

CMA Delegates to AMA meeting—Conference Room 10, 10:00 a.m.

CMA Past Presidents' Luncheon—Conference Room 7, 12:00 noon.

Stanford Alumni Breakfast—Conference Room 1, 7:30 a.m. to 9:00 a.m.

- **WEDNESDAY, APRIL 19**

CMA Section on Internal Medicine and Psychiatry/Neurology—The Impact of Public Medicine—Renaissance Room, 9:00 a.m. and 1:30 p.m.

Guest Speakers

WILLIAM A. ALTEMEIER

IRVINE H. PAGE

HERBERT M. STAUFFER

JON V. STRAUMFJORD, JR.

GEORGE ZUR WILLIAMS

Guest Speakers

William A. Altemeier, M.D., Cincinnati—Chief, Department of Surgery, University of Cincinnati College of Medicine.

Irvine H. Page, M.D., Cleveland—Director, Research Center, Cleveland Clinic.

Herbert M. Stauffer, M.D., Philadelphia—Professor and Chairman, Diagnostic Section, Department of Radiology, Temple University Science Center.

Jon V. Straumfjord, Jr., M.D., Birmingham—Professor and Chairman, Department of Clinical Pathology, Medical College, University of Alabama.

George Zur Williams, M.D., Bethesda—Chief, Clinical Center, National Institutes of Health.

SPECIAL GUESTS OF SECTIONS

Michael R. Deddish, M.D., New York City—Clinician, Sloan-Kettering Institute for Cancer Research, Clinical Associate Professor Surgery, Cornell University Medical College, and Attending Surgeon and Chief of Rectum and Colon Service, Memorial Center for Cancer and Allied Diseases.

Thomas L. Petty, M.D., Denver—Associate Professor of Medicine, Department of Medicine, Colorado University School of Medicine.

James H. Steel, DVM, Atlanta—Chief, Veterinary Public Health Service, Communicable Disease Center.

Louis J. West, M.D., Oklahoma City—Professor and Head, Department of Psychiatry/Neurology in Behavioral Sciences, University of Oklahoma Medical Center.

Index to Participants

GUEST SPEAKERS FROM OUT OF STATE

Altemeier, William Arthur, Cincinnati, Ohio 15
Deddish, Michael, New York City, N.Y. 12
Page, Irvine H., Cleveland, Ohio 15, 17
Petty, Thomas L., Denver, Colo. 21
Stauffer, Herbert M., Philadelphia, Pa. 15, 31, 35, 36
Steele, James H., Atlanta, Ga. 11
Straumfjord, Jon V., Jr., Birmingham, Ala. 14, 16, 22
Williams, George Z., Bethesda, Md. 14, 23, 29, 31, 36

Alexander, Harold B., Santa Barbara 26
Alpern, William M., Chicago 38
Altemeier, William Arthur, Cincinnati 15
Ambrus, Laszlo, Walnut Creek 32
Ancona, Giacomo R., San Francisco 20
Austin, Elizabeth, Los Angeles 39
Ayres, Samuel B., III, Beverly Hills 22

Bailey, Byron, Torrance 28
Baker, Sol R., Beverly Hills 11
Bard, Gregory, San Francisco 32
Barker, Donald E., Van Nuys 13
Barnes, Allan C., Baltimore 38
Barrows, Howard, Los Angeles 39
Becker, Alan H., Long Beach 20
Belt, Elmer, Los Angeles 19, 36
Berk, J. Edward, Los Angeles 16, 39
Beutler, Ernest, Duarte 39
Bierman, Stanley M., Los Angeles 22
Biskind, Gerson R., San Francisco 14
Bittner, Donald L., San Francisco 29
Blum, Henrik L., Martinez 33
Blyth, Donald, Los Angeles 30
Bookman, Ralph, Beverly Hills 20
Bost, Crawford, San Francisco 30
Brayton, Donald W., Los Angeles 39
Brecher, George, San Francisco 14
Brem, Thomas H., Los Angeles 15
Brooksby, Gerald A., Sunnyvale 29
Brunner, Matthew J., Pacific Palisades 23
Budd, John W., Los Angeles 19, 25
Bullock, Weldon K., Los Angeles 12
Busser, R. J., Los Angeles 20
Bustad, Leo K., Davis 11

Cailliet, Rene, Los Angeles 32, 39
Cannon, Jack, Los Angeles 37
Cantor, Edward B., Los Angeles 19, 36
Catz, Boris, Beverly Hills 38
Chaffin, Daniel S., San Rafael 17, 34
Chambers, Leslie, Los Angeles 33
Chan, Thomas, Palo Alto 22
Chang, Lennig, San Francisco 23
Charles, Allan G., Chicago 38
Cheek, David B., San Francisco 21
Cherry, Clifford, Los Angeles 16
Chope, Harold, San Mateo 33
Clark, Dan W., San Jose 29
Clark, William H., Palo Alto 15
Close, J. Robert, Los Angeles 27
Cohen, Melvin R., Chicago 38
Cohen, Sidney, Los Angeles 39
Colachis, Sam C., Los Angeles 32
Collen, Morris, Oakland 15, 33
Collins, Raymond P., San Francisco 24
Comings, David, Duarte 39
Connolly, John E., Los Angeles 18
Cook, Thomas W., Los Angeles 19, 36
Cooper, Irwin, Santa Monica 15
Corday, Eliot, Los Angeles 17
Cross, Gregory H., San Diego 13
Crowley, L. G., Palo Alto 18

Davis, M. Edward, Chicago 38
Davis, W. Clayton, Pittsburgh 37
Datnow, Boris B., Sunnyvale 29
Day, Robert W., Berkeley 30
Dayes, Lloyd A., Los Angeles 39
Deamer, William C., San Francisco 20
Deddish, Michael, New York City 12
Dennis, Robert L., San Jose 29
deVries, Pieter A., San Francisco 29
deWatteville, Hubert, Geneva, Switzerland 38
Ditman, Keith, Los Angeles 39
Dorn, C. Richard, Berkeley 11
Dorsey, John W., Long Beach 19, 36
Dowling, J. Thomas, Seattle 38
Doyle, James R., San Francisco 27
Dresser, James T., Van Nuys 13
Dubois, Edmund L., Los Angeles 22
Dumars, Kenneth, Downey 33
Dyar, Robert, Berkeley 33
Dykstra, Peter C., Los Angeles 26

Eiduson, Samuel, Los Angeles 39
Eisenberg, Ben C., Huntington Park 20
Ellis, Eldon E., Redwood City 33
Elmquist, Homer S., Los Angeles 24
Enelow, Allen J., Los Angeles 17, 34

Farber, Eugene M., Palo Alto 22
Farris, Jack M., Los Angeles 39
Fine, Roswell, Sacramento 30
Fisher, H. Russell, Glendale 14
Fister, H. William, Pasadena 12
Fletcher, Grant, Palo Alto 21
Foster, Paul D., Los Angeles 24
Fox, Leon P., San Jose 19, 25
Fowler, William M., Jr., Los Angeles 32
Frick, Oscar L., San Francisco 20

Gardipee, Charles, Berkeley 30
Gardner, Richard E., San Francisco 37
Gaster, Joseph L., Los Angeles 24
George, Frederick W., III, Long Beach 12
Getz, Richard, Los Angeles 39
Gillespie, Jerry R., Davis 11
Giorgi, Elsie, Los Angeles 33
Gordon, Bernard J., San Francisco 23
Grechman, Richard J., San Francisco 29
Guiss, Lewis, Los Angeles 37
Guth, Paul H., Orange 16, 39
Gwinn, John L., Los Angeles 31, 36

Haddad, Zaky, San Francisco 20
Halloway, Charles K., San Diego 13
Hammond, Denman, Los Angeles 38
Hanafee, William N., Los Angeles 16
Heimlich, Ernest M., Los Angeles 20
Helsper, James T., Los Angeles 12
Henricksen, Erle, Los Angeles 19, 36
Hibbard, Lester T., Los Angeles 19, 25
Hodgman, Joan E., Arcadia 15
Hopper, James, San Francisco 29
Hornberger, Ralph, Berkeley 30
Howe, Gerald E., San Diego 19, 36
Humphrey, George L., Berkeley 11
Hunnicut, Arthur J., Oakland 37
Huse, Wilfred M., Los Angeles 39
Hutch, John A., Pleasant Hill 19, 31, 36
Hutchings, John J., San Francisco 30

Ingbar, Sidney H., Boston 38
Irwin, Lowell E., Los Angeles 11
Iseri, Lloyd T., Downey 20

Jackson, Edwin W., Berkeley 11
Jefferies, Wm. McK., Cleveland 38
Johnson, Olive, Los Angeles 33
Jones, Georgeanna S., Baltimore 38
Jorgensen, Morton C., San Diego 13
Julian, Logan M., Davis 11

Kadish, Arnold, Los Angeles 15
Katz, Edward E., Beverly Hills 20
Kaufman, Herbert S., San Francisco 20
Keller, Thomas, Los Angeles 39
Kirshbaum, Jack D., Encino 12, 29
Kistner, Robert W., Boston 38
Klassen, Paul, Burbank 13
Klein, Russel R., San Rafael 12
Knight, Irving A., Beverly Hills 18
Krupp, Marcus, Palo Alto 15
Kupperman, Herbert S., New York City 38
Kuzman, William J., San Diego 21

Landing, Benjamin H., Los Angeles 12, 29
Larson, C. Philip, Jr., San Mateo 21
Lassiter, George, Long Beach 12
Lee, John, San Francisco 29
Lewis, George B., Jr., Los Angeles 21
Lindberg, Robert B., Ft. Sam Houston 37
Lucas, William, San Diego 19, 25
Luck, J. Vernon, Los Angeles 24

Macer, George, Pasadena 19, 25
MacLaggan, James C., San Diego 13
Maibach, Howard I., San Francisco 23
Mapes, Russell W., Beverly Hills 30
Markland, Colin, Minneapolis 37
Martin, Donald C., Los Angeles 37
Maxwell, John W., Jr., Pasadena 37
May, Ivan, Oakland 37
McCormick, Ruth, Los Angeles 39
McCort, James J., San Jose 12
McDonnel, Gerald M., Los Angeles 15
McHale, James J., San Diego 13
McIntosh, Thomas W., Pasadena 21
Meeker, Irving A., Jr., Los Angeles 37
Meine, Emile, Panorama City 13
Mellinkoff, Sherman, Los Angeles 15
Mendel, Werner M., Los Angeles 17, 34
Michela, Bernard J., Long Beach 32
Mihan, T. Richard, Los Angeles 22
Miller, Charles J., Lynwood 12
Miller, Curtis, Fullerton 15
Moncrief, John A., Fort Sam Houston 37
Monroe, Lee S., La Jolla 16
Monsen, David C. G., Los Angeles 27
Moore, Chester, Long Beach 13
Morris, James M., San Francisco 27
Morton, Daniel G., Los Angeles 19, 36
Moyce, Robert W., San Bernardino 19, 25
Murphy, Stephen P., San Diego 21
Murray, William R., San Francisco 27
Mustacchi, Piero O., San Francisco 20

Nagel, Carl B., Los Angeles 18
Nerenberg, Samuel T., San Francisco 14
Neufeld, Alonzo, Los Angeles 39
Newman, Ben A., Beverly Hills 22
Noble, W. Morris H., San Francisco 38
Noguchi, Thomas T., Los Angeles 29

Olney, Mary B., San Francisco 37

Page, Irvine H., Cleveland 15, 16
Palmer, John M., Palo Alto 19, 36
Paver, Robert L., San Francisco 17, 34
Pearlman, Carl K., Santa Ana 31, 36
Petit, Donald W., Los Angeles 15
Petty, Thomas L., Denver 21
Poenisch, Paul E., San Francisco 28
Powell, Malcolm R., San Francisco 15
Pritchard, William R., Davis 11

Rand, Robert W., Los Angeles 38
Ravenscroft, James, San Diego 38
Ravin, Abe, Denver 37
Reardon, Melva, Burbank 13
Rebhun, Joseph, Pomona 20
Redeker, Allan G., Los Angeles 22
Rickenberg, Robert E., Los Angeles 24
Rigler, Leo, Los Angeles 14, 35
Rochlin, Donald B., Los Angeles 18
Rose, Edward, Philadelphia 38
Rosen, Edgar, Sacramento 17, 30, 34
Rosoff, Leonard, Los Angeles 18
Ross, Joseph R., Los Angeles 11
Rubin, David, Los Angeles 39
Rudnick, Leon R., San Francisco 24

Sabella, Joseph D., San Francisco 20
Sacks, Harry J., Los Angeles 14, 29
Sargent, E. Nicholas, Los Angeles 39
Schade, Frank, Los Angeles 13
Schapiro, Melvin, Encino 16
Schneidman, Harold M., San Francisco 22
Schumer, William, Sacramento 37
Scott, Richard S., Los Angeles 39
Segre, Eugene J., Palo Alto 23
Selzer, Arthur, San Francisco 17
Shapiro, Edward, Beverly Hills 15
Shaw, Edward B., San Francisco 11
Shubin, Herbert, Los Angeles 37
Silverberg, S., Los Angeles 20
Simmer, Hans, Los Angeles 38
Simmons, Daniel, Los Angeles 11
Skaist, Leonard B., Encino 31, 36
Sly, R. Michael, Los Angeles 20
Smith, Lee E., San Diego 13
Smith, Louis L., Loma Linda 18, 39
Smythe, Vernon L., Los Angeles 27
Snyder, William H., Jr., Los Angeles 37
Stauffer, Herbert M., Philadelphia 15, 31, 35, 36

Steele, James H., Atlanta 11
Stein, Justin J., Los Angeles 12
Stempien, Stephen J., Long Beach 16
Stewart, Charles M., Los Angeles 31, 36
Stone, Martin L., New York City 38
Stoney, Ronald J., San Francisco 18
Storrs, Richard P., Los Angeles 12
Straumfjord, Jon V., Jr., Birmingham 14, 16, 22
Strick, Lawrence, Los Angeles 20
Swenson, Byron, Pasadena 35
Swezey, Robert L., Los Angeles 32

Takahashi, Mutsumasa, Los Angeles 35
Tashma, Albert, Los Angeles 24
Teall, Ralph C., Sacramento 33
Teplitz, Raymond, Duarte 39
Thayer, Kent, Long Beach 21
Theilen, Gordon H., Davis 11
Torgerson, Fernando G., Sacramento 30
Tranquada, Robert, Los Angeles 33
Tuffanelli, Denny L., San Francisco 23
Tyler, Edward T., Los Angeles 38
Tyler, Walter S., Davis 11

Van Wyk, Judson J., Chapel Hill 38
von Leden, Hans, Los Angeles 28

Walton, Robert G., Modesto, 22
Wareham, Ellsworth E., Los Angeles 39
Watts, Alan, Sausalito 39
Watts, Malcolm S. M., San Francisco 17, 34
Weidner, William A., Los Angeles 35
Weigen, John F., Palo Alto 12
Weinstein, Morton R., San Francisco 17, 34
Weitzman, Jordan J., Los Angeles 37
West, Louis J., Oklahoma City 17, 34
White, Irving L., Long Beach 28
Whitehouse, Scott, Pasadena 19, 25
Williams, George Z., Bethesda, Md. 14, 23, 29, 31
Wilson, Gabriel H., Los Angeles 35
Wilson, George T., Palo Alto 22
Wood, T. Rodman, Los Angeles 26

Yamate, Minoru, San Jose 20

Zinberg, Sheldon S., La Mirada 16
Zinn, Willard, Los Angeles 37
Zontine, W. J., Lancaster 11

PRE-CONVENTION & CONVENTION
special conferences

SATURDAY, APRIL 15—9:00 a.m.—Conference Room 2, Conference Floor

CONFERENCE ON OCCUPATIONAL HEALTH
THE WORKING MAN'S TOXIC ENVIRONMENT—REAL OR IMAGINED?

The 1967 Conference will deal with the areas of toxicology and various other environmental hazards. The various facets of these problems will be presented by specialists, in such particular fields, pointing out both old and new hazards and their effective control.

As in past years, this will be an invitational conference designed primarily for management people from industry, but will be of interest to physicians.

Program to be anounced.

SATURDAY, APRIL 15—9:30 a.m. to 4:30 p.m.—Ballroom, Galeria Floor

9:30— SPECIAL CONFERENCE ON COMPARATIVE MEDICINE

This Program is presented by the California Veterinary Medical Association and the California Medical Association

Chairman: W. J. Zontine, D.V.M., Lancaster, by invitation

Moderator: Edward B. Shaw, M.D., San Francisco

9:30—**Comparative Physiology and Pathology of Human and Equine Emphysema**—Walter S. Tyler, D.V.M., Ph.D., and Jerry R. Gillespie, D.V.M., Ph.D., both Davis, by invitation

Discussant—Daniel Simmons, M.D., Los Angeles

10:15—**Suggested Indications for Treatment of Persons Exposed to Rabies**—George L. Humphrey, D.V.M., M.P.H., Berkeley, by invitation

10:40—**Salmonella: A Major Public Health and Animal Health Problem**—James H. Steele, D.V.M., M.P.H., Atlanta, Georgia, by invitation

11:05—**The Epidemiology of Cancer in Animals**—C. Richard Dorn, D.V.M., M.P.H., Berkeley, by invitation

Discussants—Sol R. Baker, M.D., Beverly Hills, and Edwin W. Jackson, M.D., Berkeley

2:00— SPECIAL CONFERENCE ON COMPARATIVE MEDICINE
(Continued)

Chairman: W. J. Zontine, D.V.M., Lancaster, by invitation

Moderator: William R. Pritchard, D.V.M., Ph.D., Davis, by invitation

2:00—**Comparative Pathology of Hereditary Muscular Dystrophy of the Domestic Chicken**—Logan M. Julian, D.V.M., Ph.D., Davis, by invitation

2:45—**Summary of Bovine Leukemia Research**—Gordon H. Theilen, D.V.M., Davis, by invitation

Discussant—Lowell E. Irwin, M.D., Los Angeles

3:30—**Radiobiological Studies in Domestic Animals: A Review**—Leo K. Bustad, D.V.M., Ph.D., Davis, by invitation

Discussant—Joseph R. Ross, M.D., Los Angeles

4:15—**Summary**—William R. Pritchard, D.V.M., Davis, by invitation

SATURDAY, APRIL 15—9:30 a.m. to 3:30 p.m.—Renaissance Room, Galeria Floor

Committee on Cancer, California Medical Association

RADIOLOGY CONFERENCE

Chairman: John F. Weigen, M.D., Palo Alto • *Secretary:* Richard P. Storrs, M.D., Los Angeles

---

THERAPY SESSION—9:30 a.m. to 11:30 a.m.

Moderator: Justin J. Stein, M.D., Los Angeles

A panel of radiation therapists will discuss cases representative of common therapeutic problems. Questions and comments from the floor will be welcomed.

DIAGNOSTIC SESSION—1:30 p.m. to 3:30 p.m.

Moderator: John F. Weigen, M.D., Palo Alto

Presentation of diagnostic cases, with histories and films, selected to illustrate specific problems in radiological diagnosis or differential diagnosis of cancer. Audience participation and discussion are urgently requested.

3:30—**California Radiological Society Meeting**—James J. McCort, M.D., San Jose, Presiding

---

SATURDAY, APRIL 15—2:00 p.m.—Music Room, Galeria Floor

2:00— **SYMPOSIUM ON CANCER**

THE TIMELY DESCRYING OF COLORECTAL CANCER: EARLY DIAGNOSIS AND TREATMENT

This program is presented by the Professional Education Committee of the American Cancer Society, Los Angeles Branch, in conjunction with the California Medical Association

2:00—Welcome—James T. Helsper, M.D., Chairman, Professional Education Committee, American Cancer Society, Los Angeles Branch, Los Angeles

2:05— **Symposium**

Moderator: Michael Deddish, M.D., New York, by invitation

Members of the Symposium: Weldon K. Bullock, M.D., Los Angeles; Captain Frederick W. George, III, M.C., U.S.N., and Lieutenant Commander George Lassiter, M.C., U.S.N.R., both Long Beach by invitation; Russel R. Klein, M.D., San Rafael; H. William Fister, M.D., Pasadena, and Charles J. Miller, M.D., Lynwood

- **Colorectal Polyps and Their Relationship to Cancer**—Weldon K. Bullock, M.D., Los Angeles
- **The Role of Radiology in the Diagnosis and Management of Colorectal Cancer**—Capt. Frederick W. George, III, M.C., U.S.N., and Lt. Commander George Lassiter, M.C., U.S.N.R., both Long Beach, by invitation
- **Early Metastasis in Colorectal Malignant Polyps**—Russel R. Klein, M.D., San Rafael

SUNDAY, APRIL 16—9:00 a.m. to 5:00 p.m.—Galeria Room, Galeria Floor

Committee on Cancer, California Medical Association

PATHOLOGY CONFERENCE

9:00 a.m. to 12:00 Noon—2:00 p.m. to 5:00 p.m.

PRE-CONVENTION CONFERENCE ON CHILDHOOD TUMORS

Moderator: Benjamin H. Landing, M.D., Director of Laboratory, Los Angeles County Hospital, Los Angeles

Chairman: Jack D. Kirshbaum, M.D., Encino

Those who wish slides and protocols for the Conference (cost, $25.00) are requested to register with Weldon K. Bullock, M.D., Registrar, Tumor Tissue Registry, CMA Committee on Cancer, Los Angeles County Hospital, 1200 North State Street, Los Angeles 90033. The $25.00 fee includes registration. For those who wish to attend the Conference, the fee is $25.00. There is no charge for residents and interns.

MONDAY, APRIL 17—9:00 a.m.—Conference Room 1, Conference Room Floor

DISASTER MEDICAL CARE

Theme: Civilian and Military Care of Mass Casualties

General Chairman: Donald E. Barker, M.D., Van Nuys

9:00—**Outline of the Conference**—Donald E. Barker, M.D., Van Nuys

9:05—**Welcome**—James C. MacLaggan, M.D., President, California Medical Association, San Diego

9:10— **Panel Discussion**

Mass Casualty Care: Disaster Cause, Civilian Concepts

Moderator: Wayne P. Chesbro, M.D., Berkeley

Members of the Panel: Frank Schade, M.D., Los Angeles; Chester Moore, D.D.S., Long Beach, by invitation; Emile Meine, M.D., Panorama City; James T. Dresser, M.D., Van Nuys; Paul Klassen, M.D., Burbank, and Melva Reardon, R.N., Burbank, by invitation

9:15—**Community Planning and Organization for Disaster Medical Care—An Outline**—Frank Schade, M.D., Los Angeles

9:30—**Alerting Procedures and the Concept of Adequate Communications**—Chester Moore, D.D.S., Long Beach, by invitation

9:45—**Disaster Site Care**—Emile Meine, M.D., Panorama City

10:00—**Transportation, Means and Methods**—James T. Dresser, M.D., Van Nuys

10:15—**Hospital Organization for Mass Casualty Reception: Triage, Definitive Care**—Paul Klassen, M.D., Burbank and Melva Reardon, R.N., Burbank, by invitation

10:35—**Discussion**

11:00— **Panel Discussion**

Mass Casualty Care: War Cause, United States Armed Forces Concept, Vietnam

Moderator: Captain Charles K. Halloway, M.C., U.S. Navy, San Diego, by invitation

ALL PANEL MEMBERS ARE VETERANS OF THE VIETNAM WAR AND ARE WELL ACQUAINTED WITH MASS CASUALTY CARE IN MODERN WARFARE

Members of the Panel: Lieutenant Lee E. Smith, M.C., U.S.N.; Lieutenant Commander James J. McHale, M.C., U.S.N.; Commander Gregory H. Cross, M.C., U.S.N., and Lieutenant Commander Morton C. Jorgensen, M.C., U.S.N., all San Diego, by invitation

11:05—**Organization and Planning for Casualty Care**—Lt. Lee E. Smith, M.C., U.S.N., San Diego, by invitation

11:15—**Casualty Care at the Combat Site**—Lt. Commander James J. McHale, M.C., U.S.N., San Diego, by invitation

11:25—**Transportation, Means and Methods**—Commander Ervin L. Lobpreis, M.C., U.S.N., San Diego, by invitation

11:35—**Hospital Organization and Procedures for Casualty Reception: Triage, Definitive Care**—Lt. Commander Morton C. Jorgensen, M.C., U.S.N., San Diego, by invitation

11:45—**Discussion**

MESSAGE CENTER

MESSAGE CENTER (213 629-6416)—*Provided through the courtesy of the Pacific Telephone and Telegraph Company*—Registration Desk, Galeria—Open 8:30 a.m. to 4:30 p.m.—The Association will *attempt* to transmit messages to the individual physician. Each physician should notify his own office of the exact times and meetings he plans to attend, and the convention number.

Scientific Sessions

■ GENERAL MEETINGS

FIRST GENERAL MEETING

SUNDAY, APRIL 16
9:00 a.m.—Ballroom, Galeria Floor

Advances in Diagnostic Techniques

Moderator: H. Russell Fisher, M.D., Glendale

9:00—**Current Clinical Applications of Enzyme Tests**—Harry J. Sacks, M.D., Los Angeles

9:20—**Identification of the Dysproteinemias**—Samuel T. Nerenberg, M.D., San Francisco

9:45—**Reliability Judgments of Laboratory Results**—Jon V. Straumfjord, Jr., M.D., Birmingham, Alabama, by invitation

10:15—**Recess**

10:25—**The Clinical Significance of Gonadotropic Hormones in Trophoblastic Disease**—Gerson R. Biskind, M.D., San Francisco

10:40—**The Hematologic Control of Drug Therapy**—George Brecher, M.D., San Francisco, by invitation

11:00—**The Impact of Automation on Laboratory Diagnosis**—George Z. Williams, M.D., Bethesda, Maryland, by invitation

11:30— **Panel Discussion**

The Role of Automated Laboratory Determinations In Medical Practice

Moderator: George Z. Williams, M.D., Bethesda, Maryland, by invitation

Members of the Panel: Gerson R. Biskind, M.D., San Francisco; George Brecher, M.D., San Francisco, by invitation; Jon V. Straumfjord, Jr., M.D., Birmingham, Alabama, by invitation

SUNDAY, APRIL 16

2:00 p.m.—Ballroom, Galeria Floor

SPECIAL GENERAL MEETING

First Annual L. Henry Garland Memorial Lecture

Sponsored by the California Radiological Society

2:00

The Radiologist and the Search for Hidden Cancer—Leo Rigler, M.D., Los Angeles

3:00

Recess

3:15

Business Meeting and Annual Meeting of California Radiological Society

SECOND GENERAL MEETING

MONDAY, APRIL 17
2:00 p.m.—Ballroom, Galeria Floor

Advances in Radiology

Moderator: Gerald M. McDonnel, M.D., Los Angeles

2:00—**Advances in Radiological Diagnostic Techniques**—Herbert M. Stauffer, M.D., Philadelphia, Pennsylvania, by invitation

2:30—**Advances in Isotope Diagnosis**—Malcolm R. Powell, M.D., San Francisco

3:00—**Advances in Diagnosis in Occupational Medicine**—Curtis Miller, M.D., Fullerton

3:30—**Recess**

3:45—**Advances in Continuous Metabolic Monitoring**—Arnold Kadish, M.D., Los Angeles

4:15—**Advances in Mathematical Modeling in Monitoring and Diagnosis**—Irwin Cooper, Ph.D., Santa Monica, by invitation

4:45—**Question and Answer Period with Participants**

THIRD GENERAL MEETING

TUESDAY, APRIL 18
9:30 a.m.—Ballroom, Galeria Floor

The Clinician's Response to New Aspects of Laboratory Medicine

Moderator: Donald W. Petit, M.D., Los Angeles

9:30—**The Impact of the Laboratory on Clinical Medicine**—Irvine H. Page, M.D., Cleveland, Ohio, by invitation

10:00—**Discriminate or Indiscriminate Use of the Laboratory?**—Thomas H. Brem, M.D., Los Angeles

10:15—**New Laboratory Aids in Surgical Practice**—William A. Altemeier, M.D., Cincinnati, Ohio, by invitation

11:00—**Recess**

11:10— **Panel Discussion**

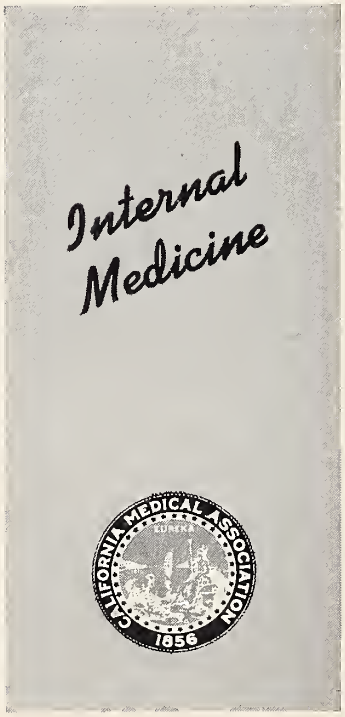
The Complete Examination—Why?—
What Laboratory Aid?

Moderator: Sherman Mellinkoff, M.D., Los Angeles

Members of the Panel: William H. Clark, M.D., and Marcus Krupp, M.D., Palo Alto; Morris Collen, M.D., Oakland; Joan E. Hodgman, M.D., Arcadia; and Edward Shapiro, M.D., Beverly Hills

ROBERT L. PAVER
Chairman

JOHN L. BENTON, JR.
Secretary



INTERNAL MEDICINE

Chairman.............ROBERT L. PAVER, M.D., San Francisco
Secretary............JOHN L. BENTON, JR., M.D., Los Angeles
Assistant Secretary...........EDGAR ROSEN, M.D., Sacramento

MONDAY, APRIL 17 **9:00 a.m.—Ballroom**
Galeria Floor

This Program is Presented by the Department of Medicine, University of California, California College of Medicine

9:00— Advances in Gastroenterology

Chairman: J. Edward Berk, M.D., Chairman, Department of Medicine, University of California, California College of Medicine, Los Angeles

Following each presentation there will be a twelve-minute Discussion Period. Microphones will be set up throughout the auditorium so that the audience may ask questions.

9:00—**Esophagus**—Melvin Schapiro, M.D., Encino
Discussion

9:27—**Stomach and Duodenum**—Stephen J. Stempien, M.D., Long Beach
Discussion

9:54—**Small and Large Intestine**—Paul H. Guth, M.D., Orange
Discussion

10:21—**Recess**

10:30—**Liver**—Lee S. Monroe, M.D., La Jolla
Discussion

10:57—**Extrahepatic Biliary Tract**—Sheldon S. Zinberg, M.D., La Mirada
Discussion

11:24—**Pancreas**—J. Edward Berk, M.D., Los Angeles
Discussion

11:51—**Business Meeting**

TUESDAY, APRIL 18 **2:00 p.m.—Galeria Room**
Galeria Floor

This Program is Being Presented Jointly by the Section on Internal Medicine and the American College of Cardiology

Moderator: Clifford Cherry, M.D., Los Angeles

2:00—**Serum Enzymes in the Differential Diagnosis of Myocardial Infarction and Myocarditis**—Jon V. Straumfjord, Jr., M.D., Birmingham, Alabama, by invitation

2:30—**Evaluation of Coronary Arteriography**—William N. Hanafee, M.D., Los Angeles

3:00—**Recess**

3:15—Differential Diagnostic Procedures in Hypertension —Irvine H. Page, M.D., Cleveland, Ohio, by invitation

3:45—Measurement of the Dynamics of Myocardial Competence—Arthur Selzer, M.D., San Francisco

4:15—Specific Evidences of Myocardial Infarction in Electrocardiography and Vectorcardiography — Eliot Corday, M.D., Los Angeles

4:45—Small Group Discussions

WEDNESDAY, APRIL 19 9:00 a.m.—Renaissance Room
Galeria Floor

Combined Meeting with Section on Psychiatry and Neurology

9:00— **The Impact of Public Medicine**

General Chairman: Robert L. Paver, M.D., San Francisco

9:00—Aid to the Totally Disabled: The Pitfalls as Seen by a Psychiatrist—Morton R. Weinstein, M.D., San Francisco

9:30—Discussion—Daniel S. Chaffin, M.D., San Rafael

9:45— **Panel Discussion**

The Role of Medicine in Society

Moderator: Malcolm S. M. Watts, M.D., San Francisco

Members of the Panel: To be announced

11:00— **Panel Discussion**

Regional Medical Programs on Heart, Stroke and Cancer

Moderator: To be announced

Members of the Panel: To be announced

12:00—Recess

WEDNESDAY, APRIL 19 1:30 p.m.—Renaissance Room
Galeria Floor

Combined Meeting with Section on Psychiatry and Neurology

The Impact of Public Medicine
(Continued)

Chairman: Werner M. Mendel, M.D., Los Angeles

1:30—Meaning of Changing Emphasis on Community Mental Health to the Practicing Physician—Allen J. Enelow, M.D., Los Angeles and Louis J. West, M.D., Oklahoma City, by invitation

2:30—Psychiatry and Urology, Chairman's Address—Werner M. Mendel, M.D., Los Angeles

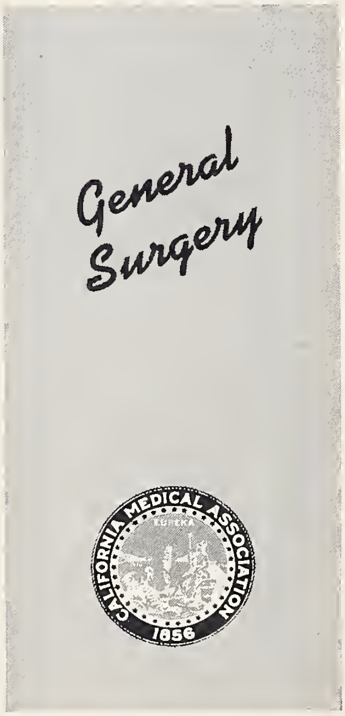
2:45—A Critical Look at Medicare—Edgar Rosen, M.D., Sacramento

ATTENTION:

CHECK TELEVISION AND
MOTION PICTURE
PROGRAMS, PAGES 39-41

SEIBERT PEARSON
Chairman

ALBERT G. CLARK
Secretary



GENERAL SURGERY

Chairman...............Seibert Pearson, m.d., Long Beach
Secretary.............Albert G. Clark, m.d., San Francisco
Assistant Secretary..........Robert Hood, m.d., Los Angeles

MONDAY, APRIL 17 **2:00 p.m.—Renaissance Room Galeria Floor**

2:00— **Panel Discussion**

The Management of Surgical Infections

Moderator: John E. Connolly, M.D., Los Angeles

Members of the Panel: To be announced

3:30— **Panel Discussion**

The Surgical Aspects of Shock

Moderator: Leonard Rosoff, M.D., Los Angeles

Members of the Panel: To be announced

TUESDAY, APRIL 18 **2:00 p.m.—Renaissance Room Galeria Floor**

2:00— **Panel Discussion**

Cancer Chemotherapy

Moderator: Donald B. Rochlin, M.D., Los Angeles

Members of the Panel: To be announced

3:30—**The Management of Occlusive Disease of the Vessels of the Aortic Arch**—Louis L. Smith, M.D., Loma Linda

3:50—**Evaluation of Carotid Endarterectomy for Transient Cerebral Ischemia**—Ronald J. Stoney, M.D., San Francisco

4:15—**The Repair of Large Incisional Hernias**—Irving A. Knight, M.D., Beverly Hills

4:35—**Clinical Experiences with Jejunal Interposition for Post-Gastrectomy Syndrome**—Carl B. Nagel, M.D., Los Angeles

4:55—**Forequarter Amputation for Extensive Metastatic Axillary Squamous Cell Carcinoma**—L. G. Crowley, M.D., Palo Alto

5:10—**Business Meeting**

ATTENTION:

CHECK TELEVISION AND MOTION PICTURE PROGRAMS, PAGES 39-41

GENERAL PRACTICE

Chairman....................J. Blair Pace, m.d., Oceanside
Secretary...........Alfred F. Kandlbinder, m.d., Monterey
Assistant Secretary...Allen F. Sterling, m.d., San Bernardino

MONDAY, APRIL 17 **9:00 a.m.—Galeria Room**
Galeria Floor

Combined Meeting with Section on Urology

9:00—**Some Ideas on Stress Incontinence in the Female**—John A. Hutch, M.D., Pleasant Hill

9:30—**Discussion**—Edward B. Cantor, M.D., and Erle Henriksen, M.D., Los Angeles

9:40— **Panel Discussion**

The Diagnosis and Treatment of Urinary Tract Infections in Females

Moderator: Elmer Belt, M.D., Los Angeles

Members of the Panel: Daniel G. Morton, M.D., and Thomas W. Cook, M.D., both Los Angeles; John M. Palmer, M.D., Palo Alto; John W. Dorsey, M.D., Long Beach; and Gerald E. Howe, M.D., San Diego

10:40—**Recess**

10:50— **Panel Discussion**

Management of Urologic Injuries in Gynecologic Surgery

Moderator: Elmer Belt, M.D., Los Angeles

Members of the Panel: Daniel G. Morton, M.D., and Thomas W. Cook, M.D., both Los Angeles; John M. Palmer, M.D., Palo Alto; John W. Dorsey, M.D., Long Beach; and Gerald E. Howe, M.D., San Diego

MONDAY, APRIL 17 **2:00 p.m.—Galeria Room**
Galeria Floor

Combined Meeting with Section on Obstetrics and Gynecology

2:00— Conference on Maternal Mortality

Moderator: Leon P. Fox, M.D., San Jose

Members of the Panel: Scott Whitehouse, M.D., Pasadena; John W. Budd, M.D., Los Angeles; Robert W. Moyce, M.D., San Bernardino; and Commander William Lucas (MC), United States Navy, San Diego, by invitation

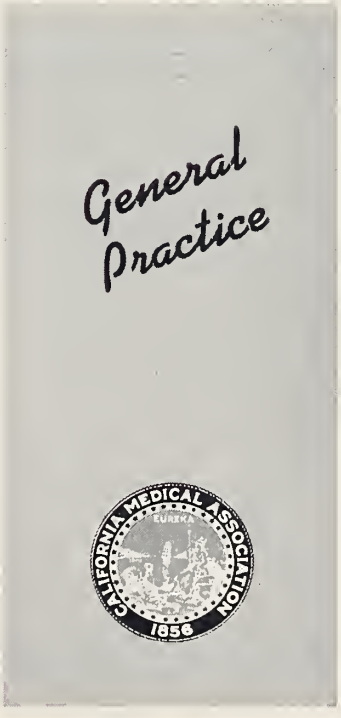
1. **A Case of Obstetrical Hemorrhage**
2. **A Case of Septic Shock**

3:00—**A Contract Called Marriage**—George Macer, M.D., Pasadena

3:30—**Chairman's Address**—Lester T. Hibbard, M.D., Los Angeles

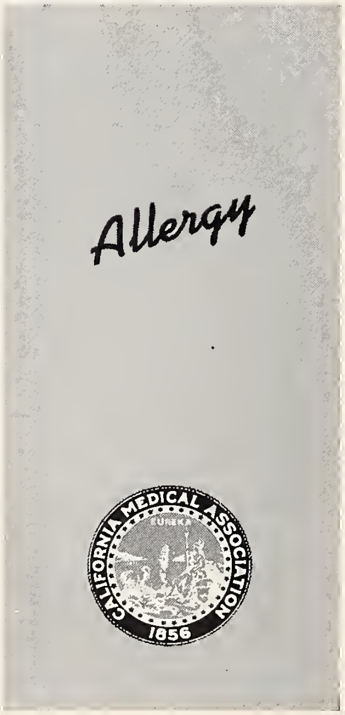
J. BLAIR PACE
Chairman

ALFRED F. KANDLBINDER
Secretary



LEO N. MELEYCO
Chairman

ERNEST J. SASLOW
Secretary



ALLERGY

Chairman.................LEO N. MELEYCO, M.D., San Jose
Secretary................ERNEST J. SASLOW, M.D., Bakersfield
Assistant Secretary..............SAMUEL ROSS, M.D., Fresno

SUNDAY, APRIL 16 9:30 a.m.—Conference Room 4
Conference Room Floor

9:30—**Quantitative Determination of Complement Levels in Normal and Atopic Subjects**—Herbert S. Kaufman, M.D., and Oscar L. Frick, M.D., San Francisco

9:50—**Judicial Use of Antitussives in Asthmatic Patients**—Joseph Rebhun, M.D., Pomona

10:10—**Antibody Response in Relationship to Fever**—Minoru Yamate, M.D., San Jose

10:30—**Exercise-Induced Bronchospasm: Evaluation of Isoproterenol, Phenylphreine and the Combination**—R. Michael Sly, M.D., Los Angeles, by invitation; Ernest M. Heimlich, M.D., and Lawrence Strick, M.D., Los Angeles; and R. J. Busser, B.S., Los Angeles, by invitation

10:50—**Effect of Ambient Cold Air Breathing on Childhood Asthmatics**—Ernest M. Heimlich, M.D., Lawrence Strick, M.D., and Seymour Silverberg, M.D., Los Angeles; and R. J. Busser, B.S., Los Angeles, by invitation

11:10—**The Confused World of Chronic Lung Disease**—Ralph Bookman, M.D., and Edward E. Katz, M.D., Beverly Hills

12:00—**Luncheon and Business Meeting with the California Society of Allergy**

SUNDAY, APRIL 16 2:30 p.m.—Conference Room 4
Conference Room Floor

2:30—**Milk Intolerance: Allergy or Enzyme Deficiency?**—Piero O. Mustacchi, M.D., Giacomo R. Ancona, M.D., and Joseph D. Sabella, M.D., San Francisco

3:00—**Scratch Test Reactions to Human Dander in Atopic and Non-atopic Individuals**—Zaky Haddad, M.D., San Francisco, by invitation

3:30—**Some Clinical Aspects of Molds Allergy in Children**—William C. Deamer, M.D., San Francisco

4:00—**Endocardial Fibroelastosis and Asthma**—Ben C. Eisenberg, M.D., Huntington Park; and Lloyd T. Iseri, M.D., Downey

4:30—**Cold Urticaria**—Alan H. Becker, M.D., Long Beach

ANESTHESIOLOGY

Chairman............Thomas W. McIntosh, m.d., Pasadena
Secretary...............Donald B. Dose, m.d., San Diego
Assistant Secretary.......Donald A. Walker, m.d., Tiburon

SUNDAY, APRIL 16 **9:30 a.m.—Conference Room 1**
Conference Room Floor

9:30—**Diagnosis and Significance of Cardiac Arrhythmias During Anesthesia**—William J. Kuzman, M.D., San Diego

9:55—**Diagnosis of Hypovolemia During Anesthesia**—C. Philip Larson, Jr., M.D., San Mateo

10:20—**Dangers of Continued Hearing During General Chemoanesthesia**—David B. Cheek, M.D., San Francisco

10:45—**Pathophysiology of Drowning**—Stephen P. Murphy, M.D., San Diego

11:10—**Prolonged Mechanical Ventilation of the Newborn —Movie Comment**—George B. Lewis, Jr., M.D., Los Angeles

11:35—**Chairman's Address**—Thomas W. McIntosh, M.D., South Pasadena

11:55—**Business Meeting**

SUNDAY, APRIL 16 **2:00 p.m.—Conference Room 1**
Conference Room Floor

2:00—**The Anesthesiologist and the Intensive Care Unit**—Grant Fletcher, M.D., Palo Alto

2:30—**The Respiratory Intensive Care Unit**—Thomas L. Petty, M.D., Denver, Colorado, by invitation

3:15—**Recess**

3:30—**The Allergist and the Intensive Care Unit**—Kent Thayer, M.D., Long Beach

4:00— **Panel Discussion**

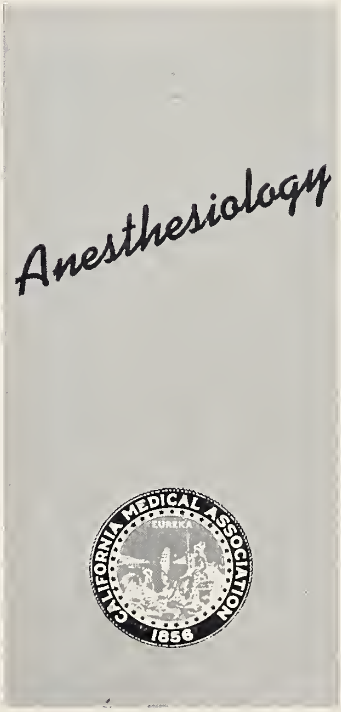
Intensive Care Unit

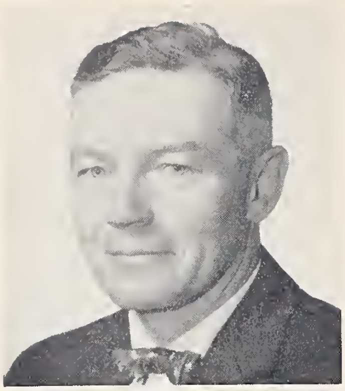
Moderator: William J. Kuzman, M.D., San Diego

Members of the Panel: Grant Fletcher, M.D., Palo Alto; and Thomas L. Petty, M.D., Denver, Colorado, by invitation

THOMAS W. McINTOSH
Chairman

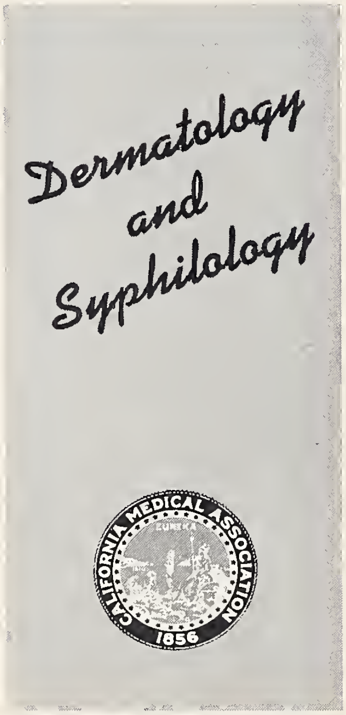
DONALD B. DOSE
Secretary





ROBERT G. WALTON
Chairman

SAMUEL AYRES, III
Secretary



DERMATOLOGY

Chairman...............Robert G. Walton, m.d., Modesto
Secretary.............Samuel Ayres, III, m.d., Beverly Hills
Assistant Secretary........George T. Wilson, m.d., Palo Alto

SATURDAY, APRIL 15 **2:00 p.m.—Good Hope Clinic**
1241 Shatto Street

Preconvention Meeting

There will be a clinical meeting of the combined Los Angeles and Metropolitan Dermatological Societies at 2:00 p.m. The meeting will be held at the Good Hope Clinic, 1241 Shatto Street, Los Angeles. Discussion of the cases will take place at 3:00 p.m. All dermatologists are invited by the Los Angeles and Metropolitan Dermatological Societies.

SUNDAY, APRIL 16 **9:00 a.m.—Convention Hall**
Embassy Auditorium
847 South Grand Avenue

9:00—**Skin Testing for Drug Sensitivity**—Harold M. Schneidman, M.D., San Francisco

9:20—**Nail Changes as a Diagnostic Aid in Systemic and Cutaneous Diseases**—Ben A. Newman, M.D., Beverly Hills

9:40—**The Proper Evaluation of the LE Phenomena—Difficulties and Pitfalls to Avoid**—Edmund L. Dubois, M.D., Los Angeles

10:10—**Stretch Garment Dermatitis**—T. Richard Mihan, M.D., Los Angeles, and Samuel B. Ayres, III, M.D., Beverly Hills

10:25—**Recess**

10:40—**Serum Electrophoretic and Isozyme Patterns in Cutaneous Medicine**—Jon V. Straumfjord, Jr., M.D., Birmingham, Alabama, by invitation

11:15—**Mycosis Fungoides: Diagnosis, Treatment and Pathogenesis**—Stanley M. Bierman, M.D., Los Angeles

11:35—**Diagnosis with the Touch Smear**—George T. Wilson, M.D., Palo Alto

11:55—**Porphyria: Office Screening Tests and the Proper Use of the Clinical Laboratory**—Allan G. Redeker, M.D., Los Angeles

SUNDAY, APRIL 16 **1:30 p.m.—Convention Hall**
Embassy Auditorium
847 South Grand Avenue

1:30—**Chairman's Address**—Robert G. Walton, M.D., Modesto

1:45—**Arthropathies in Psoriasis**—Eugene M. Farber, M.D., Palo Alto, and Thomas Chan, M.D., Palo Alto, by invitation

2:05—Investigation of the Hirsute Female—Eugene J. Segre, M.D., Palo Alto, by invitation

2:25—Useful Tests in the Diagnosis of Problems of Sweating—Bernard J. Gordon, M.D., and Howard I. Maibach, M.D., San Francisco

2:45—The Latest in Serological Tests for Syphilis—Lennig Chang, M.D., San Francisco, by invitation

3:15—Recess

3:30—Use of Bacteriology and Mycology in Diagnosis and Therapy—George Z. Williams, M.D., Bethesda, Maryland, by invitation

4:05—Helpful Diagnostic Tests in Collagen Disease—Denny L. Tuffanelli, M.D., San Francisco

4:25—Examination of the Hair in the Diagnosis of Alopecia—Matthew J. Brunner, M.D., Pacific Palisades

4:45—Business Meeting

VISIT
SCIENTIFIC
AND
TECHNICAL
EXHIBITS

FOR YOUR LEISURE LISTENING

Hear Audio-Digest Tapes of other major
Scientific meetings
Music Room Foyer, Galeria Floor
Daily

BRING PROPER IDENTIFICATION FOR REGISTRATION

INDUSTRIAL MEDICINE AND SURGERY

Chairman............LEON R. RUDNICK, M.D., San Leandro
Secretary............RAYMOND O. OJA, M.D., San Francisco
Assistant Secretary..JAMES G. FORGERSON, M.D., San Francisco

LEON R. RUDNICK
Chairman

WEDNESDAY, APRIL 19 9:00 a.m.—Conference Room 4
Conference Room Floor

9:00—**Chairman's Address**—Leon R. Rudnick, M.D., San Leandro

9:15—**Ophthalmology for the Industrial Physician**—Albert Tashma, M.D., Los Angeles

9:45—**Difficulties in the Evaluation of Lead Poisoning**—Raymond P. Collins, M.D., San Francisco

10:15—**Recess**

10:30—**Effective Radiographic Examination**—Robert E. Rickenberg, M.D., Los Angeles

11:00—**Guidelines to Diagnosis and Therapy in Dermatology**—Paul D. Foster, M.D., Los Angeles

WEDNESDAY, APRIL 19 2:00 p.m.—Conference Room 4
Conference Room Floor

2:00—**Management of Orthopedic Problems in an Industrial Practice**—J. Vernon Luck, M.D., Los Angeles

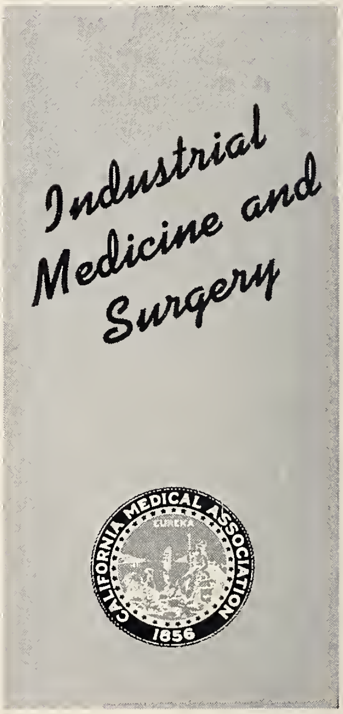
2:30—**Treatment of Depressed Fractures of the Malar Bone**—Homer S. Elmquist, M.D., Los Angeles

3:00—**Hernioplasty Under Local Anesthetic**—Joseph L. Gaster, M.D., Los Angeles

3:30—**Business Meeting and Election of Officers**

RAYMOND O. OJA
Secretary

VISIT SCIENTIFIC AND
TECHNICAL EXHIBITS


Industrial Medicine and Surgery
California Medical Association
Eureka
1856

OBSTETRICS AND GYNECOLOGY

Chairman.............LESTER T. HIBBARD, M.D., Los Angeles
Secretary..............WARREN E. JONES, M.D., Sacramento
Assistant Secretary...JAMES W. RAVENSCROFT, M.D., San Diego

LESTER T. HIBBARD
Chairman

MONDAY, APRIL 17 **2:00 p.m.—Galeria Room**
Galeria Floor

Combined Meeting with Section on General Practice

2:00— Conference on Maternal Mortality

Moderator: Leon P. Fox, M.D., San Jose

Members of the Panel: Scott Whitehouse, M.D., Pasadena; John W. Budd, M.D., Los Angeles; Robert W. Moyce, M.D., San Bernardino; and Commander William Lucas (MC), United States Navy, San Diego, by invitation

1. A Case of Obstetrical Hemorrhage
2. A Case of Septic Shock

3:00—A Contract Called Marriage—George Macer, M.D., Pasadena

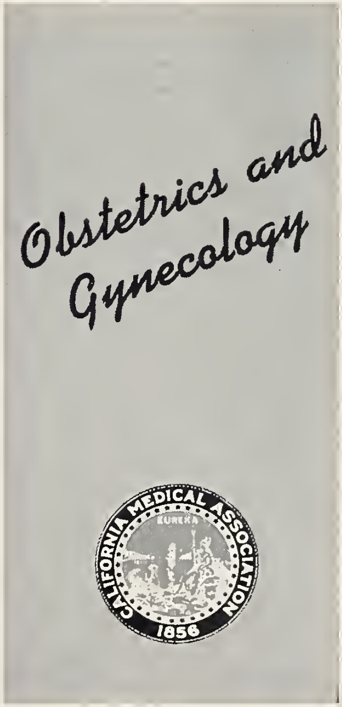
3:30—Chairman's Address—Lester T. Hibbard, M.D., Los Angeles

4:00—Business Meeting

WARREN E. JONES
Secretary

ATTENTION:

CHECK TELEVISION AND MOTION PICTURE PROGRAMS, PAGES 39-41



MESSAGE CENTER

MESSAGE CENTER (213 629-6416)—*Provided through the courtesy of the Pacific Telephone and Telegraph Company*—Registration Desk, Galeria—Open 8:30 a.m. to 4:30 p.m.—The Association will *attempt* to transmit messages to the individual physician. Each physician should notify his own office of the exact times and meetings he plans to attend, and the convention number.

OPHTHALMOLOGY

Chairman..........Victor G. Fellows, M.D., San Francisco
Secretary.............Wendell C. Irvine, M.D., Los Angeles
Assistant Secretary........Robert E. Hopkins, M.D., Stockton

VICTOR G. FELLOWS
Chairman

WENDELL C. IRVINE
Secretary

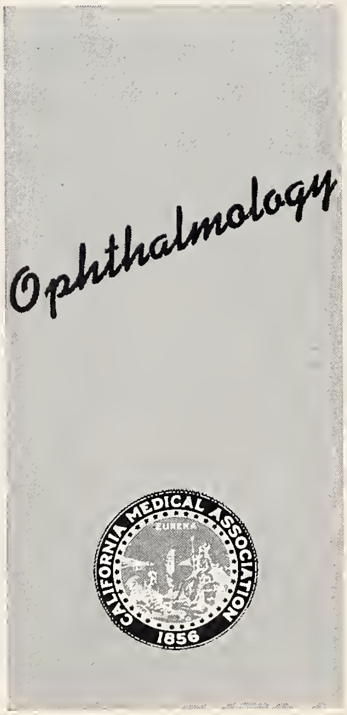
SUNDAY, APRIL 16 — **2:00 p.m.—Assembly Hall**
Embassy Auditorium
847 South Grand Avenue

2:00—**Advances in the Cytological Diagnosis of Epibulbar Carcinoma in Situ**—Peter C. Dykstra, M.D., Los Angeles

2:30—**Differential Diagnosis of Virus Conjunctivitis**—T. Rodman Wood, M.D., Los Angeles, by invitation

3:00—**Diagnostic Features of Vascular Lesions Affecting the Optic Pathways**—Harold B. Alexander, M.D., Santa Barbara

3:30—**Business Meeting**



REGISTRATION

Registration and information desks are located in the Galeria. *All members, guests, and visitors are requested to register immediately on arrival.* There is no charge for registration. Registration desks are open Saturday through Wednesday. *Admission to the general and section sessions and exhibit area is by badge only.*

Members wishing to vote in specialty sections must indicate appropriate section when registering; voting in other sections will not be allowed.

FOR YOUR LEISURE LISTENING

Hear Audio-Digest Tapes of other major Scientific meetings

Music Room Foyer, Galeria Floor
Daily

ORTHOPEDICS

Chairman..............David C. Monsen, m.d., Los Angeles
Secretary..........Arthur R. Hartwig, m.d., San Francisco
Assistant Secretary....Vernon L. Smythe, m.d., Los Angeles

SUNDAY, APRIL 16 **9:15 a.m.—Assembly Hall**
Embassy Auditorium
847 South Grand Avenue

9:15—**Common Tumors of the Hand: Diagnosis and Treatment**—James R. Doyle, M.D., San Francisco

9:40—**Surgical Indications and Pre-Operative Management in Rheumatoid Arthritis**—William R. Murray, M.D., San Francisco

10:05—**Fatigue Fractures**—James M. Morris, M.D., San Francisco

10:30—**Recess**

10:45—**Sources and Uses of Bioelectric Phenomena**—J. Robert Close, M.D., Los Angeles

11:10—**Differential Diagnosis of Fat Embolism**—Vernon L. Smythe, M.D., Los Angeles

11:35—**Chairman's Address: Giant Cell Tumor in an Unusual Location**—David C. G. Monsen, M.D., Los Angeles

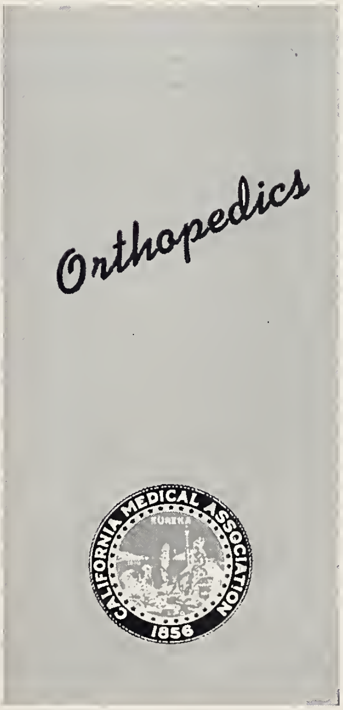
12:00—**Business Meeting**

DAVID C. MONSEN
Chairman

ARTHUR R. HARTWIG
Secretary

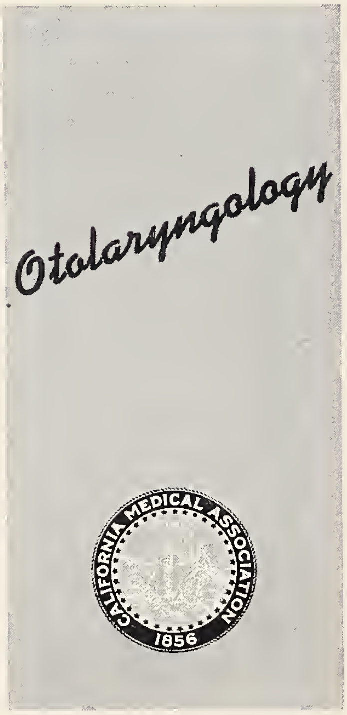
ATTENTION:

CHECK TELEVISION AND MOTION PICTURE PROGRAMS, PAGES 39-41



G. HOWARD GOTTSCHALK
Chairman

CHARLES P. LEBO
Secretary



OTOLARYNGOLOGY

Chairman........G. Howard Gottschalk, m.d., Los Angeles
Secretary..............Charles P. Lebo, m.d., San Francisco
Assistant Secretary........Irving L. White, m.d., Long Beach

SUNDAY, APRIL 16 **9:30 a.m.—Conference Room 8**
Conference Room Floor

9:30—**Indications for Cryosurgery in Otolaryngology**—Hans von Leden, M.D., Los Angeles

10:00—**Conservation Surgery in Carcinoma of the Laryngeal Anterior Commissure**—Byron Bailey, M.D., Torrance

10:30—**Endoscopic Resection of Zenker's Diverticulum**—Irving L. White, M.D., Long Beach

11:00—**Clinical Applications of Electronystagmography**—Paul E. Poenisch, M.D., San Francisco

11:30—**Business Meeting**

EMERGENCY CALLS AND MESSAGES

Convention Emergency Call Number 213 629-6416
8:30 a.m. to 4:30 p.m.

Saturday April 15 to Wednesday, April 19

VISIT TECHNICAL EXHIBITS

PATHOLOGY

Chairman.............. Frank R. Dutra, m.d., Castro Valley
Secretary.............. William H. Kern, m.d., Los Angeles
Assistant Secretary... Donald L. Bittner, m.d., San Francisco

FRANK R. DUTRA
Chairman

WILLIAM H. KERN
Secretary

**SATURDAY, APRIL 15 9:00 a.m.—Conference Room 4
Conference Room Floor**

9:00—Myocardium Tissue Cultures as a Medium in Toxicology—Thomas T. Noguchi, M.D., Los Angeles

9:15—Experimental Pulmonary Emphysema: Histology and Pulmonary Circulation—Robert L. Dennis, M.D., San Jose; Gerald A. Brooksby, B.S., M.A., and Boris B. Datnow, M.B., Sunnyvale, both by invitation; and Dan W. Clark, M.D., San Jose

9:30—Enzyme Patterns in Cardiac Diseases—Harry J. Sacks, M.D., Los Angeles

9:50—Role of the Clinical Pathologist in the Management of Cardiac Disease: The Pathologist's View—George Z. Williams, M.D., Bethesda, Maryland, by invitation

10:20—Recess

10:40—Thyroxine (T-4) by Column. Diagnostic Elution Pattern Using AN I^{131} Labeled Thyroxine Control—Donald L. Bittner, M.D., San Francisco, and Richard J. Grechman, M.D., San Francisco, by invitation

10:55—The Interpretation of T-3 and T-4 Resin Uptake Studies in Cardiovascular Disease—Richard J. Grechman, M.D., San Francisco, by invitation, and Donald L. Bittner, M.D., San Francisco

11:10—Congenital Heart Disease: Correlation of Angiocardiograms with Anatomical Lesions in Transposition of the Great Vessels—Pieter A. deVries, M.D., San Francisco

11:30—Lesions in Nephrosis: Light- and Electron-Microscopy of Renal Biopsy Specimens—James Hopper, M.D., and John Lee, M.D., San Francisco

11:55—Business Meeting

**SUNDAY, APRIL 16 9:00 a.m.-5:00 p.m.—Galeria Room
Galeria Floor**

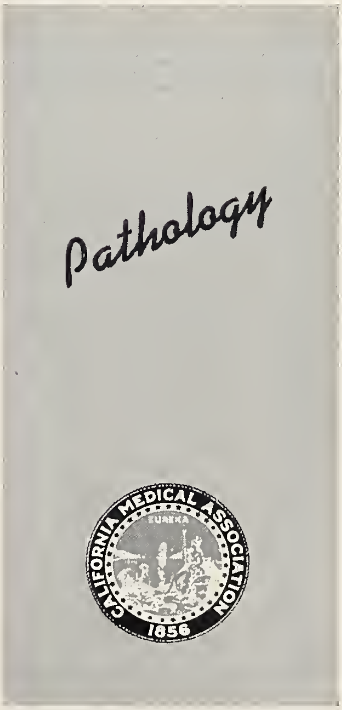
PATHOLOGY CONFERENCE

Childhood Tumors

Committee on Cancer, California Medical Association

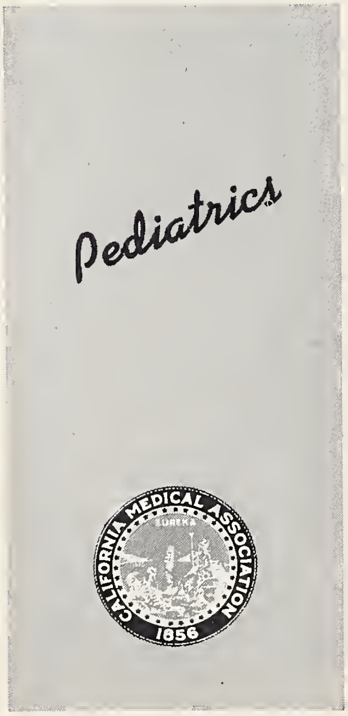
Chairman: Jack D. Kirshbaum, M.D., Encino
Moderator: Benjamin H. Landing, M.D., Los Angeles

9:00 a.m. to Noon—2:00 p.m. to 5:00 p.m.
Those who wish slides and protocols for the Conference (cost, $25.00) are requested to register with Weldon K. Bullock, M.D., Registrar, Tumor Tissue Registry, CMA Committee on Cancer, Los Angeles County Hospital, 1200 North State Street, Los Angeles 90033. The $25 fee includes registration. For those who wish to attend the Conference, the fee is $25. There is no charge for residents and interns.



RICHARD L. ANDERSON
Chairman

SHERROD C. SWIFT
Secretary



PEDIATRICS

Chairman..............RICHARD L. ANDERSON, M.D., Eureka
Secretary.................SHERROD C. SWIFT, M.D., Whittier
Assistant Secretary....ROBERT J. HARVEY, M.D., San Francisco

SUNDAY, APRIL 16 — 9:00 a.m.—Rendezvous Room Galeria Floor

9:00—**American Academy of Pediatrics**—Russell W. Mapes, M.D., Beverly Hills

9:20—**Crippled Children Services, California State Department of Public Health**—Ralph Hornberger, M.D., Berkeley

9:50—**Bureau of Maternal and Child Health, California State Department of Public Health**—Robert W. Day, M.D., Berkeley

10:10—**Bureau of Mental Retardation Service, California State Department of Public Health**—Charles Gardipee, M.D., Berkeley

10:30—**Recess**

10:45—**Children's Bureau Projects, Children's Bureau, United States Department of Health, Education and Welfare**—John J. Hutchings, M.D., San Francisco

11:10—**Medical Services Bureau, Office of Health Care Services, Health and Welfare Agency, California Department of Social Welfare**—Edgar Rosen, M.D., Sacramento

11:30—**Office of Health Care Services, California Department of Social Welfare**—Fernando G. Torgerson, Ph.D., Sacramento, by invitation

11:45—**California Department of Mental Hygiene, Bureau of Mental Retardation**—Roswell Fine, M.D., Sacramento

12:00—**Vocational Rehabilitation of Adolescents, California Department of Vocational Rehabilitation**—Mr. Donald Blyth, Los Angeles, by invitation

SUNDAY, APRIL 16 — 12:30 p.m.—Rendezvous Room Galeria Floor

12:30—**Pediatricians' Luncheon. Luncheon Address**—Crawford Bost, M.D., San Francisco

Advance reservations are necessary, and may be made with Sherrod C. Swift, M.D., 14619 Whittier Boulevard, Whittier 90605. Price $6.00

2:00—**Business Meeting**

TUESDAY, APRIL 18 **2:00 p.m.—Conference Room 1**
Conference Room Floor

Combined Meeting with Section on Urology

2:00—**Some Newer Aspects of Urologic Roentgenology**—Herbert M. Stauffer, M.D., Philadelphia, Pennsylvania, by invitation

2:30—**Laboratory Aids in Urologic Diagnosis**—George Z. Williams, M.D., Bethesda, Maryland, by invitation

3:00— **Panel Discussion**

Radiographic Techniques in Urologic Diagnosis

Moderator: Charles M. Stewart, M.D., Los Angeles

Members of the Panel: Herbert M. Stauffer, M.D., Philadelphia, by invitation; John L. Gwinn, M.D., Los Angeles; John A. Hutch, M.D., Pleasant Hill; and Leonard B. Skaist, M.D., Encino

4:50—**Urology Chairman's Address**—Carl K. Pearlman, M.D., Santa Ana

VISIT
SCIENTIFIC
AND
TECHNICAL
EXHIBITS

ATTENTION:

CHECK TELEVISION AND MOTION
PICTURE PROGRAMS, PAGES 39-41

PHYSICAL MEDICINE

Chairman................RENE CAILLIET, M.D., Los Angeles
Secretary.............ANTON A. TRATAR, M.D., San Francisco
Assistant Secretary......ELIZABETH AUSTIN, M.D., Los Angeles

RENE CAILLIET
Chairman

ANTON A. TRATAR
Secretary

TUESDAY, APRIL 18 **9:30 a.m.—Conference Room 2**
Conference Room Floor

9:30—**Chairman's Address: The Diagnosis of Neck and Arm Pain by Examination**—Rene Cailliet, M.D., Los Angeles

10:00—**Evaluation of Cervical Spine Surgery by Cineradiography**—Gregory Bard, M.D., San Francisco

10:30—**Electrodiagnosis of Neck and Arm Pain**—Sam C. Colachis, M.D., Los Angeles, by invitation

11:00—**Home and Office Exercise Program in Arthritis**—Robert L. Swezey, M.D., Los Angeles

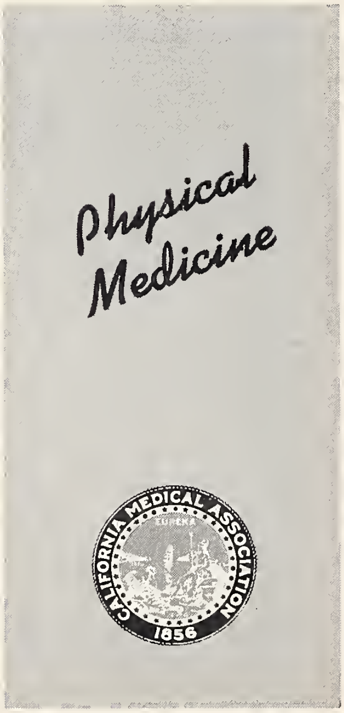
TUESDAY, APRIL 18 **2:00 p.m.—Conference Room 2**
Conference Room Floor

2:00—**Differential Diagnosis of Weakness and Atrophy**—William M. Fowler, Jr., M.D., Los Angeles

2:30—**Home and Hospital Management of Strokes**—Bernard J. Michela, M.D., Long Beach

3:00—**The Usefulness of Exercises in the Emphysema Patient**—Laszlo Ambrus, M.D., Walnut Creek, by invitation

3:30—**Business Meeting**



FOR YOUR LEISURE LISTENING

Hear Audio-Digest Tapes of other major Scientific meetings

Music Room Foyer, Galeria Floor
Daily

PREVENTIVE MEDICINE

Chairman..................HENRIK L. BLUM, M.D., Martinez
Secretary..........MAXWELL ROSENBLATT, M.D., Los Angeles
Assistant Secretary......RUSSELL B. WATSON, M.D., Santa Ana

HENRIK L. BLUM
Chairman

MONDAY, APRIL 17 **1:30 p.m.—Conference Room 4**
Conference Room Floor

1:30—**Planning for Community Health on a Regional Basis**—Robert Dyar, M.D., Berkeley

1:45—Discussion—Ralph C. Teall, M.D., Sacramento, and Eldon E. Ellis, M.D., Redwood City

2:15—**Chairman's Address: Planning for Community Health Services—The View From the Local Scene**—Henrik L. Blum, M.D., Martinez

2:45—**Business Meeting**

3:00—**Automation and Current Public Health Practices**—Olive Johnson, Los Angeles, by invitation

Discussion—Harold Chope, M.D., San Mateo, and Morris F. Collen, M.D., Oakland

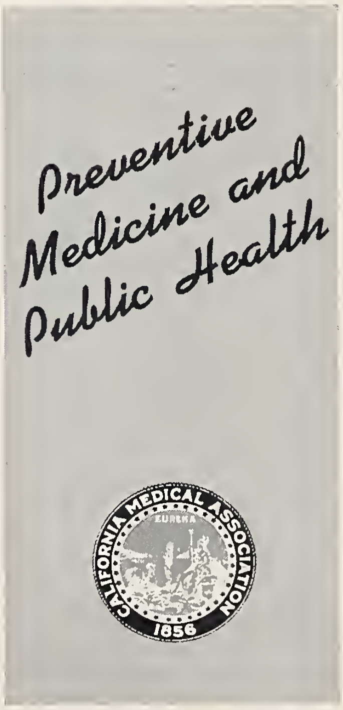
4:00—**An Integrated Approach to Environmental Health**—Leslie Chambers, Ph.D., Los Angeles, by invitation

4:20—**Public Health and Genetics**—Kenneth Dumars, M.D., Downey, by invitation

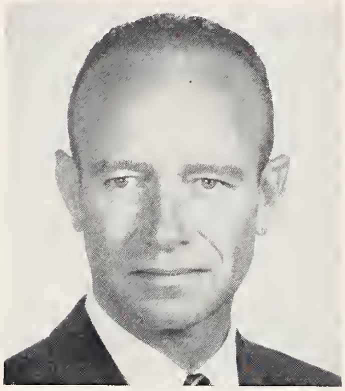
4:35—**Comprehensive Personal Health Care—The Neighborhood Family Health Center in Watts**—Robert Tranquada, M.D., and Elsie Giorgi, M.D., Los Angeles

MAXWELL ROSENBLATT
Secretary

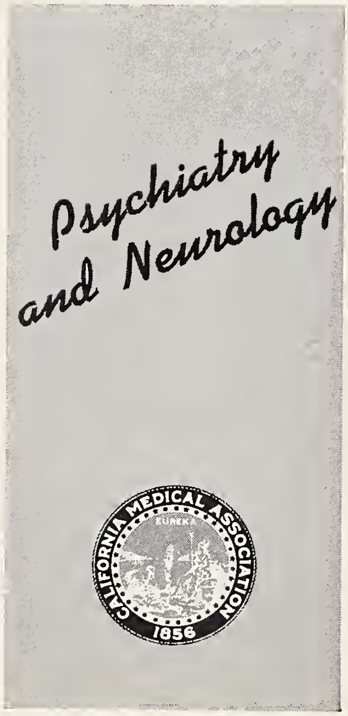
VISIT
SCIENTIFIC
AND
TECHNICAL
EXHIBITS


Preventive Medicine and Public Health
CALIFORNIA MEDICAL ASSOCIATION
EUREKA
1856

WERNER M. MENDEL
Chairman



AMES FISCHER
Secretary



PSYCHIATRY AND NEUROLOGY

Chairman..........WERNER M. MENDEL, M.D., Los Angeles
Secretary................AMES FISCHER, M.D., San Francisco
Assistant Secretary....HOWARD S. BARROWS, M.D., Los Angeles

WEDNESDAY, APRIL 19 9:00 a.m.—Renaissance Room Galeria Floor

Combined Meeting with Section on Internal Medicine

9:00— **The Impact of Public Medicine**

Chairman: Robert L. Paver, M.D., San Francisco

9:00—**Aid to the Totally Disabled—The Pitfalls as Seen by a Psychiatrist**—Morton R. Weinstein, M.D., San Francisco

9:30—**Discussion**—Daniel S. Chaffin, M.D., San Rafael

9:45— **Panel Discussion**

The Role of Medicine in Society

Moderator: Malcolm S. M. Watts, M.D., San Francisco

Members of the Panel: To be announced

11:00— **Panel Discussion**

Regional Medical Programs on Heart, Stroke and Cancer

Moderator: To be announced

Members of the Panel: To be announced

12:00—Recess

WEDNESDAY, APRIL 19 1:30 p.m.—Renaissance Room Galeria Floor

Combined Meeting with Section on Internal Medicine

The Impact of Public Medicine
(Continued)

Chairman: Werner M. Mendel, M.D., Los Angeles

1:30—**Meaning of Changing Emphasis on Community Mental Health to the Practicing Physician**—Allen J. Enelow, M.D., Los Angeles and Louis J. West, M.D., Oklahoma City, by invitation

2:30—**Chairman's Address**—Werner M. Mendel, M.D., Los Angeles

2:45—**A Critical Look at Medicare**—Edgar Rosen, M.D., Sacramento

RADIOLOGY

Chairman..............VICTOR G. MIKITY, M.D., Los Angeles
Secretary...................VERNON R. GEE, M.D., Redding
Assistant Secretary......RICHARD P. STORRS, M.D., Los Angeles

VICTOR G. MIKITY
Chairman

SUNDAY, APRIL 16 **9:30 a.m.—Conference Room 2**
Conference Room Floor

9:30—**The Role of Vertebral Angiography in Posterior Fossa Diagnosis**—Gabriel H. Wilson, M.D., Los Angeles, and Mutsumasa Takahashi, M.D., Los Angeles, by invitation

10:00—**Angiographic Studies in Disorders of the Pulmonary Circulation**—William A. Weidner, M.D., Los Angeles

10:30—**Percutaneous Needle Biopsy of the Lung for the Diagnosis of Lung Tumors**—Byron E. Swenson, M.D., Pasadena, by invitation

11:00—**The Epi-pericardial Interface: Further Evaluation of Carbon Dioxide, Opaque and Echo Cardiography in the Diagnosis of Effusion**—Herbert M. Stauffer, M.D., Philadelphia, by invitation

VERNON R. GEE
Secretary

SUNDAY, APRIL 16 **2:00 p.m.—Ballroom**
Galeria Floor

SPECIAL GENERAL MEETING

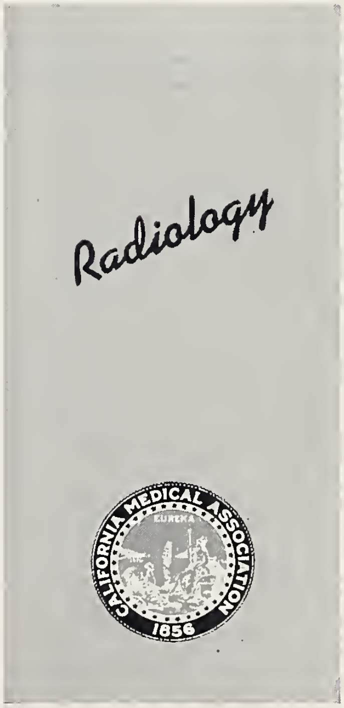
First Annual L. Henry Garland Memorial Lecture

Sponsored by the California Radiological Society

2:00—**The Radiologist and the Search for Hidden Cancer**—Leo Rigler, M.D., Los Angeles

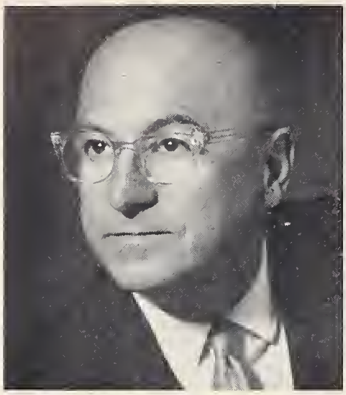
3:00—**Recess**

3:15—**Business Meeting and Annual Meeting of California Radiological Society**



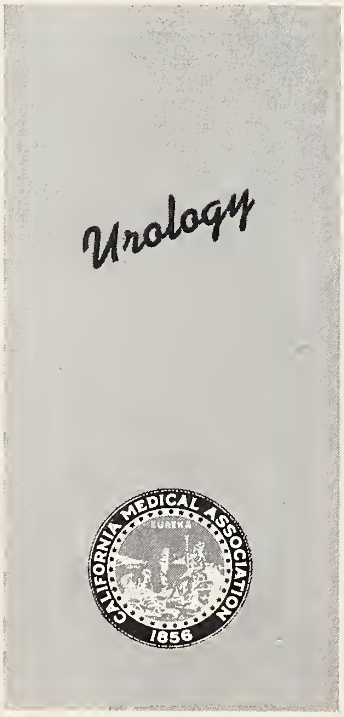
ATTENTION:

CHECK TELEVISION AND MOTION PICTURE PROGRAMS, PAGES 39-41



CARL K. PEARLMAN
Chairman

JAMES L. GOEBEL
Secretary



UROLOGY

Chairman...............Carl K. Pearlman, m.d., Santa Ana
Secretary.....................James L. Goebel, m.d., Ross
Assistant Secretary.......Robert O. Pearman, m.d., Encino

MONDAY, APRIL 17 **9:00 a.m.—Galeria Room**
Galeria Floor

Combined Meeting with Section on General Practice

9:00—**Some Ideas on Stress Incontinence in the Female**—John A. Hutch, M.D., Pleasant Hill

9:30—**Discussion**—Edward B. Cantor, M.D., and Erle Henriksen, M.D., Los Angeles

9:40— **Panel Discussion**

The Diagnosis and Treatment of Urinary Tract Infections in Females

Moderator: Elmer Belt, M.D., Los Angeles

Members of the Panel: Daniel G. Morton, M.D., and Thomas W. Cook, M.D., both Los Angeles; John M. Palmer, Palo Alto; John W. Dorsey, M.D., Long Beach; and Gerald E. Howe, M.D., San Diego

10:40—**Recess**

10:50— **Panel Discussion**

Management of Urologic Injuries in Gynecologic Surgery

Moderator: Elmer Belt, M.D., Los Angeles

Members of the Panel: Daniel G. Morton, M.D., Thomas W. Cook, M.D., both Los Angeles; John M. Palmer, M.D., Palo Alto; John W. Dorsey, M.D., Long Beach; and Gerald E. Howe, M.D., San Diego

TUESDAY, APRIL 18 **2:00 p.m.—Conference Room 1**
Conference Room Floor

Combined Meeting with Section on Pediatrics

2:00—**Some Newer Aspects of Urologic Roentgenology**—Herbert M. Stauffer, M.D., Philadelphia, Pennsylvania, by invitation

2:30—**Laboratory Aids in Urologic Diagnosis**—George Z. Williams, M.D., Bethesda, Maryland, by invitation

3:00— **Panel Discussion**

Radiographic Techniques in Urologic Diagnosis

Moderator: Charles M. Stewart, M.D., Los Angeles

Members of the Panel: Herbert M. Stauffer, M.D., Philadelphia, by invitation; John L. Gwinn, M.D., Los Angeles; John A. Hutch, M.D., Pleasant Hill; Leonard B. Skaist, M.D., Encino

4:50—**Chairman's Address**—Carl K. Pearlman, M.D., Santa Ana

5:00—**Business Meeting**

MOTION PICTURE PROGRAM

Music Room ▪ Biltmore Hotel

Co-Chairmen:

Richard E. Gardner, M.D., San Francisco, and W. Morris H. Noble, M.D., San Francisco

Five film symposia will be presented, each utilizing about two-thirds of the time for projecting of films, and one-third for discussion, questions and answers.

SUNDAY, APRIL 16 **2:00 p.m.—Music Room Galeria Floor**

2:00— Surgery

Moderator: Richard E. Gardner, M.D., San Francisco.

2:00—Introduction

2:05—**Shock. Physiochemical Principles and Treatment**—William Schumer, M.D., Sacramento.

Discussion—Herbert Shubin, M.D., Los Angeles.

2:37—**The Surgical Treatment of Pectus Excavatum (Ravitch Technique)**—Jordan J. Weitzman, M.D., and William H. Snyder, Jr., M.D., Los Angeles.

Discussion—Jordan J. Weitzman, M.D., Los Angeles.

3:17—**Ligation and Division of the Inferior Vena Cava to Prevent Recurrent Pulmonary Emboli**—W. Clayton Davis, M.D., Pittsburgh, and John W. Maxwell, Jr., M.D., Milwaukee.

Discussion—Jack Cannon, M.D., Los Angeles.

3:47—**Control of Pseudomonas Burn Wound Sepsis**—John A. Moncrief, M.D., and Robert B. Lindberg, M.D., Fort Sam Houston, Texas.

Discussion—Irving A. Meeker, Jr., M.D., Los Angeles.

4:22—**Left Colon Total Esophageal Bypass**—Ivan May, M.D., Oakland.

Discussion—Arthur J. Hunnicut, M.D., Oakland.

MONDAY, APRIL 17 **2:00 p.m.—Music Room Galeria Floor**

2:00— Cardiology

Films and Exhibits

Moderator: Willard Zinn, M.D., Los Angeles.

2:00—Introduction

2:05—**Auscultation of the Heart: Mitral Stenosis**—Abe Ravin, M.D., Denver.

Following the presentation of heart sounds there will be audience participation of cardiac sounds and self-testing unit for individual participation and study.

Discussion Leader—Willard Zinn, M.D., Los Angeles.

3:00—**The Nurse in Emergency Cardiopulmonary Resuscitation**—California Heart Association.

Discussion—Richard E. Gardner, M.D., San Francisco.

3:30— Pediatrics

Moderator: Mary B. Olney, M.D., San Francisco.

3:30—**Physical Examination of the Newborn**—Mary B. Olney, M.D., San Francisco.

Discussion—Mary B. Olney, M.D., San Francisco.

4:10—**Urinary Tract Infection in Girls**—Colin Markland, M.D., Minneapolis.

Discussion—Donald C. Martin, M.D., Los Angeles.

4:40—**Cancer in Children**—American Cancer Society.

Discussion—Lewis Guiss, M.D., Los Angeles.

TUESDAY, APRIL 18 **2:00 p.m.—Music Room Galeria Floor**

2:00— Obstetrics and Gynecology

Moderator: James Ravenscroft, M.D., San Diego.

2:00—Introduction

2:05—**Modern Management of Pregnancy in the Rh-Negative Sensitized Woman**—William M. Alpern, M.D., and Allan G. Charles, M.D., Chicago.

Discussion—Denman Hammond, M.D., Los Angeles.

2:50—**The Mechanisms of Action of the Oral Contraceptives**—Edward T. Tyler, M.D., Los Angeles, Martin L. Stone, M.D., New York, and Melvin R. Cohen, M.D., Chicago.

Discussion—Edward T. Tyler, M.D., Los Angeles.

3:35—**The Obsolete Menopause**—Allan C. Barnes, M.D., Baltimore, Georgeanna S. Jones, M.D., Baltimore, M. Edward Davis, M.D., Chicago, Herbert S. Kupperman, M.D., New York, Hubert de Watteville, M.D., Geneva, Switzerland, and Robert W. Kistner, M.D., Boston.

Discussion—Hans Simmer, M.D., Los Angeles, by invitation.

4:15—**Abdominovaginal Approach to Stress Incontinence**—Edward B. Cantor, M.D., Los Angeles.

Discussion—Edward B. Cantor, M.D., Los Angeles.

WEDNESDAY, APRIL 19 **2:00 p.m.—Music Room Galeria Floor**

2:00— Medicine

Moderator: W. Morris H. Noble, M.D., San Francisco.

2:00—Introduction

2:05—**Thyroid Deficiency—Current Concepts of Diagnosis and Treatment**—Sidney H. Ingbar, M.D., Boston, J. Thomas Dowling, M.D., Seattle, William McK. Jefferies, M.D., Cleveland, Edward Rose, M.D., Philadelphia, and Judson J. Van Wyk, M.D., Chapel Hill, N.C.

Discussion—Boris Catz, M.D., Beverly Hills.

2:48—**Cryohypophysectomy in Acromegaly**—Robert W. Rand, M.D., Los Angeles.

Discussion—Robert W. Rand, M.D., Los Angeles.

ANNOUNCEMENT ON AUDIO-DIGEST

Audio-Digest tapes will be available in the foyer of the Music Room, Galeria Floor, for those physicians who would like to review at their leisure outstanding material recorded and condensed from other major scientific meetings.

Audio-Digest is an important educational unit of the California Medical Association; and this is the first time this service has been offered at the Annual Scientific Assembly. Editorial representatives will be available to answer questions about the activities of Audio-Digest, and how this material can be used in individual and institutional educational programs.

TELEVISION PROGRAM

THE MEDICAL TELEVISION NETWORK: *"Selected RES MEDICA"*

MUSIC ROOM • BILTMORE HOTEL

PLANNED BY THE ADMINISTRATION AND PRODUCTION STAFF OF THE MEDICAL TELEVISION NETWORK

Donald W. Brayton, M.D., *Los Angeles*
Richard S. Scott, M.D., *Los Angeles, by invitation*
Mr. Richard Getz, *Los Angeles, by invitation*

Video tapes of six recent presentations • Some of the participants and producers will be present to discuss programs and answer questions • General information about the Medical Television Network, a cooperative educational project involving the Medical Community of Southern California

MONDAY, APRIL 17 **9:00 a.m.—Music Room Galeria Floor**

9:00— THE STROKE PATIENT

A comprehensive review of the work-up and management of cerebrovascular disease in a "programmed patient."

Howard Barrows, M.D., and Ruth McCormick, M.D.—Department of Neurology, University of Southern California School of Medicine, Los Angeles.

10:15—THE ANATOMY AND PHYSIOLOGY OF THE MEDICAL TELEVISION NETWORK

Postgraduate medical education by scrambled broadcast television. The organization of the network; operations and goals.

10:30— L.S.D.—PROBLEMS AND PROSPECTS

Contrasting views of the current uses and abuses of this fantastic chemical.

Keith Ditman, M.D., by invitation; Sidney Cohen, M.D.; and Samuel Eiduson, M.D., by invitation—University of California, Los Angeles, School of Medicine. Mr. Alan Watts, Philosopher, Sausalito, by invitation.

TUESDAY, APRIL 18 **9:00 a.m.—Music Room Galeria Floor**

9:00— THE MANAGEMENT OF GASTRIC ULCER

A spirited discussion between gastroenterologists, surgeons and a radiologist. Benign or malignant? Medical therapy or surgical attack? Areas of uncertainty and conflicting opinion.

J. Edward Berk, M.D., Paul H. Guth, M.D., Jack M. Farris, M.D., Thomas Keller, M.D., and E. Nicholas Sargent, M.D.—University of California, California College of Medicine, Los Angeles.

10:15—THE ANATOMY AND PHYSIOLOGY OF THE MEDICAL TELEVISION NETWORK

Postgraduate medical education by scrambled broadcast television. The organization of the network; operations and goals.

10:30— THE SEVERELY TRAUMATIZED PATIENT

Dramatic demonstrations of the important initial steps in evaluation and care of the patient with multiple injuries.

Ellsworth E. Wareham, M.D., and Alonzo Neufeld, M.D.; Wilfred M. Huse, M.D., and Lloyd A. Dayes, M.D., both by invitation; and Louis L. Smith, M.D.—Loma Linda University School of Medicine, Loma Linda.

WEDNESDAY, APRIL 19 **9:00 a.m.—Music Room Galeria Floor**

9:00— THE CONSERVATIVE MANAGEMENT OF LOW BACK PAIN

Descriptions and demonstrations of the mechanics, diagnosis and treatment of lumbosacral symptoms.

Elizabeth Austin, M.D., Rene Cailliet, M.D., and David Rubin, M.D.—Physical Medicine and Rehabilitation Staff, University of Southern California School of Medicine, Los Angeles.

10:15—THE ANATOMY AND PHYSIOLOGY OF THE MEDICAL TELEVISION NETWORK

Postgraduate medical education by scrambled broadcast television. The organization of the network; operations and goals.

10:30— GENES, CHROMOSOMES AND HUMAN DISEASE

Clear and concise explanations of many genetic syndromes and advice for sensible genetic counseling.

David Comings, M.D., and Ernest Beutler, M.D., both by invitation; and Raymond Teplitz, M.D.—City of Hope Medical Center, Duarte.

SCIENTIFIC AND ORGANIZATIONAL EXHIBITS

Rex Room and South Galeria

Scientific Exhibits

Rex Room, Below Biltmore Bowl

The American Association of Blood Banks—E. A. Dreskin, M.D., Chicago, by invitation. This exhibit presents the various activities and services of the American Association of Blood Banks. (Booth No. 1)

Cardiac Enzymes in Diagnosis of Myocardial Infarction—Eugene Coodley, M.D., Bakersfield. This exhibit illustrates the types of enzyme reactions available in the study of myocardial infarction, including chemical reactions. There is a summary in tabular form of various enzyme procedures available, and a demonstration for problems in diagnosis. (Booth No. 2)

American Medical Association—Food and Gastrointestinal Disorders—Nutrition—This exhibit asks the question, "How rational are dietary practices?" Calls for objective research that includes controls, eliminates prejudice, and psychic influence, and makes due allowance for chance variation in diet therapy. (Booth No. 58)

Subclavian Technique of Central Venous Pressure Monitoring—Robert Craig, M.D., and Gilbert Kinyon, M.D., San Diego. This exhibit describes and illustrates a useful technique for monitoring of central venous pressure via the subclavian vein as well as a valuable avenue for prolonged infusion of fluids, electrolytes, and blood. (Booth No. 59)

Bone Marrow Studies—Diagnostic Importance in Non-Hematologic Conditions—Michael Rubinstein, M.D., Beverly Hills. This exhibit presents case histories which are abstracted and illustrated. Diagnostic findings and bone marrow findings, non-hematologic conditions of bone marrow studies are described. (Booth No. 60)

Transurethral Prostatectomy: The Urologist's Choice—Robert J. Prentiss, M.D., San Diego. This exhibit shows a step-by-step approach to transurethral prostatectomy. Also shown are the statistics concerning the author's results in practice and in county hospital residents' practice using this approach. (Booth No. 63)

Human Growth Hormone (HGH)—Robert Blizzard, M.D., Bethesda, Maryland, by invitation. The purpose of this exhibit is to review with text, photographs, charts and comparative x-rays, the diagnosis and differential diagnosis of dwarfism. The considerable progress of recent research in the physiology and biochemistry of human growth hormone are presented. (Booth No. 64)

Airway Resistance As an Index of Effectiveness of Bronchodilating Drugs—Leonard Howard, M.D., Greenwich, Connecticut, by invitation. This exhibit shows the newly developed constant volume, full body, plethysmograph which is used to determine airway resistance. Significance of this is also discussed. Drug used in asthmatic and emphysematous patients was metaproterenol. (Booth No. 65)

Is The "Square Cut Toenail" A Fallacy?—Carroll Andrews, M.D., Sonoma. Color photographs, line drawings, and charts of text in outline-form will be displayed. From early childhood to elderly patients whose primary complaint is usually foot or skin exzemas. Related problems—peripheral vascular, arterial venous and/or lymphatic. Questions about avenue of extensive physio-pathological research of multidiscipline type. (Booth No. G)

Organizational and Scientific Exhibits

South Galeria, Galeria Floor

Clinical Research in Private Practice—Seymour Diamond, M.D., Chicago, Illinois, by invitation. Outlined and discussed are rationale, pre-requisites, procedures of doing controlled clinical research in private practice. Discussed are disadvantages to both patient and physician. An example of controlled study and results is given. Charts showing results of evaluation of the mean severity of patients presenting symptoms both prior to and following therapy are presented.

Rx of Salicylate Intoxication in Children—Berkeley Rich, M.D., William Dobbins, M.D., Denver, Colorado, by invitation. This exhibit identifies the magnitude and significance of salicylate intoxication as public health hazard, and focuses attention on its particular prevalence in children. Diagrammed is the progressive metabolic alterations with associated signs and symptoms. Illustrated and outlined are the various methods of management—their rationale, advantages, disadvantages, and effectiveness. Suggestions for practical prevention are made, and a graded approach to therapy when intoxication is encountered is presented. Available to viewers are handouts with more complete data and data-breakdown, documentation and expanded comments on highlighted points.

Radial Immunodiffusion: An Indispensable Diagnostic Tool—Edward Shanbrom, M.D., Los Angeles. The principles, technics, and applications of the new immunologic method, radial immunodiffusion, will be illustrated. By pictorial display, the production of antibody will be outlined. The antibody, which is rendered specific for a single serum protein, is incorporated into a supporting medium and utilized as the internal reactant in immunodiffusion for the quantitative measurement of a serum protein. Utilizing this principle, the gamma globulins (IgG, IgM, IgA immunoglobulins), transferrin, ceruloplasmin, β_{1c}/β_{1a} globulin (C'_3, a component of complement), and α_2 macroglobulin concentrations in serum may be easily determined. In addition, investigations are underway to quantitate prealbumin, α_1 anti-trypsin, and orosomucoid by means of immunodiffusion, since the significance of these serum components cannot be understood until simple quantitative methods are available. Similarly, by incorporating antigen into the media and diffusing the test serum, the technic may be used to detect precipitating and complement-fixation antibodies against certain infectious diseases (*e.g.*, histoplasmosis and coccidioidomycosis). With these technics for measuring specific serum proteins, the horizon of diagnostic and prognostic medicine has been vastly widened, and the enlightened physician can ill afford to ignore these advances.

Organizational Exhibits

Medic Alert Foundation—Nonprofit organization dedicated to urging individuals with hidden medical problems to wear adequate identification. The exhibit explains the educational program for physicians and law enforcement personnel and asks them to watch for such warnings. The exhibit also indicates important medical problems which should be known in emergency, and informs of a 24-hour collect call answering service for physicians' use in emergency.

Poison Information Center—The purpose of this exhibit is to explain and describe with displays and literature, that the Center is available to give educational talks on accidental poisonings. Table content shows common household drugs, both over-the-counter and by prescription, and garden hazards.

American Medical Association—Adverse Drug Registry—The purpose of this exhibit is to familiarize physicians with the operation of the Registry on Adverse Drug Reactions. There are panels listing adverse reactions caused by drugs, and there are special brochures discussing possible adverse reactions to drugs used by specialty groups as well as general practitioners.

California Medical Association—Committee on Continuing Medical Education—*What Goes On*—This exhibit explains *What Goes On*, a physician's guide to continuing medical education opportunities in California and Hawaii. The exhibit also will show how the information on all medical meetings in California and Hawaii can be used by physicians planning programs to avoid conflict and duplication.

California Medical Association—Commission on Community Health Services—*Health Tips*—This exhibit shows the progress made through five years of publication and growth. It graphically illustrates growth backed up by newspaper, magazine, and house organ articles as well as laudatory letters from users.

REGISTRATION

Registration and information desks are located in the Galeria. *All members, guests, and visitors are requested to register immediately on arrival.* There is no charge for registration. Registration desks are open Saturday through Wednesday. *Admission to the general and section sessions and exhibit area is by badge only.*

Members wishing to vote in specialty sections must indicate appropriate section when registering; voting in other sections will not be allowed.

WOMAN'S AUXILIARY

THIRTY-SEVENTH ANNUAL CONVENTION
APRIL 16 to 19, 1967

Headquarters: Biltmore Hotel, Los Angeles

Convention Chairman: MRS. F. LEGRAND NOYES
Convention Co-Chairman: MRS. JOHN R. BLACK

REGISTRATION—North Galeria

Sunday, April 16—9:00 a.m. to 4:00 p.m.
Monday, April 17—8:00 a.m. to 4:00 p.m.
Tuesday, April 18—8:30 a.m. to Noon

SATURDAY, April 15

6:00 p.m.—Annual Report of Woman's Auxiliary presented by the President, Mrs. Samuel K. Bacon, to the California Medical Association House of Delegates, Biltmore Bowl. Doctors' wives are invited to attend. Auxiliary members will not register for this meeting. Woman's Auxiliary Registration will start Sunday morning

SUNDAY, April 16

9:00 a.m.—Executive Committee breakfast meeting, President's Suite

2:30 p.m.—Pre-Convention Board meeting, Conference Room No. 5

6:30 p.m.—Presidents' Reception—Renaissance Room

MONDAY, April 17

8:30 a.m.—Opening Session of House of Delegates, Biltmore Bowl

11:15 a.m.—"Pronto" Luncheon to be served in Biltmore Bowl

12:45 p.m.—Afternoon Session of House of Delegates, Biltmore Bowl

TUESDAY, April 18

8:00 a.m.—Final Session of the House of Delegates, Biltmore Bowl

12:15 to 1:00 p.m.—Reception honoring incoming and outgoing officers, Ballroom Foyer

1:00 p.m.—Presidents' Luncheon honoring Mrs. Samuel K. Bacon and Mrs. William R. Flood, President-Elect; State Past Presidents; and CMA Advisory Board and their wives, Ballroom

WEDNESDAY, April 19

8:00 a.m.—Post-Convention Board meeting, Mrs. William R. Flood presiding, Conference Room No. 4

9:30 a.m.—Orientation Meeting for 1967-68 State Board Members, District Councilors, County and District Presidents. Incoming County Board Members are also welcome. Mrs. William R. Flood presiding, Conference Room No. 4

HOSPITALITY CENTER

The Hospitality Center will be open
Sunday, April 16—12:00 to 4:00 p.m.
Monday, April 17—9:00 a.m. to 5:00 p.m.
Tuesday, April 18—9:00 a.m. to Noon

MRS. SAMUEL K. BACON, *President*

MRS. WILLIAM R. FLOOD, *President-Elect*

Technical Exhibits

Technical exhibits will be housed in the Hotel Biltmore's Rex Room, located directly below the Biltmore Bowl and reached by escalator. Here the many exhibitors will present their products and services for members of the Association.

All exhibits and all products exhibited have been screened by a committee as a means of eliminating those which do not meet high standards. The exhibitors agree to this procedure and agree that by this means each will be in good company.

Here in one area will be found the latest developments in drugs, equipment and services to aid the physician in his professional activities. All physicians are urged to visit the exhibits; meetings have been planned to allow ample time for this important activity. Your visit will not only help bring your own knowledge up to date; it will demonstrate to our exhibitors, who contribute so much to the success of the meeting, that we recognize and appreciate their fine cooperation.

Exhibits are open from 9 a.m. to 5 p.m. each day, Sunday through Tuesday, with an early closing on Wednesday.

ABBOTT LABORATORIES Booth 67
North Chicago, Illinois

Abbott Laboratories invites you to visit our exhibit. Our representatives will be happy to answer any questions you may have concerning our leading products and new developments.

ALTA-DENA DAIRY Booth 37
City of Industry

Certified Milk will be served; taste the difference, produced clean, disease free, antibiotic free, and guaranteed safe, through daily laboratory tests conducted, under the supervision of the Los Angeles County Medical Association Milk Commission for over 57 years.

Kefir—Medical reports indicate good results in normalizing intestinal function following antibiotic therapy. This cultured milk is seeded with Lactobacillus Acidolphilus and Lactobacillus Bulgaricus and other friendly bacteria.

Medical Milk Commission Laboratory reports are made available for your inspection—our representatives will be glad to answer your questions about any of our outstanding products.

AMERICANA CORPORATION Booth 109
Beverly Hills

See the new 1967 Americana Encyclopedia and Min/Max Teaching Machine.

ARNAR-STONE LABORATORIES, INC. Booth 51
Mount Prospect, Illinois

Americaine Topical Anesthetic—20 per cent dissolved benzocaine in a water-soluble base—ointment, liquid, suppositories and aerosol forms. Aerosol operates right-side up or upside down for contortion-free application. Hazel-Balm—a "medicated cushion of foam" combines cooling, soothing witch hazel with emollient water-soluble lanolins; wide range of usefulness in office and hospital practice. Quinette—the vaginal insert with disposable "pink pencil" inserter for 36-hour control of symptoms—12-day treatment—in mixed or monilial vaginal infections. Tetrasule—prolonged protection against attacks of angina pectoris—b.i.d. dosage of PETN with or without sedation.

ASTRA PHARMACEUTICAL PRODUCTS, INC. Booth 55
Worcester, Massachusetts

Information and descriptive literature pertaining to Xylocaine® (lidocaine) and Citanest® (propitocaine) local and topical anesthetics, and iron preparations Astrafer® (dextriferron) for intravenous use and Jectofer® (iron sorbitex) for intramuscular administration will be available at the Astra booth presided over by our representative, Mr. Eric V. Ericson.

AUDIO-DIGEST FOUNDATION Booth 112
Pacific Medical Equipment Co.
North Hollywood

Audio-Digest Foundation (a non-profit subsidiary of the California Medical Association) gives the busy physician a time-saving tour through the best of some 600 current medical journals, plus the highlights of scores of national meetings. Time-proven, but still unique—these medical tape-recorded services are now offered in seven series—*General Practice, Surgery, Internal Medicine, Obstetrics & Gynecology, Anesthesiology* and *Ophthalmology.*

Digest subscribers listen in their car, home or office. Carefully selected tape equipment for playing the Digests is offered at the convention by Pacific Medical Equipment Co. (Authorized Sales Outlet for Audio-Digest subscriptions—and equipment).

AYERST LABORATORIES Booth 14
New York, New York

Ayerst Laboratories extends an invitation to visit our exhibit located in space number 14 where Premarin and Riopan are being featured. Our representatives will be pleased to discuss these or other Ayerst products with you.

BARNES-HIND LABORATORIES Booth 12
Sunnyvale

BEECHAM RESEARCH LABORATORIES, INC. Booth 61
New York, New York

You are cordially invited to visit our exhibit featuring Penbritin (ampicillin), the new semi-synthetic broad-spectrum penicillin for wider use in major areas of infection. Booth No. 61.

BERKELEY MEDICAL INSTRUMENTS Booth 53
Berkeley

The B.M.I. Blood Analyzer and simplified procedures for determination of hemoglobin concentration, true blood glucose, uric acid, cholesterol and B.U.N. A compact, rugged instrument to give accurate and reproducible results in mere minutes in physicians' offices, clinics, laboratories and hospitals. Also on display is the new B.M.I. electronic incubator.

BEVERLY MEDICAL SUPPLY CO. Booth 25
Beverly Hills

Beverly Medical Supply will show several of the latest equipment items, of major manufacturers. Also various new instruments and sundry items. Emphasis will be on items used in the doctor's office.

BREON LABORATORIES INC. Booth 41
New York, New York

Breon Laboratories Inc. presents a full line of products for the care of patients with chronic obstructive pulmonary diseases. Included are Alevaire, Bronkometer, Bronkospray, Bronkotabs, Bronkotab Elixir and Bronkephrine. Supplying a variety of formulas, dosage forms and actions, these products offer both prophylaxis and therapy, in chronic or acute conditions, to all ages. Breon personnel will gladly discuss specific products and therapies with you.

BRISTOL LABORATORIES Booth 18
Syracuse, New York

THE BROWN PHARMACEUTICAL COMPANY Booth 100
Los Angeles

Android—(androgen-thyroid combinations)—family of products: Cerebro-Nicin—pentalenetrazole-niacin combinations; Lipo-Nicin—nicotinic acid, plus vitamins; Aasquel—belladonna compound plus ¼ gr phenobarbital; Geramine—testosterone-estrogen-thyroid plus vitamins and minerals; Stimulon—amphetamine, plus vitamins; Glukor—human chorionic gonadotropin.

BURROUGHS WELLCOME & CO. (U.S.A.) INC. Booth 24
Tuckahoe, New York

Of particular interest at this meeting is Zyloprim brand allopurinol, which represents a totally new concept for the management of gout, uric acid nephropathy and calculi, and hyperuricemia due to neoplastic disease and/or its intensive treatment.

Zyloprim was first synthesized in the Wellcome Research Laboratories in Tuckahoe, New York, and is the first drug to be marketed which reduces the formation of uric acid at its source.

BURTON, PARSONS & COMPANY, INC. Booth 80
Washington, D.C.

You are cordially invited to stop at the Burton, Parsons & Company exhibit where our representative will be showing Konsyl, L. A. Formula, neutraCarb, and EKG Sol. Samples, descriptive literature, and information will be available on all these products. Konsyl and L. A. Formula are the original refined psyllium bulk laxatives; neutraCarb, antacid, is packaged in five delightful flavors, cherry, lemon, lime, peppermint and raspberry, 10 tablets in a convenient and handy roll; EKG Sol is the new and modern electrode cream for electrocardiography and electroencephalography.

CARNATION COMPANY Booth 42
Los Angeles

Carnation Company cordially invites you to visit Booth No. 42, where medical representatives will be pleased to welcome members and guests of the California Medical Association.

Recent literature and information regarding Carnation Evaporated, Carnation Instant Non-Fat Milk, Carnation Instant Breakfast and Carnalac are available. Any question pertaining to our physician-researched material for use in your practice or hospital will be cheerfully discussed.

CASS & JOHANSING Booth 99
Los Angeles

Representatives will be present to discuss approved County Medical Association Insurance Programs—Group Disability, Non-Cancellable Disability, Life, and Accidental Death & Dismemberment.

In addition, assistance in complete insurance programming will be available.

CIBA PHARMACEUTICAL COMPANY Booth 81
Summit, New Jersey

THE COCA-COLA COMPANY Booth 82
Atlanta, Georgia

Ice cold Coca-Cola served through the courtesy and cooperation of the Coca-Cola Bottling Company of Los Angeles, and The Coca-Cola Company.

COLE PHARMACAL COMPANY, INC. Booth 43
St. Louis, Missouri

Cole Pharmacal Company, Inc. will feature Iodo-Niacin® and its role in arteriosclerotic and bronchial therapy.

See the new anatomical art.

Helpful information on Asminyl® and Banalg® and other select Cole products will be available.

CUTTER LABORATORIES Booths 38 & 39
Berkeley

The Cutter Hospital Display Booth No. 38 will illustrate the inherent completely closed system safety features of the famous Cutter line of Saftisystem I.V. administration sets and solutions. Sub-featured will be the expanding line of Cutter products for use with specialized medical procedures.

Cutter Laboratories Booth No. 39 will feature evidence of the Laboratories' leadership in human blood fractions, research and manufacturing. Professional information is available on Hyper-Tet™ (tetanus immune globulin—human, U.S.P.) and other products resulting from this leadership.

DAIRY COUNCIL OF CALIFORNIA Booth 66
Sacramento

"Weighty Problems?"

A 1,000-calorie diet (three meals) for one day is shown to illustrate that the Food way is the best way to weight reduction. This diet is adequate in all nutrients.

Nutrition education materials will be displayed that are available without charge to aid in reinforcing diet instructions to patients.

DAYLIN MEDICAL & SURGICAL SUPPLY, INC. Booth 76
Huntington Park

Daylin will feature the latest in medical equipment by Ritter Equipment Company. The Ritter representative, Pat Kelly, will be on hand to answer all questions. Daylin will also feature the electrically illuminated diagnostic instruments by Welch-Allyn and a complete line of Daylin pharmaceuticals.

D. M. DOYLE PHARMACEUTICAL COMPANY Booth 31

A Division of The Dietene Company
Minneapolis, Minnesota

Have you tasted Meritene? Meritene is the good-tasting *protein*-vitamin-mineral food supplement prescribed to provide concentrated nutrition for patients with poor appetite or tolerance for ordinary food. Visit our booth and let us serve you a cool, refreshing Meritene nourishment.

While there, review also our Dietene Reducing Plan, designed to get better cooperation from over-weight patients. The Dietene Plan provides optimum nutrition and maximum satiety without the use of drugs.

Meritene and Dietene are advertised only to the medical profession.

E. I. DU PONT DE NEMOURS & CO., INC. Booths 48, 49, 50

Wilmington, Delaware

Symmetrel (amantadine HCl) is a molecular barrier to virus penetration. It is not a vaccine or an antibiotic, but a new synthetic chemical unrelated to any other chemotherapeutic agent. It is the first and only oral medication for the prevention of respiratory infections caused by influenza A_2 (Asian) virus tested to date. It appears to exert its antiviral effect by a unique mode of action. It prevents virus penetration of the host cell without affecting vital cell functions. It does not interfere with normal antibody response, but acts in concert with pre-existing antibody. It does not interfere with other drugs, and does not preclude the use of vaccines.

Symmetrel has been researched extensively. Double-blind clinical studies involving hundreds of patients have demonstrated that Symmetrel has a high degree of safety and possible immediate influenza A_2 protection when taken following suspected contact. It may be particularly useful in epidemics and for high-risk patients in whom the occurrence of influenza A_2 is especially hazardous.

Symmetrel is not indicated for the prevention of influenzal or respiratory illness other than influenza A_2 or for the treatment of established disease.

ENCYCLOPAEDIA BRITANNICA Booth 46

Chicago, Illinois

Encyclopaedia Britannica welcomes delegates to the California Medical Association and invites them to examine the great new edition of Britannica.

Official delegates may now purchase this magnificent set at an offer only available at our convention exhibits.

ENDO LABORATORIES INC. Booth 91

Garden City, New York

Endo Laboratories will present the latest clinical information relating to our products, Coumadin® (sodium warfarin), Numorphan® (oxymorphone) HCL, Percodan®, Percodan®-Demi, Hycomine®, Hycomine®-Compound, Hycodan®, Valpin® (anisotropine methylbromide), Valpin®-PB (anisotropine methylbromide with phenobarbital).

ESTA MEDICAL LABORATORIES, INC. Booth 79

New Brunswick, New Jersey

FACTS AND COMPARISONS, INC. Booth 29

St. Louis, Missouri

Facts and Comparisons is a drug information service consisting of:

1. Loose-leaf book listing nearly 6,000 drug products.
2. Monthly revised replacement pages to keep book perpetually up-to-date.

Unique features designed to give fast answers include:

Groups—Products are listed according to use for immediate comparisons of similarly used products.

Tables—Permit at-a-glance comparisons.

Reference Facts—Concise, pertinent reference information is given about actions, uses, side effects, contraindications and precautions of the drugs listed.

Indexed by generic name, trade name or usage.

Everything in one book, in its proper place. Contains no advertising.

FELLOWS-TESTAGAR Booth 44

Detroit, Michigan

Fellows-Testagar features: Amodex Timed Capsules (anorexigenic); Pentyate Timed Capsules (vasodilator) and Femogen Tablets (conjugated estrogens). Of special interest will be our new sampling technique—the RxAmple Program—the modern and convenient physician starter sample method.

Stop by for an official copy of the HR-2 Drug Abuse Law.

CHARLES O. FINLEY & COMPANY, INC. Booth 3

Los Angeles

California Medical Association Disability Insurance Program—Protect your most valuable asset, "Your Earning Power." New rates for the younger doctor. Applicants to California Medical Association are now eligible to apply. Private flying now covered.

FLINT LABORATORIES Booth 52

Morton Grove, Illinois

Featured Products are Synthroid® and Hu-Tet® . . . Synthroid (sodium levo-thyroxine) Tablets and Injection, the active principle of the thyroid gland, prepared synthetically in pure crystalline form for predictable and stable therapeutic results. Hu-Tet® (Tetanus Immune Globulin–Human), tetanus antitoxin of human origin for virtual freedom from sensitivity reactions. Now available from Flint Laboratories in 250-unit vials for economical and long-lasting protection.

FULLER PHARMACEUTICAL COMPANY Booth 87

Minneapolis, Minnesota

Featuring—"The Tucks Family of Products."

Tucks, medicated wet dressings for use in the management of pruritus ani, hemorrhoidal or perineorrhaphy discomfort, and other anorectal disorders.

Tucks witch hazel Ointment and Cream. Tucks witch hazel Cream HC. Tucks Take-Alongs (traveling companions to regular Tucks pads; packaged in individual foil packets).

A.R.D., butterfly-shaped anorectal dressings useful in the treatment of pruritus ani. Its unique shape is cut to conform to body contour and will remain in place without adhesive tape.

Fuller Shield, protective dressing to maintain anal, perianal or sacral dressings comfortably in place without tape.

Scopettes, proctosigmoidoscopic swabs.

GERBER BABY FOODS **Booth 27**
Fremont, Michigan

You are cordially invited to visit our exhibit. You will be briefly but reliably informed of recent developments in the only *complete* line of infant foods. New editions of *"Nutritive Values"* and *"Ingredients"* of Gerber baby foods are among a number of specialized services available for your office. Attractive packets of individualized food lists for your food-sensitive babies are just off the press and ready for your order. Please stop by.

GUARDIAN CHEMICAL CORPORATION **Booth 86**
Long Island City, New York

The company will exhibit its Clorpactin WCS-90 (sodium oxychlorosene) to prevent postoperative infections and in the treatment of antibiotic-resistant infections; Renacidin, for irrigating indwelling urethral catheters and the urinary bladder to prevent or remove encrustations due to calcification; and pHos-pHaid, for urinary acidification in order to increase calcium solubility and assist in preventing formation of calcifications in the urinary tract.

J. H. HEDRICK & CO. **Booth 106**
San Gabriel

Designers and builders of medical-dental buildings, as well as other office buildings, motels, and apartments. Home office in San Gabriel, California. Branch offices in South San Francisco, Santa Barbara, and San Diego, California.

KENWOOD LABORATORIES, INC. **Booth 28**
New Rachelle, New York

Featured in our display is Tablets Papavatral-20, a combination of a vasodilator, pentaerythritol tetranitrate and smooth muscle relaxant, ethylpapaverine HCL (ethaverine hydrochloride) for the aid in the treatment of angina.

Also featured is Elixir with Vitamin B_{12} which is indicated in nutritional and iron deficiency anemias (hypochromic anemia), where a hematinic and nutritive tonic is required. It is well tolerated, non-irritating, readily assimilated, and economical.

KEY PHARMACEUTICALS, INC. **Booth 62**
Miami, Florida

The following will be displayed: 1. Hyasorb penicillin—a sustained-action, 250,000-unit, penicillin tablet, producing detectable 10-hour blood levels. 2. Nitroglyn—sustained-action nitroglycerine, 1/50 gr, 1/25 gr, and 1/10 gr. Two to three tablets daily help prevent angina. Also, Nitroglyn-Sublingual, 1/100 gr, 1/150 gr, 1/200 gr, and 1/400 gr. 3. Proternol—sustained-action isoproterenol HCl, 30 mg tablets for Stokes-Adams and heart-block patients. 4. Theo-Nar—two or three tablets daily for long-acting relief of bronchial asthma symptoms. 5. Sedutain—delayed-release sodium secobarbital hypnotic.

LEDERLE LABORATORIES **Booth 10**
Pearl River, New York

Lederle Laboratories is proud to support the 1967 Annual Session of the California Medical Association. As one of the leaders in medical research and quality controlled production, we present for your consideration products such as Declomycin® (demethylchlortetracycline HCl), Achromycin®V (tetracycline), Aristocort® (triamcinolone), Levoprome® (methotrimeprazine) and others applicable to your practice. Our representatives in Booth No. 10 are also prepared to provide information on our numerous services to medicine.

ELI LILLY AND COMPANY **Booth 6**
Indianapolis, Indiana

You are cordially invited to visit the Lilly exhibit. Our sales representatives in attendance will welcome your questions about Lilly products. You may be particularly interested in discussing Keflin® (sodium cephalothin).

LOMA LINDA FOODS **Booth 78**
Riverside

Loma Linda Foods, one of America's oldest manufacturers of fiber-free, hypoallergenic infant soy milk, will show evidence of the nutritional adequacy of the product Soyalac. Representatives will explain why it is unusual in that it does not settle out, is milk-like in texture, and does not tend to raise infants' serum cholesterol. Uses for adult ulcer patients and in cholesterol-lowering diets will be discussed. Samples will be served and recipes presented indicating the versatility of the product in diets of those with milk allergies or who for other reasons need to eliminate dairy milk from the diet.

LOV-E' BRASSIERE COMPANY **Booth 111**
Hollywood

You are invited to view our complete line of custom-fitted brassieres designed to provide corrective support for specific breast conditions in exact accordance with the physician's instructions. We also would like to show you our new and improved breast prosthesis, the Lov-E' "Twin" breast form. This new form provides adjustable weight for perfect balance, ideal for all occasion wear, custom-fitted and weight adjusted to the individual's need. Our special representatives will be happy to greet you and answer any questions.

MEAD JOHNSON LABORATORIES **Booth 83**
Evansville, Indiana

The Mead Johnson Laboratories' exhibit has been arranged to give you the optimum in quick service and product information. To make your visit productive, specially trained representatives will be on duty to tell you about their products.

THE MEDICAL PROTECTIVE COMPANY **Booth 26**
Fort Wayne, Indiana

MEDINA AIRCRAFT COMPANY **Booth F**
Long Beach

Piper Aircraft display materials with emphasis on the $5 Introductory Flight Lesson as nationally advertised.

Eighteen Southern California Piper Aircraft Dealers will be represented.

MERCK SHARP & DOHME **Booth 84**
West Point, Pennsylvania

The Merck Sharp & Dohme exhibit has been designed to supplement the physician's therapeutic armamentarium. Technically trained personnel are present to discuss the scope and variety of services offered.

MILEX-FERTILEX CO. Booth 105
Los Angeles

MISSION PHARMACAL COMPANY Booth 70
San Antonio, Texas

Mission Pharmacal Company will be displaying its entire line of exceptional pharmaceutical specialties. Among these products will be Mission's new Liquitab line of pharmaceutical products with characteristics similar to liquids in the convenient chewable tablet dosage form.

THE NETTLESHIP COMPANY OF LOS ANGELES Booth 85
Los Angeles

Administrators of Professional Liability, Group Accident and Sickness, and Life Insurance Programs for County Medical Associations and Trusts in California.

Qualified representatives available to discuss problems pertaining to hospital or individual professional liability coverage, accident and sickness, life, or other types of insurance.

Literature which will assist in the prevention of claims and various forms to be used to protect, as far as possible, against malpractice claims.

NEUTROGENA CORP. Booth 75
Santa Monica

Neutrogena is a mild, transparent, ethanolamine base soap that contains no free alkali. It is not a detergent nor is it medicated; yet so different it was granted a U.S. patent (No. 2,820,768). Neutrogena is mild, not drying and not irritating. Whatever the etiology of a dermatosis, Neutrogena is well tolerated in the treatment of subsequent cutaneous irritation and inflammation. May be used in any area of medicine where washing is part of the treatment . . . especially where washing with soap is contraindicated.

ORGANON INC. Booth 94
West Orange, New Jersey

ORTHO PHARMACEUTICAL CORPORATION Booths 23 & 40
Raritan, New Jersey

Welcome to Booth No. 23 where Ortho® is proud to present the most complete line of medically accepted products for the control of conception. Of special note are our two latest products of the Ortho® Research Foundation—Ortho-Novum® Tablets and Delfen® Vaginal Cream and Foam.

Also on display at Booth No. 40, will be our well known products for the treatment of various forms of vaginitis.

Your questions will be welcomed by representatives in attendance.

PARKE, DAVIS & COMPANY Booth 17
Detroit, Michigan

Medical service members of our staff are in attendance at our booth to discuss important Parke, Davis specialties that will be on display.

PASADENA RESEARCH LABORATORIES, INC. Booth 71
Pasadena

Pasadena Research Laboratories will feature a variety of appetite control products, such as our Curban and Levatrol (L-amphetamine) time release capsules and also Indorex tablets.

We will be pleased to greet our many friends of the medical profession and to meet all the attending members at our display Booth No. 71.

PFIZER LABORATORIES Booth 20
New York, New York

The Pfizer Laboratories' display has been specifically arranged for your convenience and to give you the maximum in quick service and product information.

To make your visit worthwhile, technically trained Medical Service Representatives will be on hand to discuss with you the latest developments in Pfizer research.

WM. P. POYTHRESS & CO., INC. Booth A
Richmond, Virginia

The Poythress exhibit will feature:

Trocinate, a new concept in smooth muscle spasmolysis, acting directly upon muscle cells. Not an anticholinergic. No side-effects characteristic of autonomic blocking drugs. High therapeutic index. (No untoward effects in 16 years of clinical use.) Trocinate is particularly effective against functional diarrhea, mucous colitis, diverticulitis, spastic ureteritis and bladder spasms.

Mudrane combinations, established Poythress products for relief of asthma, emphysema, bronchiectasis and chronic bronchitis.

Solfoton maintains mild, continuous sedation around the clock over long periods of time without the patient's usual awareness of sedation.

RIKER LABORATORIES Booth 54
Northridge

Representatives of Riker Laboratories will be glad to supply you with complete information concerning our products which are available to the medical profession. Please feel free to request answers to any questions you may have. We will be looking forward to your visiting our booth.

A. H. ROBINS COMPANY Booth 11
Richmond, Virginia

You are cordially invited to visit the Robins display and meet our representatives who welcome the opportunity to discuss products of interest with you.

ROCHE LABORATORIES Booth 5
Nutley, New Jersey

Continuing Roche research has produced outstanding contributions to medicine since 1909. The personnel at the exhibit welcome your comments, questions or suggestions about our products and services.

J. B. ROERIG & COMPANY Booth 22
New York, New York

SANDOZ PHARMACEUTICALS Booth 19
Hanover, New Jersey

Sandoz Pharmaceuticals cordially invites you to visit our display at Booth No. 19 where we are featuring Mellaril (thioridazine, N.D.), Cafergot P-B, Sansert (methysergide maleate, N.D.), Fiorinal and Fiorinal with codeine. Any of our representatives in attendance will gladly answer questions about these and other Sandoz products.

W. B. SAUNDERS COMPANY Booth 4
Philadelphia, Pennsylvania

SAUNDERS will have on display a complete line of their medical books, including many new titles.

SCHERING CORPORATION Booth 68
Bloomfield, New Jersey

SCHERING CORPORATION invites you to visit their exhibit, Booth No. 68, where their representatives will be available to discuss with you any questions you may have on VALISONE™, ETRAFON®, GARAMYCIN®, TINACTIN®, CELESTONE®, SOLUSPAN®, AFRIN® or any other Schering product.

G. D. SEARLE & CO. Booth 13
Chicago, Illinois

You are cordially invited to visit the SEARLE booth where our representatives will be happy to answer any questions regarding SEARLE products of research.

Featured will be OVULEN for ovulation control and menstrual disturbances; and FLAGYL, a potent, new trichomonacidal agent for trichomonal vaginitis, cervicitis, urethritis and prostatitis.

SIEMENS MEDICAL OF AMERICA INC. Booth 92
Addison, Illinois

Medical technology has brought about great advancements. The SIEMENS line, on display, offers the latest in diagnostic and therapy equipment. A few minutes at our exhibit will be most informative. May we look forward to greeting you.

SIERRA MEDICAL SALES Booth 30
Riverside

DIAPULSE therapy provides safe and effective treatment through stimulation of normal body defenses. DIAPULSE is of particular value in increasing peripheral blood flow and accelerating wound healing.

THE SILVER HILL FOUNDATION Booth 101
New Canaan, Connecticut

The SILVER HILL FOUNDATION, New Canaan, Connecticut, a residential psychiatric treatment and rehabilitation center, will display pictures of patient residential and treatment facilities with descriptive copy. Brochures and informational material will be available.

SMITH KLINE & FRENCH LABORATORIES Booth 95
Philadelphia, Pennsylvania

Featured will be our comprehensive oral diuretic, DYAZIDE, each capsule containing 50 mg of DYRENIUM (brand of triamterene) and 25 mg of hydrochlorothiazide.

SMITH, MILLER & PATCH, INC. Booth 74
New York, New York

SMITH, MILLER & PATCH, INC. will appreciate the opportunity to discuss with you the latest clinical reports concerning LIPOFLAVONOID in vertigo associated with Meniere's disease and in dizziness in the older age group; VITRON-C, as an exceptionally effective well-tolerated and economical hematinic; KONDREMUL and LIPOTRIAD. Also featured are some ophthalmic products which are comforting, safe and effective in many minor eye irritations frequently encountered in general practice.

E. R. SQUIBB & SONS Booth 8
New York, New York

STACEY'S Booth 15
Palo Alto

STACEY's, one of the largest distributors of scientific and professional books in America, will be in attendance with a large display featuring the latest important books from *all* publishers, plus "up-to-the-minute" information about forthcoming books in each specialty. Be sure to stop by STACEY's Booth No. 15.

THE STUART COMPANY Booth 21
Pasadena

A cordial invitation is extended to all members and guests attending this meeting to visit THE STUART COMPANY booth. Specially trained representatives will be in attendance to answer your questions on new products, developed in our modern laboratories, which have particular interest for the medical profession. Products featured are DIALOSE®, DIALOSE PLUS®, FERANCEE®, MULVIDREN®-F, MULVIDREN® JUNIOR, MYLANTA®, MYLICON®, STUART FORMULA®, STUART PRENATAL®, and STUART PRENATAL®-F.

SYNTEX LABORATORIES, INC. Booths 9 & 69
Palo Alto

NORINYL® (norethindrone 2.0 mg with mestranol 0.1 mg) tablets, an original steroid from SYNTEX LABORATORIES, will be featured at Booth No. 69. NORINYL provides multiple contraceptive action that has produced a record of 100 per cent effectiveness.

Physicians are invited to register at the SYNTEX exhibit for complete information on this outstanding product.

SYNALAR® (fluocinolone acetonide), the topical corticosteroid designed to meet specific dermatologic needs, will be featured at Booth No. 9. SYNALAR has set a new standard of success in the treatment of a wide range of inflammatory dermatoses.

A warm invitation is extended to all physicians attending this meeting to visit our booth and discuss the latest developments from SYNTEX research.

TRU-EZE MFG. CO., INC. Booth 88
Burbank

See demonstrated the complete versatility of the TRU-TRAC Portable Intermittent Traction Machine, RT-99 Traction and Therapy Table and the FTC-20 Flexion Traction Chair. Let us show you how to utilize your present treatment table or x-ray table for horizontal cervical traction with the TS-93 Traction Stand. On display will be economical sets for patients requiring home treatment plus the original Jackson CERVIPILLO for the treatment of painful neck conditions.

UNION MUTUAL LIFE INSURANCE COMPANY Booth 47
Los Angeles

THE UPJOHN COMPANY Booths 56 & 57
Kalamazoo, Michigan

Professional representatives of THE UPJOHN COMPANY are eager to contribute to the success of your meeting. We are here to discuss with you products of UPJOHN research that are designed to assist you in the practice of your profession. We solicit your inquiries and comments.

WESTWOOD PHARMACEUTICALS Booth 45
Buffalo, New York

Westwood Pharmaceuticals invites physicians to stop by their booth to discuss their unique dermatological products.

Fostex Cream, Sebulex, Alpha-Keri, Fostex Cake, Fostril, and Keri Lotion.

These products are particularly suitable for personal use by physicians and their families who may be plagued with dandruff, acne, dry and itchy skin, and sensitivities to soap. Register, so that we may send prescription units to your home.

WINE GROWERS OF CALIFORNIA Booth C
San Francisco

WINTHROP LABORATORIES Booth 77
New York, New York

Winthrop Laboratories cordially invites you to visit Booth No. 77 where we will feature the following products:

Wingel—antacid liquid and tablets (hexitol-stabilized aluminum and magnesium hydroxides); neutralizes 300 times its weight in gastric acid for fast and prolonged relief.

Neggram (brand of nalidixic acid).

Isuprel (brand of isoproterenol) Mistometer.

WTS-PHARMACRAFT Booth 89
Division Wallace & Tiernan Inc.
Rochester, New York

Desenex: Most widely prescribed fungicide used in the prevention and treatment of athlete's foot and other superficial fungus infections of the skin.

Caldesene: Provides a lubricating and emollient film which is effective in the prevention or treatment of diaper rash and minor skin irritations.

WYETH LABORATORIES Booth 7
Philadelphia, Pennsylvania

Wyeth will feature . . .

Unit-dose dispensing—developed by Wyeth for a large number of orally administered medications. Reduces number of possible dispensing errors, eliminates exposure of medication to soil, moisture, and other contaminants. Three lightweight convenient storage forms are available—Redipak®, Soft-Pack and Bulk-Pack;

New Serax® (oxazepam) Wyeth, Capsules;

New Omnipen® (ampicillin) Wyeth.

Full information is available at Booth No. 7.

Scientific Exhibits

THE AMERICAN ASSOCIATION OF BLOOD BANKS	Booth 1
CARDIAC ENZYMES IN DIAGNOSIS OF MYOCARDIAL INFARCTION	Booth 2
AMERICAN MEDICAL ASSOCIATION FOOD AND GASTROINTESTINAL DISORDERS—NUTRITION	Booth 58
SUBCLAVIAN TECHNIQUE OF CENTRAL VENOUS PRESSURE MONITORING	Booth 59
BONE MARROW STUDIES—DIAGNOSTIC IMPORTANCE IN NON-HEMATOLOGIC CONDITIONS	Booth 60
TRANSURETHRAL PROSTATECTOMY: THE UROLOGIST'S CHOICE	Booth 63
HUMAN GROWTH HORMONE (HGH)	Booth 64
AIRWAY RESISTANCE AS AN INDEX OF EFFECTIVENESS OF BRONCHODILATING DRUGS	Booth 65
IS THE "SQUARE CUT TOENAIL" A FALLACY	Booth G

CALIFORNIA
MEDICAL
ASSOCIATION

JAMES C.
MacLaggan
president

JOHN G.
Morrison
president-elect

OFFICERS AND DELEGATES

General Officers

James C. MacLaggan, San Diego	*President*
John G. Morrison, San Leandro	*President-Elect*
William F. Quinn, Los Angeles	*Speaker, House of Delegates*
Joseph W. Telford, San Diego	*Vice-Speaker, House of Delegates*
Carl E. Anderson, Santa Rosa	*Chairman of Council*
Helen B. Weyrauch, San Francisco	*Secretary*
Dwight L. Wilbur, San Francisco	*Editor*
Howard Hassard	*Executive Director*
Peart, Hassard, Smith & Bonnington	*Legal Counsel*

House of Delegates

TOTAL DELEGATES (361)

James C. MacLaggan, San Diego	President
John G. Morrison, San Leandro	President-Elect
William F. Quinn, Los Angeles	Speaker, House of Delegates
Joseph W. Telford, San Diego	Vice-Speaker, House of Delegates
Carl E. Anderson, Santa Rosa	Chairman of Council
Helen B. Weyrauch, San Francisco	Secretary
Dwight L. Wilbur, San Francisco	Editor

COUNCILORS

Roger C. Isenhour (1967)	First District
Henry V. Eastman (1967)	Office No. 1, Second District
Frank C. Melone (1968)	Office No. 2, Second District
Malcolm C. Todd (1968)	Office No. 1, Third District
Elmer F. Gooel (1969)	Office No. 2, Third District
Richard L. Taw, (1967)	Office No. 3, Third District
Lewis T. Bullock (1968)	Office No. 4, Third District

DELEGATES EX-OFFICIO (73)

Joseph P. O'Connor (1969)	Office No. 5, Third District
Franklin F. Ham (1967)	Office No. 6, Third District
Wilbur G. Rogers (1968)	Office No. 7, Third District
Jean F. Crum (1969)	Office No. 8, Third District
Joseph F. Boyle (1967)	Office No. 9, Third District
Joseph F. Maguire (1967)	Fourth District
Ralph W. Burnett (1969)	Fifth District
Richard S. Wilbur (1967)	Office No. 1, Sixth District
Albert G. Miller (1968)	Office No. 2, Sixth District
Malcolm S. M. Watts (1969)	Office No. 1, Seventh District
Roberta Fenlon (1967)	Office No. 2, Seventh District
Harold Kay (1969)	Office No. 1, Eighth District
William F. Kaiser (1967)	Office No. 2, Eighth District
Carl E. Anderson (1967)	Ninth District
James H. Yant (1968)	Tenth District
Forest J. Grunigen (1967)	Eleventh District
William P. Longmire, Jr. (1967)	Scientific Board Representative

ELECTED DELEGATES (265)

Delegates	*Alternates*
ALAMEDA-CONTRA COSTA (19)	
Ackerman, Frederick	Adams, Robert
Anderson, Conrad E.	Barron, Gilbert
Armstrong, James R.	Byers, Gilbert
Bassett, J. Brandon	Charvet, Leonard W.
Black, Daniel W.	Duffy, Charles C.
Davis, Aaron E.	Goetsch, Carl
Donald, William G., Jr.	Harms, Herbert E.
Eisenberg, Harold J.	Kirk, Ralph
Hart, Charles J.	Maloney, Harold
Holden, Herbert	Miller, A. Donald
Hoskins, H. Dean	Morrison, J. Evans
Hudson, Charles B.	Powell, Oscar M.
Kerns, Claude	Reedy, Richard N.
Kunkel, Peter	Richards, Dexter
Lewis, Gwilym B.	Rosen, Edgar
Purcell, Edward F.	Ross, Joseph
Rihn, Richard J.	Thompson, Preston G.
Truman, Stanley R.	Whiting, E. Gale
Twigg, Edward	Wiesinger, Warren E.
BUTTE-GLENN (2)	
Elmendorf, Thomas	Chiapella, William C.
Murphy, Franklin L.	Ritter, Dale W.
FORTY FIRST (2)	
FRESNO (5)	
Argo, W. L.	Corbus, Howard
Howard, Arthur F.	Millar, Max S.
Kass, Robert	Steinberg, Theodore
Smith, Robb	Swift, Coe T.
Snyder, L. J.	Whitten, Richard

Delegates	*Alternates*
HUMBOLDT-DEL NORTE (2)	
Loring, Theodore	Bux, Donald E.
Henderson, Justus	Smith, E. Kenneth
IMPERIAL (2)	
Jaquith, George	Ajalat, M. P.
Schoensee, Burke	Worthman, Robert H.
INYO-MONO (2)	
Denton, Robert W.	Alm, Theodore
Hartwig, W. Ray	Christenson, Donald L.
KERN (4)	
Day, Robert L.	Anderson, Gene
Osell, L. N.	Clark, M. Marlin
Strongin, Seymour	Friend, James R., Jr.
Vaughan, J. E.	Reese, Thomas V.
KINGS (2)	
Brookshier, R. W.	Barreiro, A. L.
Kerr, Edwin E.	Dittes, William
LASSEN-PLUMAS-MODOC-SIERRA (2)	
Bross, Willard S., Jr.	Batson, Wilbur C.
Quinn, William J.	Christian, Maynard
LOS ANGELES (89)	
Adams, Donald A.	Alexakis, Peter
Allin, John G.	Anderson, James E.
Andersen, George C.	Anderson, Richard E.
Asher, Leonard M.	Arcadi, John A.
Attyah, Albert M.	Baker, Thomas
Austin, R. Reed	Beattie, Arthur S.

LOS ANGELES—*Continued*

Delegates	*Alternates*
Axelrod, Bernard	Berkaw, Kenneth
Bailey, Wilbur	Bleecker, Harry H., Jr.
Baker, Jack W.	Botzbach, Henry A.
Barber, Clifford A.	Brennan, John C.
Barnes, Roger W.	Brown, Albert F.
Barry, Donald J.	Bruch, John T.
Beasom, Ralph D.	Cheverton, William G.
Blood, William	Cobb, Dudley M., Jr.
Bowen, Gordon T.	Cobley, George G.
Boyle, Joseph F.	Deen, Richard
Buehler, George S.	Dorn, Robert M.
Bullis, John A.	Evans, Creed M.
Bullock, Lewis T.	Evashwick, George
Burke, Donald E.	Feinberg, Munish
Campion, George M.	Feldman, David H.
Case, Walter G.	Fischer, Barton L.
Compton, Russell F.	Fisher, Neal R.
Cope, Jerome A.	Ford, James H.
Crane, Jay J.	Fox, James B.
Crum, Jean F.	Fox, Saul L.
Donahue, D. W.	Frankenfeld, Robert H.
Elshire, H. Donel	Fredericks, Robert E.
D'Orazio, Edward	Freidin, Morris
Dummer, Jerome	Furer, Stanford A.
Ellerbeck, Walter P.	Gates, Earl F.
Ellison, G. Roger	Golden, Robert F.
Fields, Albert	Goldstein, David
Fitch, Donald R.	Goodwin, William E.
Gondek, Frank R.	Greco, Donald J.
Gooel, Elmer F.	Hoffman, Peter L.
Halasey, Thomas	Horton, James W.
Hamel, Neal C.	Houts, Richard E.
Hanchett, Richard B.	Hull, Forrest E.
Haskell, M. M.	Jackson, Newton R.
Heiser, Saul	Karlan, Mitchell S.
Hill, Harry E.	Kawakami, Iwao G.
Hoffman, Eugene F.	Kelso, Raymond, Jr.
Hohl, Mason	Keltner, Mark R.
Hood, Robert T., Jr.	Kern, William H.
Jones, Henry A.	Lambert, Ross W.
Kiddie, Thomas	Lau, Michael W.
Kransdorf, Charles D.	Lefevre, Timothy M.
Kuntz, George S.	Lopez, Henry L.
Longmire, William P., Jr.	Marcus, Stanley
Ludwig, J. Lafe	Mauer, Edgar F.
MacInnis, Douglas N.	Mayne, John C.
Mailman, Richard H.	McCandless, Harrison
May, Lewis H. V.	McCleary, Jack E.
Miller, Richard D.	McLaughlin, Henry M.
Milliken, Ralph M.	Michels, Arthur G.
Morgan, Frank M.	Mueller, Robert L.
Morgan, Henry G.	Nippell, Robert M.
Murray, Gregory C.	Odou, Eugene R.
Murrieta, A. J., Jr.	Oetting, Henry K.
Neuenschwander, Robert S.	Olch, David I.
Ottalini, Guy Adams	O'Neill, J. Norman
Payne, J. Howard	Palmer, Robert H.
Pheasant, Homer C.	Parlour, Richard R.
Platz, Edward	Parris, Edward B.
Plechas, N. Peter	Penka, Ernest J.
Pollack, John V.	Penn, Sidney
Quinn, William F.	Peterson, John S.
Richards, Melvin	Rubell, Earl B.
Rogers, Frank A.	Silver, Bernard B.
Rothenberg, Sanford F.	Smiley, Douglas F.
Shapiro, Marvin J.	Stauffer, Floyd R.
Smart, Reginald H.	Summers, L. F.
Smith, Eldon E.	Tashma, Albert
Smith, Seth Willard	Taylor, William A.
Smith, Thayer, A.	Thom, John G.
Snell, Marvin V.	Turrill, Fred L.
Starr, Harvey E.	Vogel, Philip J.
Stragnell, Robert	Walter, LeRoy E.
Taw, Richard L.	Warren, Ward B.
Wagner, Joseph	Wasserman, John M.
Watson, Robert L., Jr.	Weiss, Benjamin J.
Weber, Robert A.	Weiss, Murray J.
Whipple, Winston	Westerbeck, Charles W.
Wiener, Thomas	Wong, Thomas A.
Woodruff, John H.	Woolley, Morton M.
Woolington, Sam S.	Wunderlich, E. E.
Yates, Paul D.	Zimmerman, Norval F.
Zinn, Willard J.	

MARIN (3)

Delegates	*Alternates*
Ablin, Arthur R.	Alderson, Joseph J.
McGee, John	Raney, Gus
Weden, Elmer A.	Wagner, Leo A.

MENDOCINO-LAKE (2)

Delegates	*Alternates*
Roberson, B. B.	Waring, William
Smalley, Robert B.	Wilson, L. B.

MERCED (2)

Delegates	*Alternates*
Jackson, Edward A.	Corbett, Thomas
McDowell, Bruce E.	Soderstrom, Edwin

MONTEREY (3)

Delegates	*Alternates*
Hull, Osman H.	Eldredge, Eugene E.
Klinefelter, Robert P.	Kandlbinder, A. F.
Turner, Joseph E.	Pulford, James H.

NAPA (2)

Delegates	*Alternates*
Brignoli, Walter H.	Ashley, Robert C.
Rose, Edgar Kash	Ledwich, Thomas

ORANGE (13)

Delegates	*Alternates*
Altman, Richard F.	FitzGerald, P. J.
Ball, Dexter T.	Fraide, Raul
Carroll, Vincent P.	Hastings, Charles M.
Eastman, Henry V.	Kammerman, Richard F.
Galbraith, Harold F.	Kay, Fred M.
Gerrie, Wallace A.	Martin, Walter H.
Graham, Ralph E.	McFarland, Philip H.
Hawkins, G. William	Morgan, Russell M.
Jett, Jim	Nielsen, David I.
Mosier, Laurance A.	Schneider, Shirley M.
Paul, Carl J.	Siemonsma, Harry L.
Plumb, Hugh J., Jr.	Stonestreet, Marshall P.
Price, J. B.	Thompson, Arthur F.

PLACER-NEVADA (2)

Delegates	*Alternates*
Dubin, Nathan	Olrich, Frank M.
Shamhart, H. William	Wauters, Clifford W.

RIVERSIDE (4)

Delegates	*Alternates*
Abbott, Donald	Kinney, William
Ivanoff, John C.	Lyons, John C.
Jones, Cecil	Silver, Harrison E.
Stone, Herman H.	Zweig, Robert M.

SACRAMENTO (7)

Delegates	*Alternates*
Berg, John A.	Babbin, George
Berman, A. E.	Hause, Donald P.
Cook, Orrin S.	Lambert, Lewis
Farley, James O.	Martin, James W.
Grayson, Charles E.	Rooney, Pierce A.
Horn, Carl E.	Smiley, Stanley
Pope, Glenn A.	Terry, Daniel

SAN BENITO (2)

Delegates	*Alternates*
Haruff, John J.	Quinn, Robert D.
Moore, Ernest	Yip, Roderick L.

SAN BERNARDINO (6)

Delegates	*Alternates*
Gibson, Thomas	Hill, Harold M.
Halburg, Clarence T.	Judd, Richard
Hendrickson, M. A.	Krikes, Nicholas P.
Miano, Ben D. A.	Sterling, Allen F.
Sprague, Charles P.	Wake, Donald K.
Varden, Arthur	Wilson, Richard J.

SAN DIEGO (13)

Delegates	*Alternates*
Carpenter, Walter F.	Brumbaugh, Simon C., Jr.
Fairchild, L. H.	Flaiz, John W.
Hippen, Robert L.	Ford, John R.
Hokr, William K.	Herrick, William C.
King, Ralph M.	Kirtland, Howard B., Jr.
Levy, Edward I.	Moore, Stanley A.
Messenger, Harold	Parkinson, Gaylord
Peck, Sam	Peabody, Homer D., Jr.
Robinson, Frank H.	Peck, J. Haddon A., Jr.
Rumsey, John M.	Pierangelo, Anthony J.
Tancredi, Chester	Plumb, Robert T.
Telford, Joseph W.	Tisdale, William
Youel, Milo A.	Wells, John J.

SAN FRANCISCO (20)

Delegates	*Alternates*
Baer, Charlotte C.	Baron, Shirley H.
Biskind, Gerson R.	Barrios, Xavier O.
Bonfilio, Nicholas D.	Bender, William T.
Clark, Albert G.	Cohn, Bradford
Combs, Robert C.	Cullen, Stuart C.
Feldman, Sanford E.	Erskine, John M.
Fullenlove, Tom M.	Fleming, Ruth
Gallagher, Donald M.	Hopp, Eugene S.
Gibbons, Henry, III	Hurwitz, Samuel
Herrod, Chester E.	Jacobs, Alvin H.
Herzog, George K., Jr.	Kiyasu, William S.
Lebo, Charles P.	Milburn, Lloyd J.
Overstreet, Edmund W.	Musser, Don C.
Pillsbury, Philip L.	Pevehouse, Byron C.
Saunders, John B. deC. M.	Rixford, Emmet L.
Schaffarzick, Ralph W.	Robinson, Saul J.
Schaupp, John B.	Rosenberg, Alan J.
Wayburn, Edgar	Salomon, Maurice S.
Webb, Eugene M.	Sirbu, Abraham B.
Williams, A. Justin	Webb, Gilbert A.

Delegates	*Alternates*
SAN JOAQUIN (4)	
Benn, James J.	Clark, Stanley A., Jr.
Harrington, Donald C.	Evans, Wesley
McNally, John	Morozumi, John
William, George	Salter, Robert
SAN LUIS OBISPO (2)	
Chambers, James R.	Cletsoway, Richard W.
Kirk, Stanley	Warren, L. H.
SAN MATEO (7)	
Brown, Henry A.	Aycinena, Juan
Fox, Norman C.	Healy, Francis A.
Hart, Ward L.	Kohn, Martin M.
Hills, Oscar W.	Larsen, William G.
Lindsey, Howard W.	Richanbach, Henry S.
Novak, Frank J.	Rossiter, Stanford B.
Saidy, John T.	Storli, Edgar A.
SANTA BARBARA (4)	
Dalton, James	Blanchard, John
McNiece, Kenneth	Domz, Casimir
Miles, Harold	Gebhart, William
Ziemba, Joseph	Heiges, L. E.
SANTA CLARA (13)	
Besson, Gerald	Blanchard, Leland
Boice, Clyde	Brown, James L.
Clark, William H.	Cramer, Harold
David, Burt L.	George, John W.
Fox, Leon P.	Houck, George
Giansiracusa, Frank	Hull, David I.
Liston, Edward	Johnson, Allen
McCort, James J.	Kaufman, S. Fred
Mitchell, Sidney	Manson, R. Morton
O'Neill, Robert J.	Nola, Vincent F.
Scarborough, C. Gerald	Parker, Malcolm
Silver, Emmanuel	Ramsay, George
Skillicorn, Stanley A.	Rowles, Donald
SANTA CRUZ (2)	
Mills, Richard G.	Monteith, Stanley
DePuy, J. L.	Standage, Harlow
SHASTA-TRINITY (2)	
Miller, Charles D.	Keye, John, Jr.
Polka, Michael G.	Vaupel, Vance
SISKIYOU (2)	
Geyman, John P.	Chappell, Harry
Schnack, William	
SOLANO (2)	
Gullock, Alvin H.	Garrett, Robert L.
Olson, William J.	Schmutz, Melvin A.
SONOMA (3)	
Barnett, Richard C.	Craven, Wayne
Clary, David T.	Dunn, William J.
Zieber, R. L.	Johnston, Richard T.
STANISLAUS (3)	
Nelson, William	Hatch, F. N.
New, David J.	Pote, William W.
Purvis, Robert	Woolley, J. S.
TEHAMA (2)	
Ingle, Gerald	Griffiths, Robert O.
Wolfe, Lynn E.	Jourdan, Harve
TULARE (2)	
Goettle, James W.	Brauner, E. P.
Lavers, George D.	Wulff, Verna
VENTURA (3)	
Huff, W. Cloyce	Hair, Charles M.
Moore, James W.	Hart, William L.
Rulfo, Henry J.	McAtee, Richard M.
YOLO (2)	
Sobeck, Frederick J.	Edmondson, Robert C.
Wilson, B. Kent	Vaughn, T. Neil
YUBA-SUTTER-COLUSA (2)	
Boyer, Warren	Cusick, George
Wright, Bayard A.	Wallace, Robert N.

EX-OFFICIO SCIENTIFIC BOARD (18)

Belford, William W.
Bettman, Jerome W.
Blanchard, Leland B.
Brockman, Seymour J.
Clinco, Arthur A.
Dillon, John B.
Farber, Eugene M.
Grayson, Charles E.
Goerke, L. S.
Jones, Ellis W.
Keeney, Edmund L.
McCandless, Carl M.
Petit, Donald W.
Prentiss, Robert J.
Richards, Victor
Rubin, David
Russell, Keith P.
Zaik, Edward J.
Halter, Bert L.
Kelly, Hobart M.
Stein, Justin J.
Wood, David A.

EX-OFFICIO PAST PRESIDENTS (23)

Peers, Robert A.	1935
Wilson, Harry H.	1940
Molony, William R., Sr.	1942
Schaupp, Karl L.	1943
Goin, Lowell S.	1944
Cline, John W.	1947
Askey, E. Vincent	1948
Kneeshaw, R. Stanley	1949
Cass, Donald	1950
MacLean, H. Gordon	1951
Green, John W.	1953
Morrison, Arlo A.	1954
Shipman, Sidney J.	1955
Charnock, Donald A.	1956
MacDonald, Frank A.	1957
West, Francis E.	1958
Reynolds, T. Eric	1959
Foster, Paul D.	1960
Bostick, Warren L.	1961
Wheeler, Omer W.	1962
Sherman, Samuel R.	1963
Doyle, James C.	1964
Teall, Ralph C.	1965

EX-OFFICIO HONORARY PAST PRESIDENT (1)

Murray, Dwight H.

CALPAC REPORTS

Biltmore Bowl

Immediately Following the Opening Session of the CMA House of Delegates

House of Delegates • 1967 Annual Session

AGENDA

Biltmore Bowl

Speaker............................William F. Quinn, Los Angeles
Vice-Speaker........................Joseph W. Telford, San Diego
Secretary.......................Helen B. Weyrauch, San Francisco

FIRST MEETING, Saturday, April 15, 1967, at 6:00 p.m.

ORDER OF BUSINESS

1. Call to order.
2. Report of Committee on Credentials, and Organization of the House of Delegates—Roll Call.
3. Announcements and approval of Reference Committees and Miscellaneous Announcements.
    (a) Committee on Credentials. (Delegates must register with the Committee.)
    (b) Reference Committee on the Reports of Officers, the Council, the Commissions and Standing and Special Committees. (Reference Committee No. 1.)
    (c) Reference Committee on Finance, to study and make recommendations to the House of Delegates on the budget submitted by the Council and appropriate committees, and the amount of dues for the ensuing year. (Reference Committee No. 2.)
    (d) Reference Committee on Resolutions and New and Miscellaneous Business. (Reference Committee No. 3.)
    (e) Reference Committee (No. 3A) on Resolutions and New and Miscellaneous Business.
    (f) Reference Committee on Amendments to the Constitution and Bylaws. (Reference Committee No. 4.)
    (g) Reference Committee on CPS Business.
4. Honored Guests.
    (a) Fifty-year Members.
    (b) Past Presidents.
    (c) Allied Health Groups.
5. Recognition of President of the Woman's Auxiliary to the CMA—Mrs. Samuel Bacon.
6. Address by President—James C. MacLaggan.
7. Report of the President—James C. MacLaggan.
8. Report of the President-Elect—John G. Morrison.
9. Report of the Speaker and Vice-Speaker of the House of Delegates—William F. Quinn and Joseph W. Telford.
10. Report of the Trustees of the California Medical Association—James C. MacLaggan.
11. Report of Physicians' Benevolence Fund, Inc.—James C. MacLaggan.
12. Report of the Secretary—Helen B. Weyrauch.
13. Report of the Editor—Dwight L. Wilbur.
14. Report of the Executive Director—Howard Hassard.
15. Report of the Controller—Lytton O. Hetland.
16. Report of Legal Counsel—Peart, Hassard, Smith & Bonnington.
17. Report of the Committee for Emergency Action—James C. MacLaggan.
18. Report of the Council—Carl E. Anderson, Chairman.
19. Report of CPS Board of Trustees—Richard L. Wilbur, Chairman, Board of Trustees.
20. Reports of Commissions.
    (a) Commission on Medical Services—Arthur F. Howard, Fresno.
    (b) Commission on Public Agencies—Albert G. Miller, San Mateo.
    (c) Commission on Community Health Services—Harold Kay, Oakland.
    (d) Commission on Communications—Malcolm C. Todd, Long Beach.
    (e) Commission on Professional Welfare—George K. Herzog, Jr., San Francisco.
    (f) Judicial Commission—Francis E. West, San Diego.
    (g) Commission on Allied Health Professions and Services—Forest J. Grunigen, Los Angeles.
    (h) Commission on Hospital Affairs—Bert L. Halter, San Francisco.
    (i) Scientific Board—William P. Longmire, Jr., Los Angeles.
21. Reports of Other Committees.
    (a) Bureau of Research and Planning—Samuel R. Sherman, San Francisco.
    (b) Role of Medicine in Society—Malcolm S. M. Watts, San Francisco.
    (c) Organizational Review and Planning—Ralph C. Teall, Sacramento.
    (d) Committee on Legislation—Dan O. Kilroy, Sacramento.
    (e) Finance Committee—Harold Kay, Oakland.
    (f) Medical Executives Conference—Rueben Dalbec, Los Angeles.
    (g) Delegates to the AMA—Burt L. Davis, Palo Alto.
22. Old and Unfinished Business.
23. New Business.

CALPAC REPORTS

Biltmore Bowl

Immediately Following the Opening Session of the CMA House of Delegates

SECOND MEETING, Tuesday, April 18, 1967, at 3:00 p.m.

(To be recessed and reconvened at 9:00 a.m., Wednesday, April 19)

ORDER OF BUSINESS

1. Call to order.
2. Supplemental report of Credentials Committee—Roll Call.
3. Introduction of President-Elect of Woman's Auxiliary—Mrs. William Flood.
4. Address by President-Elect—John G. Morrison.
5. Secretary's announcement of Council's selection of time and place for the 1968 Annual Session.
6. Election of Officers:
    - (a) President-Elect.
    - (b) Speaker.
    - (c) Vice-Speaker.
    - (d) Councilors (three-year terms):
        - (1) First District—Roger C. Isenhour, San Diego (term expiring).
          First District—San Diego County.
        - (2) Second District—Office No. 1—Henry V. Eastman, Tustin (term expiring).
          Second District—Imperial, Inyo, Mono, Orange, Riverside and San Bernardino Counties.
        - (3) Third District—Office No. 3—Richard L. Taw, Los Angeles (term expiring).
        - (4) Third District—Office No. 6—Franklin F. Ham, Van Nuys (term expiring).
        - (5) Third District—Office No. 9—Joseph F. Boyle, Los Angeles (term expiring).
          Third District—Los Angeles County.
        - (6) Fourth District—Joseph F. Maguire, Ventura, (term expiring).
          Fourth District—San Luis Obispo, Santa Barbara, and Ventura Counties.
        - (7) Sixth District—Office No. 1—Richard S. Wilbur, Palo Alto (term expiring).
          Sixth District—Monterey, San Benito, San Mateo, Santa Clara and Santa Cruz Counties.
        - (8) Seventh District—Office No. 2—Roberta Fenlon, San Francisco (term expiring).
          Seventh District—San Francisco County.
        - (9) Eighth District—Office No. 2—William F. Kaiser, Berkeley (term expiring).
          Eighth District—Alameda and Contra Costa Counties.
        - (10) Ninth District—Carl E. Anderson, Santa Rosa, (term expiring).
          Ninth District—Del Norte, Humboldt, Lake, Marin, Mendocino, Napa, Solano and Sonoma Counties.
        - (11) Eleventh District—Forest J. Grunigen, Los Angeles (term expiring).
          Eleventh District—Forty First Medical Society.
    - (e) Delegates to the American Medical Association: (Delegates and Alternates to the American Medical Association are elected for terms of two calendar years. The Delegates and Alternates to be elected at this meeting will serve for two calendar years starting January 1, 1968, except as otherwise noted).

      Incumbents:
        - (1) Francis E. West, San Diego (term expiring).
        - (2) Samuel R. Sherman, San Francisco (term expiring).
        - (3) Henry Gibbons, III, San Francisco (term expiring).
        - (4) John M. Rumsey, San Diego (term expiring).
        - (5) Eugene F. Hoffman, Los Angeles (term expiring).
        - (6) Warren L. Bostick, Los Angeles (term expiring).
        - (7) J. B. Price, Santa Ana (term expiring).
        - (8) Ralph C. Teall, Sacramento (term expiring).
        - (9) James C. Doyle, Beverly Hills (term expiring).
        - (10) Wilbur G. Rogers, Glendale (term expiring).
        - (11) Charles B. Hudson, Oakland (term expiring).
        - (12) New Office—To serve two-year term commencing immediately—January 1, 1967 through 1968.
    - (f) Alternates to the American Medical Association: (Terms of all incumbents expiring. All offices for two-year terms starting January 1, 1968, except as otherwise noted.)

      Incumbents:
        - (1) *Vincent P. Carroll, Laguna Beach (alternate to Francis E. West).
        - (2) Albert G. Miller, San Mateo (alternate to Samuel R. Sherman).
        - (3) A. B. Sirbu, San Francisco (alternate to Henry Gibbons, III).
        - (4) Carl E. Anderson, Santa Rosa (alternate to John M. Rumsey).
        - (5) Joseph F. Boyle, Los Angeles (alternate to Eugene F. Hoffman).
        - (6) Walter H. Brignoli, St. Helena (alternate to Warren L. Bostick).
        - (7) Donald C. Dodds, Oakland (alternate to J. B. Price).
        - (8) Robb Smith, Orange Cove (alternate to Ralph C. Teall).

---

*Council appointee to replace Nicholas V. Oddo, Deceased.

(9) Herman H. Stone, Riverside (alternate to James C. Doyle).
(10) Carl M. Hadley, San Bernardino (alternate to Wilbur G. Rogers).
(11) Robert L. Watson, Jr., Los Angeles (alternate to Charles B. Hudson).
(12) New Office—To serve two-year term commencing immediately—January 1, 1967 through 1968.

6. Election of CPS Trustees (three-year terms):
Report of CMA Council as Nominating Committee.
Incumbents, terms expiring:
   Mr. John Cowee, Berkeley.
   Mr. Thomas Hadfield, San Francisco.
   Donald C. Harrington, Stockton (ineligible for reelection).
   Mr. Clinton Fair, San Francisco.
   Milo A. Youel, San Diego (ineligible for reelection).

7. Announcements by Secretary.
Council's nominations of members of Commissions and Committees.
   (For approval by the House of Delegates.)

8. Reports of Reference Committees:
(a) Reports of Reference Committee No. 1 on Reports of Officers, the Council, Commission and Standing and Special Committees.
(b) Report of Reference Committee No. 2 on the Budget submitted by the Council and appropriate committees, and the dues for the ensuing year.
(c) Report of Reference Committee No. 3 on Resolutions and New and Miscellaneous Business.
(d) Report of Reference Committee No. 3A on Resolutions and New and Miscellaneous Business.
(e) Report of Reference Committee No. 4 on Amendments to Constitution and Bylaws.
(f) Report of Reference Committee on CPS Business.

9. Unfinished Business.
10. New Business.
11. Presentation of Officers:
President—Presentation of Plaque to President James C. MacLaggan.
President-Elect.
Speaker.
Vice-Speaker.
12. Approval of Minutes. (Committee to edit.)
13. Adjournment.

William F. Quinn, *Speaker*
Helen B. Weyrauch, *Secretary*

1966 CONSTITUTIONAL AMENDMENTS, FOR ACTION IN 1967

Six constitutional amendments were introduced in the 1966 House of Delegates and, under the terms of the Constitution, must lie on the table until the next regular meeting of the House of Delegates.

The proposed Constitutional amendments are required to be printed in two issues of California Medicine before they come before the House of Delegates for action.

CONSTITUTIONAL AMENDMENT NO. 1-66

PHYSICIANS' BENEVOLENCE FUND

Introduced by: Council

Resolved: That Article IV, Section 6, of the Constitution of the CMA be amended by adding the language in italics and deleting the language shown in parentheses so it shall read:

"Section 6.—Physicians' Benevolence Fund

"The Council shall determine from year to year, at the time of the Annual Meeting, (at least $1.00 out of) *the portion* of the annual dues paid by each active member of the Association (shall) *to* be allocated to the Physicians' Benevolence Fund, Inc., a corporation. (and) *Funds so allocated* shall be used for the purposes as set forth in that corporation's articles and bylaws."

* * *

CONSTITUTIONAL AMENDMENT NO. 2-66

COMPOSITION OF THE COUNCIL

Introduced by: Council

Resolved: That Article III, Part B, Section 9, of the California Medical Association Constitution, be deleted and the following amendment be adopted:

"The Council shall consist of:

"(a) Elected councilors from the councilor districts set forth in Section 10. Each councilor district shall be entitled to elect one (1) councilor for each 1,000 active members, or major fraction thereof, according to its membership as of the first day of September of the preceding year; provided, that each councilor district shall be entitled to a minimum of one (1) councilor.

"(b) Elected councilors from any one district shall not, at any time, exceed 40 per cent of the total Council membership.

"(c) The president, president-elect, immediate past president, speaker and vice-speaker.

"(d) The secretary and editor, when they are members of the Association, and one member of the Executive Committee of the Scientific Board, who shall be elected by the Executive Committee of that body from representatives of the scientific sections or members-at-large. These three persons shall be ex officio members of the Council without the right to vote."

CONSTITUTIONAL AMENDMENT NO. 3-66

OFFICERS

Introduced by: Council

Resolved: That Article VI, Section 1, of the Constitution of the California Medical Association be amended by adding the language in italics and deleting the language shown in parentheses so that it shall read:

"Section 1.—Officers

"The officers of this Association shall be a President, a President-Elect, (a Secretary) *a Chairman of the Council, a Vice-Chairman of the Council,* a Speaker of the House of Delegates, a Vice-Speaker of the House of Delegates *and, when they are members of the Association, a Secretary and an Editor."*

* * *

CONSTITUTIONAL AMENDMENT NO. 4-66

CONSTITUTIONAL AMENDMENT TRANSFERRING AMADOR COUNTY TO FIFTH DISTRICT

Introduced by: Tenth District

Whereas, Article II, Part B, Section 10 of current CMA Bylaws places Amador County in Councilor District Number Ten; and

Whereas, only two of the 12 medical doctors licensed in Amador County maintain county society affiliation in a Tenth District society, the majority, because of travel patterns and hospital locations, preferring to affiliate with a county medical society in the Fifth District; and

Whereas, the Fifth and Tenth District delegations have endorsed such transfer of Amador County to the Fifth District; now, therefore, be it

Resolved: That Article II, Part B, Section 10 of the Constitution of the CMA be amended by adding the words in italics and deleting the words in parentheses, and to read:

"District Number Five, comprising *Amador,* Kern, Kings, Tulare, Fresno, Madera, Mariposa, Merced, Stanislaus, San Joaquin, Calaveras and Tuolumne Counties.

"District Number Ten, comprising Sacramento, (Amador,) Alpine, El Dorado, Placer, Nevada, Sierra, Yuba, Sutter, Yolo, Colusa, Glenn, Butte, Plumas, Tehama, Trinity, Shasta, Lassen, Modoc and Siskiyou Counties."

CONSTITUTIONAL AMENDMENT NO. 5-66

ESTABLISHING A SEPARATE COUNCILOR DISTRICT FOR THE COUNTY OF ORANGE

Introduced by: Orange County Medical Association

Resolved: That Article III, Part B, Section 10, Councilor District of the CMA Constitution be deleted and the following Section 10 substituted therefor.

"There are 12 districts as follows:

"District Number One, comprising San Diego.

"District Number Two, comprising Imperial, Riverside, San Bernardino, Mono and Inyo Counties.

"District Number Three, comprising Orange County.

"District Number Four, comprising the County of Los Angeles.

"District Number Five, comprising Ventura, Santa Barbara and San Luis Obispo Counties.

"District Number Six, comprising Kern, Kings, Tulare, Fresno, Madera, Mariposa, Merced, Stanislaus, San Joaquin, Calaveras and Tuolumne Counties.

"District Number Seven, comprising Monterey, San Benito, Santa Cruz, Santa Clara and San Mateo Counties.

"District Number Eight, comprising San Francisco County.

"District Number Nine, comprising Alameda County and Contra Costa County.

"District Number Ten, comprising Marin, Solano, Napa, Sonoma, Lake, Mendocino, Humboldt and Del Norte Counties.

"District Number Eleven, comprising Sacramento, Amador, Alpine, El Dorado, Placer, Nevada, Sierra, Yuba, Sutter, Yolo, Colusa, Glenn, Butte, Plumas, Tehama, Trinity, Shasta, Lassen, Modoc and Siskiyou Counties.

"District Number Twelve, consisting of any society which is not limited as to geographical area, or the area of which overlaps the area covered by one or more existing component societies; such society and its members shall not be considered to be members of any other councilor district."

* * *

CONSTITUTIONAL AMENDMENT NO. 6-66

REVISION OF DISTRICT ONE

Representing: Imperial
Introduced by: Burke E. Schoensee

Resolved: That Article III, Part B, Section 10 of the Constitution be amended by revising District Number One to include San Diego and Imperial Counties, and that Imperial County be deleted from District Number Two, and that otherwise said Section remain unaltered.

ANNUAL REPORTS FOR 1966

REPORT OF THE

Council

To the President and the House of Delegates:

The Council of the California Medical Association is responsible for managing the affairs of the organization; implementing the policies approved by the CMA House of Delegates at its annual meeting, and determining CMA policies between annual meetings of the House of Delegates.

The Council usually meets 10 times in regular session between meetings of the House of Delegates; this year a special meeting was called in November to consider recommendations for appointments to be made by the new State administration.

Minutes of all Council meetings are published in CALIFORNIA MEDICINE in the earliest possible issue after each meeting. Many details of Council considerations and actions are contained in these reports. Council highlights are published on the Monday immediately after each meeting in the weekly *Medical Executives Memo.* Additional details also are available from the Association's office and staff. Each member should feel free to request any desired information.

The composition of the Council is geographical, numerically uniform and representative of all major fields of practice of California physicians.

Reports by all standing commissions and committees and by special committees appointed for specific purposes may be given at each Council meeting.

By inviting representatives of other health-oriented organizations, officials of state agencies concerned with public health and deans of medical schools in California to its regular meetings, the Council maintains constant contact and communication with them. During the past year, attendance and interest by these organizations in the meetings of the Council have indicated their desire to maintain and build the liaison that has been developed.

Ample opportunity is given to all members and guests to speak freely and in detail. Officers of county medical societies are invited to attend each Council meeting. County society executive secretaries attend regularly.

Councilor attendance at meetings is usually 100 per cent with an occasional absence for good cause.

This report will not go into detail on Council actions but will discuss briefly some of the major areas of interest and activities which have engaged the Council during the past year.

1. *Actions of the 1966 House of Delegates.* The Council supervised the implementation of all resolutions adopted by the 1966 House of Delegates. Copies of all resolutions adopted and a report on the actions will be mailed in advance of the 1967 House of Delegates meeting to all members of the House. Wherever feasible, the author of each resolution has been contacted and the actions on his resolution made known to him. Where resolutions have been introduced in the name of a delegation, the chairman of the delegation and/or the major county society within that delegation area has been informed and has been requested to notify the appropriate author.

2. *Legislation and government programs.* Legislation and legislative activities dominated Council actions during 1966 and early 1967.

Medicare, Medi-Cal and related programs occupied much of the Council's time and attention. The Task Force on P.L. 89-97, which had carried most of the burden of watch-dogging, advising and educating for the association under the able guidance of Chairman Ralph Teall, was discharged with thanks and commendation.

- Among Medicare problems tackled by the Council was the reimbursement of physicians on the staffs of county and teaching hospitals. After surveying the ethics of various methods of payment and how payments actually were being handled, the Council adopted a statement on reimbursement that reaffirmed CMA policy that hospitals should not benefit financially from payment for physicians' services, and that physicians should not benefit from payments for hospital services.

- Soon after the implementation of Medi-Cal, the Council authorized two regional institutes to be held for review committees of county societies. The institutes explained and discussed "reasonable charges" under A.B. 5; the review mechanism used by CPS-Blue Shield in processing Medi-Cal claims; the role of Blue Shield as fiscal intermediary in the program, and other items of interest and confusion. Good attendance and lively discussion at both sessions indicated that these regional institutes were both needed and welcome.

- The Council also gave approval to a series of institutes that were held by the Joint Council to Improve the Health Care of the Aging. These institutes explained to persons concerned with extended care facilities the conditions of participation under Medicare.

- The Council also appointed a committee, at the request of the State Department of Public Health, to determine exactly what and where free-standing clinics are, and what the standards should be for their participation in Medi-Cal.

- The Council adopted the policy that physicians serving on review committees should not be compensated for their services, but pointed out that government funds can be used to defray administration expenses.

- A contract between CMA and the State Department of Public Health that would make the CMA Medical Staff Survey Committee available to act as consultants to hospitals with provisional certification for Medicare was authorized. Seventy-three hospitals in California fall into this class.

- A CMA Appeals Committee to review disputes between physicians and the fiscal intermediary that cannot be resolved on a county level was established by the Council.

- The Council urged adoption of a regulation that would allow registered physical therapists to be reimbursed for services under Medi-Cal.

- A committee was appointed to explore with CPS-Blue Shield ways that physicians could bill Medi-Cal patients directly.

- The Council reaffirmed that Crippled Children's Services should be integrated into the Medi-Cal program.

- Changes in Medi-Cal regulations to allow more liberal psychiatric benefits without prior authorization were endorsed by the Council.

Despite the almost overwhelming amount of time that was necessary to cope with Medicare and Medi-Cal problems, the Council did not neglect other legislative matters.

Soon after the House of Delegates meeting in April, the Council endorsed the delegates' action in urging broader *therapeutic abortion* laws and asked CMA legal counsel to draft legislation to be introduced to the State legislature in 1967.

The *California Committee on Regional Medical Programs,* a voluntary state-wide committee, was formed to implement recent national legislation (P.L. 89-239) for regional medical programs. Giving its support to this committee, the Council designated three members—James C. MacLaggan, John G. Morrison and Ralph C. Teall—as CMA representatives, and approved California Medical Education and Research Foundation as fiscal agent for the California committee.

The Council's ad hoc Committee on *Workmen's Compensation* announced that an adaptation of the 1964 *Relative Value Study* with a coefficient factor of $5.50 had been approved by the State Division of Industrial Accidents as a basis for payment of workmen's compensation cases.

A study by the Committee on Adverse Drug Reactions of the Scientific Board on *aplastic anemia deaths* in California was approved by the Council to be submitted to the State legislature. This study found that "no reasonable basis exists for enactment of a special legislative category to restrict the use of the drug chloramphenicol by licensed physicians in California . . . and the risk/use factor does not provide sufficient basis to single out this drug for special legislation."

The Council authorized preparation of a bill that would preserve the *medical confidentiality of teachers* who undergo legally required physical examinations when applying for a position in California's public school system.

The Council also endorsed legislation for a stronger *TB control/eradication program* in California.

3. *Public Health.* A strong position paper on *smoking and health* was adopted by the Council that (1) reaffirms that cigarette smoking is harmful to health and urges close cooperation with the Inter-Agency Council on Smoking and Health; (2) endorses the concept of mass public education campaigns; (3) recommends that CMA reinforce the need for physicians to instruct and advise patients and people not to smoke; (4) says the CMA should play a leading and active role in the Inter-Agency Council; (5) suggests CMA should encourage and assist more effective control measures; and (6) says that physicians who are convinced that cigarette smoking is dangerous should set an example of abstention.

The Council urged the State Board of Public Health to reinstate *rubella* as a reportable disease.

It recommended that county societies work with the State Department of Public Health in sponsoring *clinics for migrant workers.*

A *"lapse of consciousness"* regulation, earlier approved by the Council, was adopted by the State Board of Public Health.

4. *Allied Health Fields.* The year just past was an eventful one for *nursing*. CMA Council approved a statement giving support and encouragement to schools and hospitals that offer various courses leading to a degree or diploma in nursing; adopted a statement urging "adequate compensation for all members of the health care team," and encouraging the achievement of this goal in a professional manner within the framework of voluntary democratic processes; and approved recommendations from the Asilomar Conference on the Shortage of Nursing Personnel in California, which emphasized the need for better utilization of nursing service; realignment of nursing education; increased and more efficient production of nursing personnel; recruitment, retention and functioning of nurse manpower; continuation of the committee that produced these recommendations, and the formation of a voluntary group to carry out the recommendations of the conference.

The CMA Council asked the Board of *Licensed Vocational Nurse Examiners* to include physicians in LVN training programs, especially to explain medical ethics, and offered CMA help in this regard.

The Council approved a policy stating that the extent of routine laboratory procedures performed on *incoming hospital patients* should be determined by individual physicians and the hospital medical staff.

In emergency action, the Council requested the AMA Board of Trustees to defer action on a resolution that would have endorsed the opening of all hospital graduate training programs to graduates of schools of *osteopathic medicine.* The CMA asked to inform the AMA Board of Trustees of its experiences before the Trustees made a decision on this matter.

5. *Health Facilities Planning.* With government agencies keeping a close watch on area-wide health facilities planning, CMA has been encouraging more active physician participation. The Council approved preparation of a set of guiding principles for area-wide health facilities planning, the development of a brochure on the need for planning and agreed to sponsor, with California Hospital Association, teams of physicians/administrators to visit problem areas and areas where no planning exists.

6. *Committee on Mediation.* The Council defined the role of the Committee on Mediation. The Committee, the Council said, should consider cases referred by county society review committees, insurance carriers for commercial programs, nonmembers and cases where county society committees refused to act.

7. *Rural Health.* Rural health came into increasing prominence during 1966 and the Council gave approval to a program to attract physicians to rural areas proposed by the CMA Committee on Rural Health. Approval also was given to a plan that would allow medical students to work as externs on rural health projects during the summer months.

8. *Malpractice Insurance.* With the rates for malpractice insurance rapidly increasing, the Council assigned the Commission on Professional Welfare to study the problems. As a result of the preliminary study, a state-wide survey will be made to pinpoint problem areas.

In other actions during the year, the Council appointed a committee to look into CMA/county society relationships and approved the exploration of a Community Health Council, which would be made up of representatives from various community agencies interested in health.

Actions taken by the Council after the preparation of this report will be submitted in a supplemental report at the 1967 meeting of the House of Delegates in Los Angeles.

The Council is especially indebted to President James C. MacLaggan and President-Elect John G. Morrison, as well as to Immediate Past President Ralph C. Teall. All three have devoted an extraordinary amount of time, effort and talent representing the California Medical Association before any number of private and public bodies and working with our own committees and other organizations in this year of complex new government programs and problems of increasing scope. They have been unstinting in their services and to them we owe a debt of gratitude. The same applies to a number of Councilors, commission and committee members, AMA delegates and others who have given so freely of their time and talents in presenting the true picture of medical practice in California to so many interested listeners. Special thanks are also due our dedicated staff of loyal employees who must and do adjust to the demands of the moment. Without their devoted services the work of the Association could not be accomplished.

Respectfully submitted,
Carl E. Anderson, m.d., *Chairman*

REPORT OF THE

AMA Delegation

To the President and the House of Delegates:

I am pleased to report to you that we had a hard working, serious, respected and influential delegation at the 115th Annual Convention of the American Medical Association in Chicago. The attendance was excellent, with 42 out of our 44 delegates and alternates present. Our delegation met each morning at 7:30 in caucus and recessed to attend meetings of the House or of the reference committees for the balance of the day. On 29 June the House held a virtually unprecedented evening session which lasted until 11:00 p.m.

More than 150 recommendations stemming from resolutions and Board of Trustee reports were considered by the House. It was necessary to form two sections of the Reference Committee on Insurance and Medical Service—one just to handle the 30 items related to Medicare alone.

Of the 21 resolutions presented by California and of their fate, 50 per cent were approved and 25 per cent more were given other favorable action on consideration. As to the four which were not adopted, their intent was given extensive discussion and their arguments were well presented to the reference committees and to the House of Delegates. The subject of Federal Health Legislation created the most discussion, particularly the matters of direct billing, utilization review, compensation for service on such committees *only* in exceptional circumstances, and the proper placement of the onus for any failure in the Medicare Program.

Dr. Hudson, the incoming president, presented a four-point program:

1. To "demonstrate the competency of the private practice of medicine."

2. To stop the trend toward dependence upon government.

3. To encourage the government to stimulate and support private enterprise in the health field.

4. To "offer our government the knowledge and the competency of the health profession in consultation and cooperation, acting as free, strong independent agencies, not as extensions of government."

While considering the principles of billing for hospital-based physicians, the House was informed that Medicare Regulation No. 5 had been released by the Social Security Administration on 28 June —just three days before the effective date of the law. These regulations did not, in the opinion of Organized Medicare, conform to the intent of Congress as expressed in Section 1801 of P.L. 89-97.

The House instructed "the Board of Trustees and the Executive Vice President to request from the Social Security Administration an extension of date of final adoption of the proposed regulations of not less than 90 days, in order that the American Medical Association and all other interested medical organizations be allowed reasonable time to study, and to submit to the Social Security Administration data, views or arguments and pertinent constructive comments and suggestions."

The House further stated: "To preserve the professional independence of medical practice that the Board of Trustees and Officers of the AMA be instructed to immediately inform the membership that Medicare Reg. No. 5 will not apply to physicians (whether hospital-based or not) who

"1. have no financial relationship with a hospital covering medical services to patients;

"2. do not accept assignments but bill directly.

"*The AMA News* and other appropriate media be used to advise all physicians who are developing contractual relationships with hospitals for professional service that they should delay the finalization of any agreements pending further analysis of the implementing regulations."

On the subject of discrimination, the House directed Council on Constitution and Bylaws to prepare such changes as may be necessary to permit the Judicial Council to receive and act upon appeals filed by applicants who allege that they have been unfairly denied membership in a local and/or state society.

The House approved an increase in dues from $45 to $70 effective January 1967.

The CMA was honored by an award for the largest contribution to The American Medical Education and Research Foundation.

Section Delegates and Alternates who reside in California were invited to join in any caucuses with the delegation, since they also express the desires of California doctors. They were a welcome and constructive addition to our manpower.

In order to give proper consideration to the 106 resolutions, 38 reports of the Board of Trustees and more than 20 other reports, it was found necessary to establish two committees of our delegation. One committee, chaired by Dr. Eugene Hoffman, Sr., considered the proposals introduced by California. The other, chaired by Dr. Wilbur Rogers, considered resolutions introduced by other delegations. These groups met frequently, both separately and together, in order to give coordinated direction to the delegation's efforts.

Your delegation to the Clinical Convention of the American Medical Association in Las Vegas in November 1966 was highly successful in conveying to the House of Delegates, both on the floor and through its Reference Committees, the sentiments of the California Medical Association on the five resolutions which you and the delegation considered and approved for transmission to the AMA House. Jim Feldmayer, Pat Hudson, Mal Todd, Buck Rogers and I served on various reference committees. The resolution on AMA "Current Procedural Terminoloy" was given particular attention by the Reference Committee which reported back to the House that although the Reference Committee did not see a need for a new committee to evaluate this publication, as suggested in our resolution, the Committee felt that the present edition of "Current Procedural Terminology" was a preliminary document, admittedly incomplete, and not as inclusive as it must of necessity be if it is to serve as a desirable source of reference. It stated that this was not a static document, but is a dynamic one reflecting the changing nature of the practice of medicine, and must be kept so by pertinent and timely revision. This must be made clear in future distribution by including with each copy of the first edition distributed, an insert which clearly sets forth this information. This addition should allay some of the concern expressed. The Reference Committee noted that the first edition was published in a minimum length of time, due to various pressures, and felt that the ends sought by the California resolution were well on their way toward accomplishment under the present arrangements. Our resolution was not adopted, but the House instructed that future distribution of the first edition contain all of the explanatory inserts mentioned above. I might state at this point that the revision of "Current Procedural Terminology" is delegated to its Council on Medical Service and more specifically to the Committee on Insurance and Prepayment Plans. Your delegation was very happy to report that Doctor Homer Pheasant, Chairman of the California Committee on Fees, has been made a member of the Committee on Insurance and Prepayment Plans, and, therefore, although the resolution was not adopted *per se,* the spirit of the resolution was implemented. Your representatives could do no better than to have one of our own men who is vitally interested in this subject, as a member of the AMA committee that is going to have supervision over it.

Resolution 41 dealt with billing options under Public Law 89-97, Title 19. Five other resolutions of the same nature were considered together with it. The Reference Committee had been informed that the Council on Legislative Activities recently recommended that Title 19 be amended to permit reimbursement to a patient for payments made by him to his physician. The Board of Trustees had already approved this recommendation. The Committee agreed wholeheartedly with our resolution and submitted a substitute for all six which had been presented. The *resolve* portion of this states:

"Resolved, That the American Medical Association strongly support amendment of the Social Security Act including Title 19 to permit reimbursement of payments for medical care made by the patient to the provider of the services."

Report D of the Council on Medical Service brought up the question of usual, customary and reasonable charges, to which many members of the California delegation spoke. The House adopted the following statement:

"On a number of occasions the House of Delegates has expressed its preference that programs for financing physicians' services be based upon the concept of 'usual and customary charges' for professional services. Confusion exists because the words 'usual, customary and prevailing' are used in different ways by physicians, government and other agencies. Physicians have long used the word 'usual' in relation to charges made by an individual physician. In Public Law 89-97 such charges are referred to as 'customary.' Physicians in some areas have used the word 'customary' in relation to a full range of charges for the same professional services made by doctors in a geographical area. In other areas 'usual' and 'customary' are considered synonymous. In Public Law 89-97 such full range of charges is designated as 'prevailing.' This term is recognized as one which will continue to have application in that program's administration."

Our resolution dealing with the Millis Report was adopted by the House. There was a full and open hearing of the Millis Report under the aegis of the Board of Trustees, held at a time when the Reference Committees were not in session. Thus all members of the House and other interested parties were able to participate in the discussions. I am sure that all points of view were well represented. The Board of Trustees intends to have the Council on Medical Education hold further discussions and meetings with other interested individuals and organizations before recommendations are considered and made by the Board to the House next June.

The California delegation was commended by the House for introducing a resolution concerning packaged disaster hospitals and for its elaboration of the resolution before the Reference Committee. We made some minor changes in our resolution when it was being considered by the Reference Committee, and it was adopted. Our resolution on physician representation on governing boards of hospitals, was a reaffirmation of a resolution introduced last June pertaining to open staff hospitals. The Reference Committee recommended, and the House adopted as the amendment, that each hospital should have at least one doctor of medicine, who preferably should either be appointed or elected by the hospital medical staff from among its membership, as a full, voting member of the hospital's governing board.

The Reference Committee, of which I was chairman, received a 22-page report of the Board of Trustees dealing with procurement of physicians for military service and the setting up in the National Selective Service System, of a technique for making this selection through a proposed national commission on health resources and medical manpower. The Reference Committee strongly recommended, and the House concurred, that such a commission include strong representation of those most familiar with civilian medical needs—the physicians in active practice.

This same Reference Committee also received resolutions regarding nurses' compensation and nursing education, and while it advocated increase of nurses' compensation commensurate with training, location, degree of responsibility, etc., it did not feel that a national wage scale would be possible at this time. The Nursing Association had also requested that the AMA comment on the paper which advocated that all qualified nurses be trained through university-affiliated schools. The Committee and House did not agree, but recommended the continuing of the present type of nursing education, realizing, of course, that gradually there would be an increase of those who would come from universities, although at the present time 70 per cent of nursing care is rendered by graduates of diploma schools.

Respectfully submitted,
BURT L. DAVIS, *Chairman*

REPORT OF THE

Physicians' Benevolence Fund, Inc.

To the House of Delegates:

Physicians' Benevolence Fund, Inc., maintained its benevolent activities during 1966 in keeping with previous years. It continued to make monthly contributions to Los Angeles County Physicians' Aid and responded to requests for temporary assistance from physicians or their families throughout the state.

The Fund received $29,907 in income during the fiscal year ended 30 June 1966, and made expenditures of $25,671 during that period. The excess of income over expenses in the amount of $4,236 went directly into reserves. Total assets now stand at $183,258. Income in the amount of $29,907 was derived from $21,524 contributed by the California Medical Association at the rate of $1 from the dues of each active member, $4,007 contributed by the Woman's Auxiliary and $4,376 received in interest from investments and loans to affiliated organizations.

The Operating Committee periodically meets and reviews both policies and activities of the Fund. All contributions are reviewed and passed on by the Committee, based on immediate need and are not designed to constitute a retirement income program. All appeals are investigated at the local level and periodic reports on recipients are reviewed. All cases of real need are tactfully and sympathetically handled. The recipients are for the most part widows and children of physicians.

The Operating Committee consists of Clyde L. Boice, Chairman, and Doctors Elizabeth Mason Hohl, Dudley M. Cobb, Jr., Alexander Fraser, Don C. Musser and George Wolf. They have been continually interested and active in guiding the policies of the Fund. There are no paid employees of the Fund and the only operating expenses of the Fund are the cost of an annual audit and the cost of meetings of the Operating Committee.

An expression of appreciation is due the Woman's Auxiliary and the California Medical Association for their continued support in maintaining this benevolent assistance program for the temporary relief of our distressed physicians, their widows and children.

Respectfully submitted,

JAMES MACLAGGAN, M.D., *President*

REPORT OF THE

California Physicians' Service —Blue Shield—

To the President and the House of Delegates:

Membership

As of 31 December 1966, the membership of California Physicians' Service-Blue Shield totaled 1,154,011 persons, compared with membership of the preceding year of 1,158,078 persons. Membership figures would have shown a gain rather than the slight loss had it not been for termination of one very large union case representing 15,000 persons, plus the loss of about 7,000 members previously covered under the Santa Barbara Prepaid Medical Care Plan, which was absorbed by the new California Medical Assistance Program during 1966.

Physician Membership as of 1 January 1967, was 17,592, an increase of 142 physicians during the year 1966.

Financial

Standard Programs

Based on actual claims experience for the first 11 months of the year, plus estimated figures for December 1966, operating income for the year from California Physicians' Service standard programs was approximately $73,500,000. Disbursements under these programs amounted to approximately $64,000,000.

The stabilization reserve fund amounted to approximately $19,300,000 at year-end, based on actual figures for the first 11 months plus a projection for the month of December. This amount is equal to about four months of claims costs plus administrative expense, conforming to the reserve formula recommended by the National Association of Insurance Commissioners for Blue Shield and Blue Cross Plans. The reserve fund at the end of 1965 amounted to $18,430,000.

Government-Financed Programs

Medicare. Since the inception of the Social Security Medicare program on 1 July 1966, California Blue Shield had administered Part B (medical benefits) of Medicare throughout California, except for Los Angeles and Orange counties. As of 1 January, more than half a million Medicare claims had been processed, amounting to a payment of some $11 million.

Medi-Cal. The California Medical Assistance Program (Medi-Cal), California's welfare medical plan operating under Title XIX of Public Law 89-97, went into operation 1 March 1966. Blue Shield and Blue Cross contracted to jointly handle administration of this program, with the two California Blue Cross Plans handling institutional care benefits, while Blue Shield dealt with the professional services of physicians, dentists, druggists, and others.

At year's end the program was generating some 65,000 claims per day, and by the end of 1966 Blue Shield had processed more than nine million claims under the Medi-Cal program. During the first 10 months of operation under the program, $112,025,895 was paid as follows:

Physician services	$56,888,156
Other medical	14,427,967
Dental	18,189,220
Drugs	22,520,552

Military Dependents Medical Care Program. The Military Dependents program underwent several changes during the past year, including an official name change. The program is now officially known as the Civilian Health and Medical Program, Uniformed Services, or "CHAMPUS." As of 1 October, outpatient care benefits, including prescription drugs, were added to the program, and the maximum amounts payable were increased. During the calendar year 1966, a total of 69,857 claims were processed, representing payment of $5,394,303.

Fee Schedules—Standard Programs

The policy of updating and upgrading fee schedules followed for the past several years has resulted in the following distribution of the membership, as of 31 December 1966:

Schedule	*Conversion Factor*	*Per Cent of Membership*
A and B Schedules	......	0.1
1960 RVS—J Schedule	$ 4.00	5.2
1964 RVS—J Schedule	4.00	0.3
1957 RVS—C Schedule	5.00	0.5
1960 RVS—D Schedule	5.00	30.4
1964 RVS—D Schedule	5.00	37.1
1964 RVS—E Schedule	6.00	0.1
Extended Service D and H Schedule		
D	5.00	10.9
H	Usual Fee	
"Usual" Fee—No Schedule	......	6.6
Foundations for Medical Care Programs	......	7.1
Indemnity	......	1.7
TOTAL		100.0

With more than 92 per cent of the membership now based on schedules with a conversion factor of $5.00 or more, California Blue Shield is now in a position to upgrade schedules in an orderly fashion, and to equate them with cost factors in the economy without resorting to the crash tactics which in the past have proven costly in terms of cancellations. By comparison, one year ago 75 per cent of the Blue Shield membership was on a $5.00 unit or better.

California Physicians' Insurance Corporation

CPIC, the indemnity insurance company of which California Blue Shield owns all outstanding stock, attained a gross annualized premium income of $6,933,100 as of 30 September 1966, up from $5,460,300 on the corresponding date in 1965. Marketing group indemnity surgical-medical-hospital coverages, group major medical expense benefits, and group life policies, CPIC's assets on 30 September 1966 were $2,391,464 as compared with admitted assets of $2,010,137 on 30 September 1965. These asset figures are exclusive of furniture, fixtures and reserves for depreciation.

General

1966 was above all a year of great change for the California Blue Shield plan. The increase in workload is clearly demonstrated by the fact that 246,000 claims were processed during December 1965, while more than 1,250,000 claims were processed during the final month of 1966. While undertaking the two large, complex government programs, Medicare and Medi-Cal, vast internal changes in management and organization have taken place. R. S. "Rusty" Saylor, who had recently retired from the Indiana Blue Shield plan was named by the Board of Trustees in February to serve as Acting President while an intensive search was conducted for a permanent chief executive for California Physicians' Service. In August, Thomas C. Paton left his position as chief executive of the Minnesota Blue Shield plan to become President of California Physicians' Service. By year's end, Mr. Paton had built a new management team designed to cope effectively with the substantial tasks facing California Blue Shield.

Respectfully submitted,

RICHARD S. WILBUR, M.D.
Chairman of the Board
California Physicians' Service-
Blue Shield

REPORT OF THE

Controller

To the President and the House of Delegates:

This report is made in connection with the financial, accounting and membership activities and status of the California Medical Association and its affiliate organizations. The period covered by this report will be essentially the fiscal year ending 30 June 1966.

1. *General.* The Association maintains its principal office and records at 693 Sutter Street, San Francisco, California, a building owned by the Trustees of the California Medical Association, an affiliated corporation whose members are at all times the members of the CMA Council. While several tenants are housed in the building, the Association occupies approximately three-quarters of all usable space. An architectural study was made to determine the feasibility of expanding our present facilities, and it was determined that no additional floors could be added to the present structure. During the latter part of 1966 the Trustees of the California Medical Association purchased the three-story building adjacent to our present building as the only means of enlarging our present facilities and enhancing the value of the property, which now has a 90-foot frontage on Sutter Street in San Francisco.

The Los Angeles office has one executive and one secretary. The Sacramento office has one executive employee and one secretary and continues as a joint effort with the Public Health League of California, and has provided needed space to serve as headquarters for the Legislative Committee and legislative activities during legislative sessions and is available the year round for committee members and others whose assignments take them to the State Capitol.

Members of other professional groups comprising the Public Health League of California also find the new office a convenience throughout the year.

Membership in the California Medical Association has continued to increase and each month continues to see a new high point in total membership. As pointed out in previous reports over the past years, increased membership brings with it increased demands for service. Added services and projects mean added personnel and increased expenses. Some limited new activities can be taken on by the present staff and its facilities, while others require additional office space, executive and clerical expense, and travel and meeting costs necessarily determined by the nature of the project undertaken.

2. *Financial.* The handling of finances runs into several millions of dollars and includes the management of the Association itself and four subsidiary organizations:

Trustees of the California Medical Association
Physicians' Benevolence Fund, Inc.
Six Ninety Three Sutter Publications, Inc.
California Medical Education and Research Foundation

Annual financial reports of all of these organizations appear on another page of this issue in the form of balance sheets and operating statements for fiscal year ending 30 June 1966. These figures are taken directly from the audited reports of John F. Forbes & Company, Certified Public Accountants.

The California Medical Association annual report for the fiscal year ending 30 June 1966 shows a net gain of $63,731, which is the net amount of income over expenses, plus other credits and charges; as compared with a net gain of $41,103 for the preceding fiscal year. This represents a gain of $22,628, or 55.1 per cent over the preceding fiscal year surplus.

The report shows a total income from members' dues of $1,479,506, an increase of $36,553, or 2.5 per cent over dues income of $1,442,953 for the preceding fiscal year. These figures are after allocation of $3 per member to CALIFORNIA MEDICINE subscriptions.

The combined administrative and expenses other than administrative totaled $1,522,580, which was $46,919 or 3.2 per cent over the $1,475,661 recorded for the preceding year.

Expenses for scientific, educational and communication activities totaled $697,278, which is $17,483 or 2.6 per cent over the $679,795 for the preceding fiscal year.

While the total income for the current fiscal year ending 30 June 1966, was up $38,266 over the preceding fiscal year, there was an increase in the excess of expense over income for CALIFORNIA MEDICINE in the amount of $19,979. Thus, the net income for the fiscal year was at $18,587 over the preceding fiscal year.

CALIFORNIA MEDICINE reflects an excess of expenses over income for the fiscal year ending 30 June 1966 in the amount of $31,192 as against an excess of expenses over income for the previous fiscal year of $11,513. The increased deficit was due to a low point in advertising income coupled with the fact that the fiscal year reflected only a very small portion of the gain realized by the consolidation of advertising in the five western medical journals, covering 11 western states and Hawaii. The first half of the current fiscal year, which ends on 30 June 1967, indicates a substantial increase in advertising income and in all likelihood we will realize the projected budgeted income of $200,000. We anticipate a substantial reduction in the deficit for the next fiscal year ending 30 June 1967 due to increased revenue and curtailment in expenses brought about by the consolidation of advertising in the western medical journals. The journal continued its program of improved quality during 1966 and remains as one of the pre-eminent state medical journals in the country. And where the decline in advertising income for the fiscal year ending 30 June 1966 resulting from cultailment of many pharmaceutical producers of the journal advertising, a trend which has held up for several years, the prospects for 1967 are more promising.

The financial report of Trustees of the California Medical Association, which is a holding company for accumulative net assets of the Association, shows an excess of income over expenses in the amount of $26,457 as compared with a net gain of $32,944 for the preceding fiscal year. This was a reduction of $6,487 brought about primarily due to increased taxes, janitorial services, and other maintenance costs.

3. *Membership*. On 1 September 1966, the date on which an official membership count is taken for purposes of representation in the House of Delegates, the Association showed 21,817 active members, a gain of 459 members over the total recorded a year earlier. Membership gains were reported by 21 component societies while seven showed a membership loss and six, no change.

The active membership of all component societies as of 1 September 1966 and the same date a year earlier is shown in the following table:

Active Membership in the CMA by Component Societies

Societies	*Sept. 1, 1966*	*Sept. 1, 1965*
Alameda-Contra Costa	1,796	1,752
Butte-Glenn	116	106
Forty First	137	225
Fresno	388	371
Humboldt-Del Norte	95	93
Imperial	47	48
Inyo-Mono	17	15
Kern	263	258
Kings	41	37
Lassen-Plumas-Modoc-Sierra	23	24
Los Angeles	8,755	8,695
Marin	237	242
Mendocino-Lake	52	60
Merced	77	72
Monterey	200	210
Napa	88	88
Orange	1,156	1,072
Placer-Nevada	95	89
Riverside	343	324
Sacramento	625	540
San Benito	7	9
San Bernardino	479	442
San Diego	1,194	1,165
San Francisco	1,856	1,819
San Joaquin	279	270
San Luis Obispo	92	92
San Mateo	597	579
Santa Barbara	335	323
Santa Clara	1,191	1,145
Santa Cruz	134	128
Shasta-Trinity	79	77
Siskiyou	22	25
Solano	89	89
Sonoma	214	205
Stanislaus	179	183
Tehama	18	17
Tulare	127	122
Ventura	237	215
Yolo	66	61
Yuba-Sutter-Colusa	71	71
TOTAL	21,817	21,358

4. *Conclusions*. While no attempt has been made to present a detailed financial analysis of the California Medical Association, we have attempted to highlight those items which we feel would be of interest to the membership.

For the 1967-68 fiscal year the staff has been working with the Finance Committee of the Council in preparing a budget which will finance the projected activities of the Association. The proposed budget approved by the Finance Committee of the Council will be given to the Council for approval and then will go to the 1967 House of Delegates, which must give final approval.

Respectfully submitted,

LYTTON O. HETLAND, *Controller*

REPORT OF

Certified Public Accountants

California Medical Association:

We have examined the balance sheets or statement of assets at 30 June 1966, and the related statements of income and expenses or expenditures for the year then ended of California Medical Association (a nonprofit association) and the following affiliated organizations:

Trustees of the California Medical Association
Physicians' Benevolence Fund, Inc.
California Medical Education and Research Foundation
Six Ninety Three Sutter Publications, Inc.

Our examinations were made in accordance with generally accepted auditing standards, and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances.

In our opinion, the statements referred to above present fairly the financial position of each of the foregoing organizations at 30 June 1966, and the results of their operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

The accompanying supplemental schedules referred to in the aforementioned financial statements, although not considered necessary for a fair presentation of the financial condition and the results of operations of the respective organizations, have been subjected to the audit procedures applied in the examinations of the financial statements, and in our opinion are fairly presented.

John F. Forbes & Company

San Francisco, California
August 4, 1966

CALIFORNIA MEDICAL ASSOCIATION

(A Nonprofit Association)

Balance Sheet
June 30, 1966 and 1965 and Comparison

	JUNE 30		Increase
	1966	1965	(Decrease)
ASSETS			
CASH	$230,907	$ 92,562	$ 138,345
CERTIFICATE OF DEPOSIT—CROCKER-CITIZENS NATIONAL BANK		800,000	*(800,000)*
UNITED STATES TREASURY BILLS, AT COST	593,275		593,275
ACCOUNTS RECEIVABLE, NET	56,942	79,485	*(22,543)*
ACCRUED INTEREST	4,780	6,050	*(1,270)*
NOTES RECEIVABLE:			
Central California Blood Bank	74,000	84,500	*(10,500)*
Less allowance for possible loss		*(84,500)*	84,500
Trustees of the California Medical Association		30,000	*(30,000)*
Total notes receivable	74,000	30,000	44,000
PREPAID EXPENSES AND OTHER ASSETS:			
Retirement program premium (Note 1)	14,855	16,431	*(1,576)*
Insurance	3,021	1,590	1,431
Deposits	1,484	1,440	44
Other	1,665	2,532	*(867)*
Total prepaid expenses and other assets	21,025	21,993	*(968)*
FURNITURE AND FIXTURES (at nominal value) (Note 2)	1	1	
	$980,930	$1,030,091	$*(49,161)*
LIABILITIES			
ACCOUNTS PAYABLE:			
American Medical Education Foundation	$207,985	$ 203,655	$ 4,330
Other	70,634	130,775	*(60,141)*
Total accounts payable	278,619	334,430	*(55,811)*
LOAN PAYABLE—PHYSICIANS' BENEVOLENCE FUND, INC.		70,000	*(70,000)*
DEFERRED INCOME:			
Dues and subscriptions applicable to the succeeding fiscal year	667,272	654,579	12,693
Other	1,327	1,101	226
Total deferred income	668,599	655,680	12,919
EXCESS OF ASSETS OVER LIABILITIES (LIABILITIES OVER ASSETS) (Note 3)	33,712	*(30,019)*	63,731
	$980,930	$1,030,091	$*(49,161)*

See notes to financial statements

Statement of Income and Expenses
Years Ended June 30, 1966 and 1965 and Comparison

	YEAR ENDED JUNE 30		Increase	Per Cent of
	1966	1965	(Decrease)	Change
INCOME:				
Membership dues less portion allocated to CALIFORNIA MEDICINE subscriptions	$1,479,506	$1,442,953	$ 36,553	2.5
Booth rentals at annual session	40,515	39,245	1,270	3.2
Postgraduate fees	16,560	14,553	2,007	13.8
Fee for collection of American Medical Association dues	9,494	9,347	147	1.6
Interest earned	11,462	13,541	*(2,079)*	*(15.4)*
Other	1,006	638	368	57.7
	1,558,543	1,520,277	38,266	2.5
(Excess) of expenses over income of CALIFORNIA MEDICINE (Schedule 1)	*(31,192)*	*(11,513)*	*(19,679)*	*(170.9)*
	1,527,351	1,508,764	18,587	1.2
EXPENSES:				
Administration (Schedule 2)	589,778	562,640	27,138	4.8
Other than administration (Schedule 3)	932,802	913,021	19,781	2.2
	1,522,580	1,475,661	46,919	3.2
EXCESS OF INCOME OVER EXPENSES FOR THE YEAR	4,771	33,103	*(28,332)*	*(85.6)*
OTHER CREDITS AND (CHARGES):				
Reduction of allowance for possible loss on Central California Blood Bank note	84,500	8,000	76,500	956.3
Refund of pension premium on past service, net	20,460		20,460	
Payment under loan guaranty agreement (Note 4)	*(46,000)*		*(46,000)*	
	58,960	8,000	50,960	637.0
EXCESS OF ASSETS OVER LIABILITIES (LIABILITIES OVER ASSETS):				
Increase for the year	63,731	41,103	22,628	55.1
At beginning of year	*(30,019)*	*(71,122)*	41,103	57.8
At end of year	$ 33,712	$ *(30,019)*	$ 63,731	212.3

See notes to financial statements

Income and Expenses of "California Medicine" Years Ended June 30, 1966 and 1965 and Comparison

SCHEDULE 1

	YEAR ENDED JUNE 30		Increase (Decrease)	Per Cent of Change
	1966	1965		
INCOME:				
Advertising	$174,904	$186,135	*$(11,231)*	6.0
Subscriptions:				
Allocated from dues	66,489	65,299	1,190	1.8
Nonmembers	3,020	2,793	227	8.1
Reprints	6,691	7,419	*(728)*	*(9.8)*
	251,104	261,646	*(10,542)*	*(4.0)*
EXPENSES:				
Printing	180,028	176,127	3,901	2.2
Illustrations	2,843	2,852	*(9)*	*(.3)*
Advertising sales expenses	21,000	19,121	1,879	9.8
Cash discounts	3,033	3,271	*(238)*	*(7.3)*
Addressing	1,986	1,750	236	13.5
Postage and mailing	11,084	10,480	604	5.8
Reprints	4,585	5,474	*(889)*	*(16.2)*
Salaries	44,276	41,257	3,019	7.3
Editorial assistance	4,025	2,325	1,700	73.1
Rent	3,516	3,516		
Telephone and telegraph	3,475	3,729	*(254)*	*(6.8)*
Office supplies and sundry	2,445	3,257	*(812)*	*(24.9)*
	282,296	273,159	9,137	3.3
(EXCESS) OF EXPENSES OVER INCOME	*$(31,192)*	*$(11,513)*	*$(19,679)*	*(170.9)*

Administration Expenses Years Ended June 30, 1966 and 1965 and Comparison

SCHEDULE 2

	YEAR ENDED JUNE 30		Increase (Decrease)	Per Cent of Change
	1966	1965		
Rent	$ 12,672	$ 12,952	$ *(280)*	*(2.2)*
Telephone and telegraph	15,486	16,139	*(653)*	*(4.0)*
Postage	5,878	5,621	257	4.6
Equipment purchases and maintenance	7,219	5,824	1,395	24.0
Office supplies and expense	17,131	13,654	3,477	25.5
Office services	12,955	3,547	9,408	265.2
Salaries (see note below):				
Administrative	47,988	53,354	*(5,366)*	*(10.0)*
Clerical	61,080	54,214	6,866	12.7
Los Angeles office	10,652	10,509	143	1.4
Taxes	23,698	22,004	1,694	7.7
Insurance	4,224	3,682	542	14.7
Physicians' Placement Service	16,730	21,450	*(4,720)*	*(22.0)*
Professional fees and expense	42,155	43,858	*(1,703)*	*(3.9)*
Meeting expenses:				
Annual session	53,515	51,016	2,499	4.9
American Medical Association	62,627	65,693	*(3,066)*	*(4.7)*
Component society officers	10,606	8,139	2,467	30.3
Student—American Medical Association	1,122	5,414	*(4,292)*	*(79.3)*
Council	11,349	11,152	197	1.8
Medical Society executives	7,400	7,829	*(429)*	*(5.5)*
Travel expenses:				
Officers	32,107	25,575	6,532	25.5
Council	36,324	30,047	6,277	20.9
Administrative and staff	12,068	16,002	*(3,934)*	*(24.6)*
Memberships and subscriptions	5,061	6,239	*(1,178)*	*(18.9)*
Woman's Auxiliary	8,057	7,971	86	1.1
Retirement and employee benefits	69,695	59,225	10,470	17.7
Miscellaneous	1,979	1,530	449	29.3
	$589,778	$562,640	$ 27,138	4.8

NOTE: The total payroll for years ended June 30 was $528,946 for 1966 and $497,609 for 1965, of which $381,644 and $362,378, respectively, are included in other expense classifications or were charged to other organizations.

Expenses other than Administrative Years Ended June 30, 1966 and 1965 and Comparison

SCHEDULE 3

	YEAR ENDED JUNE 30 1966	YEAR ENDED JUNE 30 1965	Increase (Decrease)	Per Cent of Change
SCIENTIFIC, EDUCATIONAL, AND COMMUNICATIONS:				
Medical services	$ 24,493	$ 42,203	$*(17,710)*	*(42.0)*
Public agencies	35,009	39,103	*(4,094)*	*(10.5)*
Community health services	68,750	60,591	8,159	13.5
Professional welfare	19,654	30,015	*(10,361)*	*(34.5)*
Hospital affairs	40,211		40,211	
Bureau on communications	172,403	179,463	*(7,060)*	*(3.9)*
Special project	33,435	34,381	*(946)*	*(2.8)*
Bureau of research and planning	90,527	63,882	26,645	41.7
Library (socio-economic)	10,754	12,871	*(2,117)*	*(16.4)*
Allied health professions and services	17,511		17,511	
Scientific board:				
Commission and miscellaneous committees	51,612	62,217	*(10,605)*	*(17.0)*
Medical education	42,848	33,249	9,599	28.9
Cancer committee	6,156	10,144	*(3,988)*	*(39.3)*
Other committees	83,915	111,676	*(27,761)*	*(24.9)*
	697,278	679,795	17,483	2.6
CONTRIBUTIONS:				
Physicians' Benevolence Fund, Inc.	21,524	21,181	343	1.6
Nurse recruitment	3,000	3,000		
American Medical Education Foundation	206,000	201,000	5,000	2.5
California Commission for the Accreditation of Nursing Homes and Related Facilities		2,750	*(2,750)*	
Medical libraries	5,000	5,295	*(295)*	*(5.6)*
	235,524	233,226	2,298	1.0
	$932,802	$913,021	$ 19,781	2.2

Notes to Financial Statements June 30, 1966

NOTE 1: In addition to the Group Pension Program which became effective on January 1, 1961, the California Medical Association has completed the funding of a Past Service Pension Plan for certain full-time employees. The amount on deposit with the administrator at June 30, 1966, aggregated $108,897. These funds are to be used to purchase annuity contracts as eligible employees retire. Neither the amount on deposit nor any liability for pensions is carried on the books of the Association or shown on the accompanying balance sheet.

NOTE 2: The Association carries its furniture and fixtures at a nominal value of $1. The firm of Marshall and Stevens has conducted an appraisal and has reported that the sound value of these assets at May 13, 1966 was $104,415.

NOTE 3: The Trustees of the California Medical Association, a wholly-owned incorporated subsidiary of the California Medical Association, holds in trust a large portion of the assets utilized by the Association and related organizations. The combined net worth of the two organizations is summarized as follows:

Entity	JUNE 30 1966	JUNE 30 1965	Increase (Decrease)
California Medical Association	$ 33,712	$ *(30,019)*	$ 63,731
Trustees of the California Medical Association	1,341,170	1,314,713	26,457
	$1,374,882	$1,284,694	$ 90,188

The combined net worth at June 30, 1966 shown in the summary above does not include the following items:

California Medical Association:	
Excess of the appraised value of furniture and fixtures over the nominal carrying value (see Note 2)	$104,414
Audio-Digest Foundation, a wholly-owned subsidiary; net worth at June 30, 1966 as shown by the Foundation's audited balance sheet	246,905
	351,319
Trustees of the California Medical Association—Excess of net worth over Trustees' investment in their wholly-owned subsidiary, Pacific Magnetic Tape Co., based on that company's unaudited balance sheet at June 27, 1966	14,399
	$365,718

NOTE 4: During the year the California Commission for the Accreditation of Nursing Homes and Related Facilities was liquidated and the California Medical Association, as a guarantor of the Commission's note to the Crocker-Citizens National Bank, paid $46,000 as its share of the obligation. This obligation was anticipated and was provided for in the 1965-1966 budget.

TRUSTEES OF THE CALIFORNIA MEDICAL ASSOCIATION
(A Nonprofit Corporation)

Balance Sheet June 30, 1966 and 1965 and Comparison

ASSETS	JUNE 30 1966	 1965	Increase (Decrease)
CASH	$ 3,311	$ 29,521	$(26,210)
MARKETABLE SECURITIES, AT COST OR MATURITY VALUE (market value, $1,011,881) (Note 2):			
United States Treasury bonds	1,119,152	1,121,000	(1,848)
United States Treasury bill	19,779		19,779
Total marketable securities	1,138,931	1,121,000	17,931
NOTE AND ACCOUNT RECEIVABLE	2,400	2,633	(233)
ACCRUED INTEREST ON SECURITIES	2,307	1,536	771
INVESTMENTS IN WHOLLY-OWNED SUBSIDIARIES, AT COST:			
Pacific Magnetic Tape Equipment Co. (Note 1)	9,000	9,000	
Six Ninety Three Sutter Publications, Inc.	1,000	1,000	
Total investments in wholly-owned subsidiaries	10,000	10,000	
PROPERTY, AT COST (subject to deed of trust):			
Land	87,400	87,400	
Building and improvements	295,938	295,938	
	383,338	383,338	
Less accumulated depreciation	69,817	59,749	10,068
Property, net	313,521	323,589	(10,068)
EQUIPMENT, AT NOMINAL VALUE	1	1	
CASH SURRENDER VALUE OF LIFE INSURANCE	35,708	30,780	4,928
DEPOSIT IN ESCROW	10,000		10,000
PREPAID INSURANCE	2,525	375	2,150
REAL ESTATE TAXES APPLICABLE TO SUBSEQUENT YEAR (contra)	11,500	10,500	1,000
	$1,530,204	$1,529,935	$ 269
LIABILITIES			
ACCOUNTS PAYABLE AND ACCRUED EXPENSES:			
California Medical Association	$ 800	$ 32,507	$(31,707)
Interest	199	246	(47)
Real estate taxes (contra)	11,500	10,500	1,000
Total accounts payable and accrued expenses	12,499	43,253	(30,754)
NOTE PAYABLE WITH DEED OF TRUST AS COLLATERAL (payable in quarterly installments of $2,506 including interest to March 1, 1973)	56,295	63,728	(7,433)
TRUST FUNDS (Note 2)	106,740	97,741	8,999
DEFERRED COMPENSATION PAYABLE	13,500	10,500	3,000
EXCESS OF ASSETS OVER LIABILITIES (Note 3):			
Contributed	883,193	883,193	
Excess of income over expenses	457,977	431,520	26,457
	1,341,170	1,314,713	26,457
	$1,530,204	$1,529,935	$ 269

See notes to financial statements

Statement of Income and Expenses Years Ended June 30, 1966 and 1965 and Comparison

	YEAR ENDED JUNE 30 1966	 1965	Increase (Decrease)	of Per Cent Change
INCOME:				
Excess of rental income over property expenses (Schedule 4)	$ 12,370	$ 15,728	$ (3,358)	(21.4)
Interest on United States Treasury bonds and bills	28,324	28,772	(448)	(1.6)
Dividend—Pacific Magnetic Tape Equipment Co.	900	900		
Miscellaneous	34	53	(19)	(35.8)
	41,628	45,453	(3,825)	(8.4)
EXPENSES (other than property):				
Fees and insurance	2,339	2,158	181	8.4
	39,289	43,295	(4,006)	(9.3)
OTHER CHARGES:				
Net premium on life and retirement insurance policy	4,176	4,351	(175)	(4.0)
Provision for retirement or other benefit of an employee of an affiliated organization	3,000	3,000		
Provision for deferred compensation	3,000	3,000		
Loss on sale of securities	2,656		2,656	
	12,832	10,351	2,481	24.0
EXCESS OF INCOME OVER EXPENSES:				
Current year	26,457	32,944	(6,487)	(19.7)
Beginning of year	431,520	398,576	32,944	8.3
End of year	$457,977	$431,520	$ 26,457	6.1

See notes to financial statements

TRUSTEES OF THE CALIFORNIA MEDICAL ASSOCIATION

Rental Income and Property Expenses Years Ended June 30, 1966 and 1965 and Comparison

SCHEDULE 4

	YEAR ENDED JUNE 30		Increase (Decrease)	Per Cent of Change
	1966	1965		
INCOME FROM RENTALS:				
California Medical Association	$52,332	$52,332		
Others	18,304	19,226	$ *(922)*	*(4.8)*
	70,636	71,558	*(922)*	*(1.3)*
EXPENSES:				
Utilities	5,212	4,838	374	7.7
Janitor service and maintenance	15,636	15,012	624	4.2
Repairs—Plumbing and electric	5,481	4,097	1,384	33.8
Alterations	4,008	3,645	363	10.0
Insurance	655	854	*(199)*	*(23.3)*
Elevator inspection and service	1,574	1,376	198	14.4
Supplies	910	694	216	31.1
Other operating expenses	914	221	693	313.6
	34,390	30,737	3,653	11.9
Taxes	11,206	10,172	1,034	10.2
Interest on mortgage	2,565	2,873	*(308)*	*(10.7)*
Other	37	1,980	*(1,943)*	*(98.1)*
Total expenses before depreciation	48,198	45,762	2,436	5.3
Depreciation	10,068	10,068		
	58,266	55,830	2,436	4.4
EXCESS OF RENTAL INCOME OVER PROPERTY EXPENSES	$12,370	$15,728	$*(3,358)*	*(21.4)*

Notes to Financial Statements June 30, 1966

NOTE 1: The Trustees of the California Medical Association owns all of the outstanding stock of the Pacific Magnetic Tape Equipment Co., which was formed for the purpose of merchandising magnetic tape equipment as an adjunct to the activities of the Audio-Digest Foundation, a wholly-owned subsidiary of the California Medical Association. An unaudited balance sheet of the Pacific Magnetic Tape Equipment Co., at June 27, 1966, shows a net worth of $23,399 at that date, which is not carried on the books of the Trustees.

NOTE 2: The portion of the Trust Funds applicable to the retirement or similar benefit to Mr. and Mrs. Ben H. Read, amounting to $54,000 at June 30, 1966, has not been segregated from other assets of the corporation as provided by the bylaws, as such funds are presently invested in United States Treasury securities of indivisible denominations.

NOTE 3: The Trustees of the California Medical Association is a wholly-owned subsidiary of the California Medical Association. The Trustees hold in trust a large portion of the assets utilized by the California Medical Association. The combined net worth of the two organizations is summarized as follows:

Entity	YEAR ENDED JUNE 30		Increase
	1966	1965	
California Medical Association	$ 33,712	$ *(30,019)*	$ 63,731
Trustees of the California Medical Association	1,341,170	1,314,713	26,457
Combined net worth	$1,374,882	$1,284,694	$ 90,188

The combined net worth at June 30, 1966 shown in the summary above does not include the following items:

California Medical Association:	
Excess of the appraised value of furniture and fixtures over the nominal carrying value	$104,414
Audio-Digest Foundation, a wholly-owned subsidiary; net worth at June 30, 1966 as shown by the Foundation's audited balance sheet	246,905
	351,319
Trustees of the California Medical Association—Excess of net worth over Trustees' investment in their wholly-owned subsidiary, Pacific Magnetic Tape Co., based on that company's unaudited balance sheet at June 27, 1966	14,399
	$365,718

PHYSICIANS' BENEVOLENCE FUND, INC.

(A Nonprofit Corporation)

Statement of Assets June 30, 1966

ASSETS

CASH—CROCKER-CITIZENS NATIONAL BANK		$ 31,293
INVESTMENTS (market value $116,989):		
U.S. Treasury 2½% bonds at maturity values:		
Maturity date, December 15, 1969	$10,000	
Maturity date, December 15, 1972	34,000	
U.S. Treasury bills, at cost	77,139	121,139
NOTE RECEIVABLE WITH DEED OF TRUST AS COLLATERAL—LOS ANGELES COUNY PHYSICIANS' AID ASSOCIATION (original amount, $50,000, maturing in quarterly installments of $1,000 plus interest at 2½% per annum)		30,000
ACCRUED INTEREST RECEIVABLE		826
		$183,258

SOURCE OF ASSETS

CONTRIBUTED AT ORGANIZATION	$ 92,132
EXCESS OF INCOME OVER EXPENDITURES	91,126
	$183,258

Statement of Income and Expenditures Year Ended June 30, 1966

INCOME:		
Contributions:		
California Medical Association (see note)	$21,524	
The Woman's Auxiliary of the California Medical Association	4,007	$25,531
Interest earned:		
U.S. Treasury bonds and bills	2,661	
Loans	1,715	4,376
		29,907
EXPENDITURES:		
Payments to beneficiaries:		
Los Angeles County Physicians' Aid Association	8,700	
Others	16,400	
	25,100	
Other	571	25,671
EXCESS OF INCOME OVER EXPENDITURES:		
Current year		4,236
Beginning of year		86,890
End of year		$91,126

NOTE: The constitution of the California Medical Association, Article IV, Section 6, provides: "At least $1 out of the annual dues paid by each active member of the Association shall be allocated to the Physicians' Benevolence Fund, Inc., a corporation, and shall be used for the purposes as set forth in that corporation's Articles and Bylaws."

CALIFORNIA MEDICAL EDUCATION AND RESEARCH FOUNDATION

(A Nonprofit Corporation)

Balance Sheet June 30, 1966

ASSETS

CASH IN BANK		$12,069
ACCRUED INTEREST RECEIVABLE		127
UNITED STATES TREASURY BILLS, AT COST		37,591
		$49,787

LIABILITIES AND FUND BALANCES

ACCOUNT PAYABLE—CALIFORNIA MEDICAL ASSOCIATION		$ 8,190
FUND BALANCES:		
United States Department of Health, Education, and Welfare grant (Note 1)	$ 583	
Audio-Digest Foundation grant	27,500	
General fund	13,514	41,597
		$49,787

See notes to financial statements

Statement of Income, Expenditures and Fund Balances Year Ended June 30, 1966

	Total	Audio-Digest Foundation	U.S. Department of Health, Education, and Welfare (Note 1)	Department of Mental Hygiene State of California	General Fund
INCOME:					
Grants and restricted contributions	$46,065	$30,000	$ 5,000	$11,065	
Other	268				$ 268
	46,333	30,000	5,000	11,065	268
EXPENDITURES:					
Contributions:					
California College of Medicine	38,750	38,750			
Los Angeles County Medical Society	2,500	2,500			
Services:					
Administrative	10,516		6,112	4,404	
Other	1,635		562	1,073	
Supplies and printing	2,280		124	2,156	
Keypunch processing	175		175		
Mailing and postage	42		15	27	
Telephone and telegraph	28		6	22	
Travel and meeting	65			65	
Charge to cover payroll taxes and employee benefits	803		651	152	
Professional fees	575			575	
Other	34		3	31	
	57,403	41,250	7,648	8,505	
EXCESS OF INCOME OVER EXPENDITURES (EXPENDITURES OVER INCOME)	*(11,070)*	*(11,250)*	*(2,648)*	2,560	268
FUND BALANCE, JUNE 30, 1965	52,667	38,750	3,231	12,426	*(1,740)*
INTERFUND TRANSFERS (Note 2)				*(14,986)*	14,986
FUND BALANCE, JUNE 30, 1966	$41,597	$27,500	$ 583		$13,514

See notes to financial statements

Notes to Financial Statements June 30, 1966

NOTE 1: The Foundation has been awarded community health project grants by the United States Department of Health, Education, and Welfare, which have been applied as follows:

Period of Grant	Amount of Grant	Funds Received	Expenditures Applied	Unused Balance
6/1/62 - 5/31/63	$14,700	$11,777	$ 8,182	$ 3,595*
6/1/63 - 5/31/64	26,131	14,132	13,606	4,121*
6/2/64 - 11/30/65	43,113	15,778	16,668	3,231*
12/1/65 - 6/30/66		5,000	7,648	583
		$46,687	$46,104	$ 583

*Applied to succeeding grant.

NOTE 2: The Foundation received a contract in the amount of $44,265 from the California State Department of Mental Hygiene for the study of means of meeting the shortage of mental health personnel in California. This contract was completed during the year at a total cost of $29,279. The balance of unexpended funds aggregating $14,986 was transferred to the Foundation's general fund for use as the directors may deem necessary.

SIX NINETY THREE SUTTER PUBLICATIONS, INC.

Balance Sheet
June 30, 1966

ASSETS

CASH	$13,938
ACCOUNTS RECEIVABLE	2,675
INVENTORY, AT COST	7,933
DEPOSIT AND PREPAID EXPENSES	170
	$24,716

LIABILITIES AND STOCKHOLDER'S EQUITY

LIABILITIES:		
Accounts payable:		
California Medical Association	$ 5,042	
Other	293	
Federal income tax payable	2,453	
Total liabilities		$ 7,788
STOCKHOLDER'S EQUITY:		
Capital stock (authorized, 2,500 shares of $10.00 par value each; issued and outstanding, 100 shares) (see note)	1,000	
Retained earnings	15,928	
Total stockholder's equity		16,928
		$24,716

NOTE: The Trustees of the California Medical Association is the sole stockholder of this Corporation.

Statement of Income and Retained Earnings
Years Ended
June 30, 1966

NET SALES			$27,419
COST OF SALES			10,461
GROSS PROFIT			16,958
EXPENSES:			
Office:			
Services	$2,200		
Rent	300		
Supplies	566		
Fees	400		
Other	19	$3,485	
Shipping:			
Services	315		
Postage, freight, and express, net	1,035	1,350	
Personal property taxes		557	
State franchise and sales tax		417	5,809
INCOME BEFORE FEDERAL INCOME TAX			11,149
FEDERAL INCOME TAX			2,453
NET INCOME			8,696
RETAINED EARNINGS, JUNE 30, 1965			7,232
RETAINED EARNINGS, JUNE 30, 1966			$15,928

Iow, now, Mrs. Forsythe, we've never lost a cold patient yet.

hen she's experiencing acute discomfort from cold symptoms, it's small wonder the patient becomes distressed oout her condition.

ne will breathe easier when you prescribe Novahistine LP.

ovahistine LP is a long-acting decongestant that helps restore normal mucus secretion and ciliary activity—physiogic mechanisms which prevent infection of the respiratory tract. A dose of two tablets taken in the morning nd repeated in the evening will usually keep air passages clear for 24 hours.

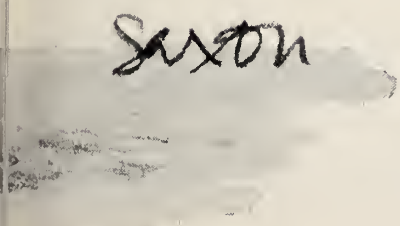
se cautiously in individuals with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. aution patients who operate machinery or motor vehicles at drowsiness may result.

ach Novahistine LP tablet contains: phenylephrine hydronloride, 25 mg., and chlorpheniramine maleate, 4 mg.

PITMAN-MOORE Division of The Dow Chemical Company, Indianapolis

NOVAHISTINE® LP

For relief of nasal congestion.



Saxon

BOOKS RECEIVED

Books received by CALIFORNIA MEDICINE *are acknowledged in this column. Selections will be made for more extensive review in the interest of readers as space permits.*

CEREBRAL VASCULAR DISEASE—Transactions of the Fifth Conference Held under the Auspices of The American Neurological Association and The American Heart Association, Princeton, New Jersey, January 5 to 7, 1966; Conference Supported by a Grant from National Institute of Neurological Diseases and Blindness—By Clark H. Millikan, Chairman; Robert G. Siekert and Jack P. Whisnant, Editors. Published for The American Neurological Association and The American Heart Association by Grune & Stratton, New York and London, 1966. 278 pages, $7.75.

CIBA FOUNDATION STUDY GROUP NO. 23—Egg Implantation, in Honour of Professor R. Courrier—Edited by G. E. W. Wolstenholme, O.B.E., M.A., F.R.C.P., F.I.Biol., and Maeve O'Connor, B.A. Little, Brown and Company, Boston, Mass., 1966. 112 pages, $3.50.

CIBA FOUNDATION STUDY GROUP NO. 24—Histones: Their Role in the Transfer of Genetic Information, in Honour of Academician W. A. Engelhardt—Edited by A. V. S. de Reuck, M.Sc., D.I.C., A.R.C.S., and Julie Knight, B.A. Little, Brown and Company, Boston, Mass., 1966. 115 pages, $3.50.

CIBA FOUNDATION SYMPOSIUM—The Thymus: Experimental and Clinical Studies, in Honour of Sir MacFarlane Burnet, O.M.—Edited by G. E. W. Wolstenholme, O.B.E., M.A., F.R.C.P., F.I.Biol., and Ruth Porter, M.R.C.P. Little, Brown and Company, Boston, Mass., 1966. 538 pages, $15.00.

CIBA FOUNDATION SYMPOSIUM—Touch, Heat and Pain—Edited by A. V. S. de Reuck, M.Sc., D.I.C., A.R.C.S., and Julia Knight, B.A. Little, Brown and Company, Boston, Mass., 1966. 389 pages, $14.00.

DERMATOLOGIC ALLERGY: IMMUNOLOGY, DIAGNOSIS, MANAGEMENT—By Leo H. Criep, M.D., F.A.C.P., Clinical Associate Professor of Medicine, School of Medicine, University of Pittsburgh; Chief of Allergy, Montefiore Hospital; Director, Central Allergy Laboratory, Veterans Administration; Consultant, Medical Staff, Presbyterian-University and Magee Hospitals. With the collaboration of 15 contributors. W. B. Saunders Company, Philadelphia and London, 1967. 605 pages, $17.00.

THE DOCTORS—By Martin L. Gross. Random House, New York, 1966. 605 pages, $6.95.

FEMININE MIND AND BODY, THE—The Psychosexual and Psychosomatic Reactions of Women—By J. Dudley Chapman, D.O., formerly Associate Professor of Obstetrics & Gynecology, College of Osteopathic Medicine and Surgery; President of the American College of Osteopathic Obstetricians and Gynecologists. Philosophical Library, New York, 1967. 325 pages, $6.75.

MECHANISMS AND THERAPY OF CARDIAC ARRHYTHMIAS—The Fourteenth Hahnemann Symposium—Editors: Leonard S. Dreifus, M.D., Clinical Associate Professor of Medicine and Director, Electrocardiographic Unit, and William Likoff, M.D., Professor of Medicine and Head, Cardiology Section, Hahnemann Medical College and Hospital, Philadelphia, Pa. Associate Editor: John H. Moyer, M.D., Professor and Chairman, Department of Medicine, Hahnemann Medical College and Hospital, Philadelphia, Pa. Grune & Stratton, Inc., New York, N.Y., 1966. 704 pages, $23.00

THE NEW MEDIA AND EDUCATION—Their Impact on Society—Edited by Peter H. Rossi and Bruce J. Biddle. Aldine Publishing Company, 320 West Adams Street, Chicago, Ill., 1967. 417 pages, $8.95.

PRACTICAL DERMATOLOGY—3rd Edition—By George M. Lewis, M.D., F.A.C.P., Late Clinical Professor Emeritus of Medicine (Dermatology), Cornell University Medical College; and Clayton E. Wheeler, Jr., M.D., Professor of Dermatoloic Medicine, University of North Carolina Medical School. W. B. Saunders Company, Philadelphia and London, 1967. 679 pages, $12.50.

RANDOM RHYMES—By Ursula Greenshaw Mandel. Exposition Press, New York, 1966. 313 pages, $6.00.

SCLERODERMA—By Marvin A. Sackner, M.D., Associate Director, Cardiopulmonary Laboratory, Mount Sinai Hospital, Miami Beach, Florida; Assistant Professor of Physiology and Medicine, University of Miami Medical School; American College of Physicians Research Fellow, 1961-1964. (Modern Medical Monographs: Irving S. Wright, M.D., Editor in Chief.) Grune & Stratton, Inc., New York and London, 1966. 174 pages, $13.75.

STACEY'S for any Medical Book in Print
Retail Stores in Palo Alto and San Francisco

NEW BOOK

PRINCIPLES OF INTERNAL MEDICINE. Edited by T. R. Harrisan, M D., et al. 5th ed. 1,920 pages. Illustrated. 1966. Blakistan. $29.50. (twa volumes); $22.50 (one volume).

Stacey's
2575 HANOVER STREET
PALO ALTO, CALIFORNIA 94304
326-9955

Please send me an appraval:__________

Name__________
Street__________
City__________ State__________

Cancer Conference in San Francisco February 25 at Lane Hall

The Presbyterian Medical Center in collaboration with the San Francisco Chapter, California Academy of General Practice, has announced a postgraduate conference in cancer to be held Saturday, February 25, at Lane Hall, 2380 Sacramento Street in San Francisco.

Designed for the practicing physician, this course will concentrate on the diagnosis and treatment of cancer. The morning session will consist of brief lectures on specific anatomic areas and panel discussion periods. Physicians attending are asked to submit questions or problems of special concern to them with their application for registration.

In the afternoon session, following three lectures on current and newer methods of diagnosis and treatment, registrants will form small seminar groups for study of individual problems or questions raised. Patients will be presented.

Registration period will be from 8:00 to 8:25. The introduction to the morning session, outlining the purpose of the seminar, will be given by Robert S. Pollack, M.D., chairman of the conference.

Subjects in the morning session include "The Skin," "Breast," "Head and Neck," "Rectal and Colon," "Gynecology," followed by the luncheon break at 12:10 p.m.

The afternoon session will lead off with "Cytologic Diagnosis of Cancer," followed by "Current Status of Cancer Chemotherapy," "Arterial Perfusion Therapy of Cancer," round table and small group discussions. The session will close with a "Tumor Conference and Case Review" with slide projector, moderated by Doctor Pollack.

Brochure with detailed program can be obtained by writing Continuing Education, Presbyterian Medical Center, Clay and Webster Streets, San Francisco, California 94115. The $30.00 registration fee includes lunch. Interns, residents, Fellows, and military personnel may register without fee.

The Needle in the Knee

A housewife drops a sewing needle on the carpet and kneels to look for it.

A flash of pain in one knee. She gasps. Leaps to her feet. Blindly she yanks the tiny steel shaft from her flesh and applies an antiseptic to the wound. In the searing moment of pain she does not notice that part of the needle is missing, broken off in the knee joint by her sudden change of position.

Days later she will visit her physician with a knee that is throbbing and swollen.

The needle-in-the-knee pattern of injury is almost uniform and thus easily recognizable, two orthopedic surgeons report in the January 2 *Journal of the American Medical Association.* Their paper was a presentation at the AMA annual convention last summer.

After hearing the victim's story of injury and subsequent pain and swelling, the physician should suspect immediately that needle fragments are still in the knee, said R. H. Ramsey, M.D., Dearborn, Mich., and Floyd G. Goodman, M.D., St. Louis, Mo.

The surgeons also recommended that immediate measures be taken to locate the fragment by x-ray and remove it from the joint.

"It is well known that small metallic foreign bodies may be tolerated in the body for an indefinite period without causing symptoms and do not necessarily require removal," they said.

"However, it is likely that needle fragments, located in articular cartilage, subchondral bone, synovium, or adjacent soft tissues or diarthrodal joints will cause continuing symptoms until surgically removed. The potential of serious infection introduced by the needle also would be a factor favoring early surgical removal."

The typical pattern of injury was revealed in 21 cases recorded at hospitals in Dearborn and St. Louis and studied by Drs. Ramsey and Goodman.

All the victims were children playing on the floor or women who were kneeling to search for the lost needle.

Although either end of the needle may penetrate the knee, the authors found it more common for the point of the needle to be in the carpet and the eye end to penetrate the joint.

In each case the pain of the puncture caused the victim to cry out and leap to a standing position.

The needle, imbedded in bone, cartilage or other tough tissue, was snapped into two or more pieces by the sudden shearing motion.

Because the fragments may be difficult to locate the x-ray search should be meticulous, the authors said. In five of the 21 cases studied, two operations were necessary to find and remove all fragments.

He leaves to make
an urgent call
But doesn't use
the phone at all

Parepectolin for quick relief of acute diarrhea
...soothes colicky pain with paregoric
...consolidates fluid stools with pectin
...adsorbs irritants with kaolin, and protects intestinal mucosa.

Whether it's a 24-hour "bug", a food problem, or simply nervousness and anxiety, Parepectolin will bring the diarrhea under control until etiology can be determined. In some cases, Parepectolin may be all the therapy necessary.

Parepectolin®

Each fluid ounce of creamy white suspension contains:
Paregoric (equivalent).........(1.0 dram) 3.7 ml.
Contains opium (¼ grain) 15 mg. per fluid ounce.
warning: may be habit forming
Pectin.....................(2½ grains) 162 mg.
Kaolin (specially purified)....(85 grains) 5.5 Gm.
(alcohol 0.69%)
Usual Adult Dose: One or two tablespoonfuls three times daily.

RORER
WILLIAM H. RORER, INC.
Fort Washington, Pa.

New
low-cost tetracycline/antifungal therapy

for broad-spectrum activity
plus specific antifungal prophylaxis
at significant patient savings

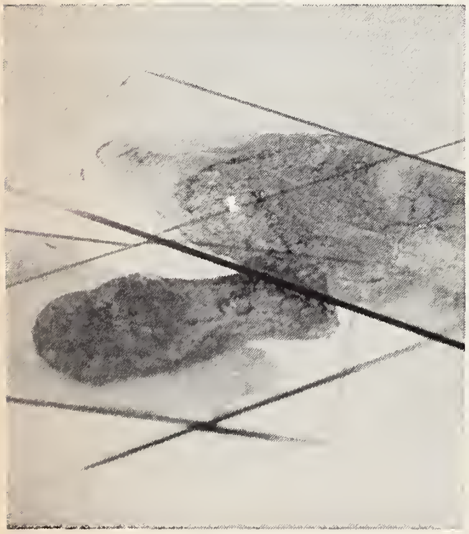
Whenever tetracycline is indicated in these candidates for Candida:

1. diabetic patients

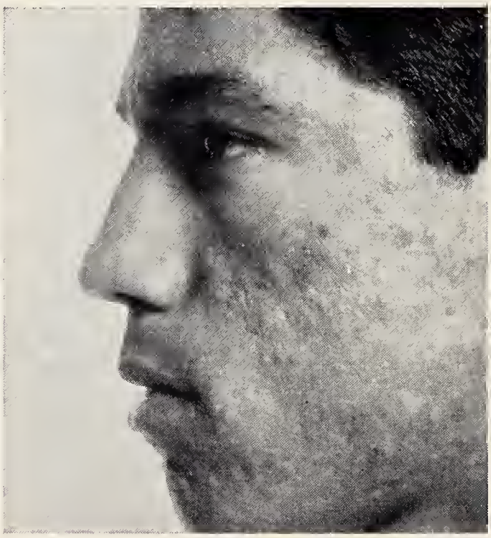
2. nonpregnant women with a history of recent or recurrent monilial vaginitis

3. elderly or debilitated patients

4. patients with a past history of moniliasis



5. patients on long-term tetracycline or corticosteroid therapy



BRISTOL THERAPEUTIC SUMMARY: For complete i
formation consult Official Package Circular. *Indication*
Infections of respiratory, gastrointestinal and genitourina
tracts and skin and soft tissues due to tetracycline-sens
tive organisms, in patients with increased susceptibili
to monilial infections. *Contraindications:* The drug is co
traindicated in patients hypersensitive to its componen
Warnings: Photodynamic reactions have been produced
tetracyclines. Natural and artificial sunlight should
avoided during therapy. Stop treatment if skin discomf
occurs. With renal impairment, systemic accumulation a
hepatotoxicity may occur. In this situation, lower do
should be used. Tooth staining and enamel hypoplasia m
be induced during tooth development (last trimester
pregnancy, neonatal period and childhood). *Precautio*
Bacterial superinfection may occur. Infants may devel
increased intracranial pressure with bulging fontanels.
gonorrheal therapy, serologic tests for syphilis should
conducted initially and monthly for 3 months. *Adve*
Reactions: Glossitis, stomatitis, nausea, diarrhea, fla
lence, proctitis, vaginitis, dermatitis, and allergic reacti
may occur. *Usual Adult Dosage:* 1 capsule *q.i.d.* Conti
therapy for 10 days in beta-hemolytic streptococcal inf
tions. Administer one hour before or 2 hours after me
Supply: Capsules, bottles of 16. Each capsule contains
racycline phosphate complex equivalent to 250 mg. te
cycline HCl activity and 250,000 units of nystatin.

BRISTOL

BRISTOL LABORATORIES
Division of Bristol-Myers Company
Syracuse, New York

Tetrex-F®

Each capsule contains tetracycline phosphate complex equivalent to tetracycline hydrochloride 250 mg. and nystatin 250,000 uni

Tetrex-F is priced lower
than most
tetracycline-antifungal products.

The nutritional statements in this series of calorie-restricted diets have been reviewed by the Council on Foods and Nutrition of the American Medical Association and found consistent with authoritative medical opinion.

FOR THE PHYSICIAN concerning the Calorie-Restricted Diets for weight reduction...1000, 1200, 1400, 1800 levels

Estimate of calorie level for weight loss

A gross estimate of total calories needed to maintain the present weight of a sedentary or moderately active adult may be achieved by multiplying present weight by 12-15 Calories per pound. Thus a man leading a sedentary or moderately active life and weighing 175 pounds may need between 2100 and 2625 Calories to maintain his weight.

A deficit of 500 Calories per day under maintenance needs allows loss of about one pound per week. In the absence of disease, satisfactory weight loss can be achieved in most men with 1400-1800 Calories per day and in women with 1000-1400 Calories per day. However, in a given patient, final selection of total calories for weight reduction will be arrived at by trial, with an increase in calories if the patient is losing too rapidly and a decrease if too slowly.

Table II presents desirable weights for men and women adapted by the Statistical Bureau of the Metropolitan Life Insurance Company.

Nutrient content of diets

Table I shows the nutrient content of the 1000, 1200, 1400 and 1800 Calorie diets, compared with the Recommended Dietary Allowances established by the Food and Nutrition Board, National Academy of Sciences—National Research Council.

The Food and Nutrition Board, in setting up the Recommended Dietary Allowances, relates the recommendation for thiamine to the total calories of the diet, providing 0.4 milligram for each 1000 Calories for adults on ordinary levels of calorie intake, but holding to not less than 0.8 milligram daily when the caloric intake is less than 2000. The 1200, 1400 and 1800 Calorie diets provide more than 1 milligram of thiamine daily and otherwise meet the Recommended Dietary Allowances.

The iron content of the diets falls below the Recommended Allowances for 18-55 year old women. If a calorie-restricted diet is prescribed to continue for prolonged periods, the patient may benefit from a multi-vitamin and mineral supplement.

Criteria for selection of foods recommended

The diets provided have been planned with certain criteria in mind. The foods are familiar to the average American. The plan at each calorie level is a simple one and can serve as a basic pattern for the rest of the patient's life. The alternates listed for the basic food groups offer the patient opportunity for personal choice and interest in menus, an item of major importance in gaining adherence to any diet plan. Each plan is realistic and possible

TABLE I.

SUMMARY—NUTRIENT CONTENT OF DIETS*

	CHO GM.	PRO. GM.	FAT GM.	CALCIUM GM.	IRON GM.	VITAMIN A I.U.	ASCORBIC ACID MG.	THIAMINE MG.	RIBOFLAVIN MG.	NIACIN EQUIVALENTS MG.
1000 CALORIE LEVEL	100	75	40	1.03	10.4**	16065**	158**	.95	2.46	26.5
1200 CALORIE LEVEL	115	85	50	1.06	11.8**	17650**	146**	1.08	2.68	30.3
1400 CALORIE LEVEL	120	90	65	1.03	11.8**	18457**	134**	1.06	2.64	30.6
1800 CALORIE LEVEL	155	110	90	1.38	13.9**	20413**	137**	1.31	3.33	37.3
RECOMMENDED DIETARY ALLOWANCE* **										
MEN, 18-35 YEARS		70		0.80	10.0	5000	70	1.20	1.70	19.0
35-55		70		0.80	10.0	5000	70	1.00	1.60	17.0
55-75		70		0.80	10.0	5000	70	0.90	1.30	15.0
WOMEN, 18-35 YEARS		58		0.80	15.0	5000	70	0.80	1.30	14.0
35-55		58		0.80	15.0	5000	70	0.80	1.20	13.0
55-75		58		0.80	10.0	5000	70	0.80	1.20	13.0

**Principal source for food composition: Nutritive Value of Foods, Revised 1964. U.S. Department of Agriculture, Washington, D.C., plus other standard sources as needed. When originally calculated, Food, The Yearbook of Agriculture, 1959 was used as the principal source. Differences between the two sets of calculations are slight. Carbohydrate, protein, and fat have been rounded to the nearest 5.*

***If liver is not used weekly, iron, vitamin A, and ascorbic acid will be less.*

****Recommended Dietary Allowances, Revised 1964. Publication 1146, National Academy of Sciences–National Research Council, Washington, D.C. All entries represent allowances for the midpoint of the specified age period.*

to follow when carrying on customary activities while reducing.

The metabolic response of individuals undergoing weight reduction has been extensively studied. Among college students several investigators have demonstrated that during caloric restriction, retention of nitrogen, calcium and phosphorus was less than when the caloric intake was adequate. A decrease in 24 hour basal caloric requirements has been noted which may account in part for weight loss occurring at a decreasing rate as the reducing period continues. The phenomenon of decreasing rate of weight loss also has been observed in middle-aged adults. Several explanations have been offered for this phenomenon—the decrease in basal energy requirements, lowered energy requirement for activity incident to the lower body weight, less expenditure of energy with dieting and weight loss, and possibly other physiologic adaptations. The effects of weight reduction continue to be a subject of research.

A food record

A history of usual eating habits is useful to give direction to a weight control program. As weight reduction proceeds, a simple daily record of all food consumed can help the patient "stay on his diet." The "uncooperative" patient may be one who actually isn't fully conscious of what he eats. The food record can guide him to understanding and make it easier for the physician to help his patient along the frequently difficult course of weight reduction.

The length of time that such a record would be continued would necessarily be at the physician's discretion. In an informal survey the question of willingness to "write down each day what you eat and how much" was posed to a group of people representative of those who may seek medical advice. The majority answered in the affirmative but some added the qualification "for a while."

Different forms could be used but perhaps the simplest is to suggest a sheet of paper marked into sections for the purpose with headings as shown on the miniature sample in the next column.

FOOD	AMOUNT
BREAKFAST	
BETWEEN MEALS	
LUNCH	
BETWEEN MEALS	
DINNER	
BEDTIME	

Bibliography

New weight standards for men and women. *Statistical Bulletin, Metropolitan Life Insurance Company 40.1 (Nov.-Dec.) 1959.*

Ohlson, M.A., Brewer, W.D., Kereluk, D., Wagoner, A., and Cederquist, D.C. Chapter 15· "Weight control through nutritionally adequate diets," **Weight Control** *Ames· Iowa State University Press. 1955.*

Olson, R.E. **Obesity as a nutritional disorder.** *Fed. Proc. Vol. 18, No. 2 (July) 1959.*

Turner, D. **Handbook of Diet Therapy.** *4th Ed. Chicago: The University of Chicago Press. 1965*

Young, C.M., Gehring, B.A., Merrill, S.H., and Kerr, M.E. **Metabolic responses of young women while reducing.** *J. Am. Diet. Assn. 36:447 (May) 1960.*

Young, C.M., Empey, E.L., Serraon, V.U., and Pierce, Z. H. **Weight reduction in obese young men.** *J. of Nutrition 61 437 (Mar.) 1957*

Young, C. M. **Weight reduction using a moderate-fat diet. I. Clinical responses and energy metabolism.** *J. Am. Diet. Assn. 28:410 (May) 1952.*

Young, C. M. **Weight reduction using a moderate-fat diet. II Bio chemical responses.** *J. Am. Diet. Assn. 28.529 (June) 1952.*

TABLE II.
DESIRABLE WEIGHTS

MEN, AGES 25 AND OVER*			
HEIGHT (IN SHOES)**	WEIGHT IN POUNDS		
	SMALL FRAME	MEDIUM FRAME	LARGE FRAME
5 ft. 2 in.	112-120	118-129	126-141
5 ft. 3 in.	115-123	121-133	129-144
5 ft. 4 in.	118-126	124-136	132-148
5 ft. 5 in.	121-129	127-139	135-152
5 ft. 6 in.	124-133	130-143	138-156
5 ft. 7 in.	128-137	134-147	142-161
5 ft. 8 in.	132-141	138-152	147-166
5 ft. 9 in.	136-145	142-156	151-170
5 ft. 10 in.	140-150	146-160	155-174
5 ft. 11 in.	144-154	150-165	159-179
6 ft.	148-158	154-170	164-184
6 ft. 1 in.	152-162	158-175	168-189
6 ft. 2 in.	156-167	162-180	173-194
6 ft. 3 in.	160-171	167-185	178-199
6 ft. 4 in.	164-175	172-190	182-204

WOMEN, AGES 25 AND OVER*			
HEIGHT (IN SHOES)**	WEIGHT IN POUNDS		
	SMALL FRAME	MEDIUM FRAME	LARGE FRAME
4 ft. 10 in.	92- 98	96-107	104-119
4 ft. 11 in.	94-101	98-110	106-122
5 ft.	96-104	101-113	109-125
5 ft. 1 in.	99-107	104-116	112-128
5 ft. 2 in.	102-110	107-119	115-131
5 ft. 3 in.	105-113	110-122	118-134
5 ft. 4 in.	108-116	113-126	121-138
5 ft. 5 in.	111-119	116-130	125-142
5 ft. 6 in.	114-123	120-135	129-146
5 ft. 7 in.	118-127	124-139	133-150
5 ft. 8 in.	122-131	128-143	137-154
5 ft. 9 in.	126-135	132-147	141-158
5 ft. 10 in.	130-140	136-151	145-163
5 ft. 11 in.	134-144	140-155	149-168
6 ft.	138-148	144-159	153-173

**Prepared by the Metropolitan Life Insurance Company, 1959.*

***1-inch heels for men and 2-inch heels for women.*

SAMPLE DIET SHEET—available for your patients

NAME: ______________________

1000 Calories—The day's total

The foods listed below have been planned to give the protein, vitamins, minerals, and other nutrients which are needed for good health.

A daily meal pattern and the sample menus opposite show how the foods can be arranged into meals. Plan to eat the indicated amounts daily, not more or less.

Milk, skim, 1 pint or 2 (8 ounce) glasses

Meat (with visible fat removed), Poultry, or Fish or use Cheese or Eggs for part of the meat, 1 serving at each meal—the total for the day should equal the protein in 7 ounces of cooked meat (a small meat patty is about 2 ounces.)

Vegetables and Fruits, 6 servings—Include dark green or deep yellow vegetables; and citrus fruit or tomatoes

Bread, enriched or whole grain, 2 slices or alternates

Butter, 2 teaspoons or small pats, or alternates

Vary your menus by using the alternates listed on the back of this sheet.

Special Recommendations

____

____

______________________ M.D.

A Daily Meal Pattern

Breakfast

Citrus Fruit, ½ cup or alternate
Egg, 1 } or Cereal, cooked
Bread, 1 slice } ½ cup or ¾ cup ready-to-eat + milk, ½ cup
Butter, 1 pat—or at another meal
Coffee or Tea, if desired
(without sugar or cream)

Two Sample Menus for Breakfast

Grapefruit, ½ small
Poached Egg, 1
Toast, 1 slice
Butter, 1 small pat
Coffee or Tea

Orange Juice, ½ cup
Oatmeal, ½ cup or
Cornflakes, ¾ cup
with milk, ½ cup
Coffee or Tea

Lunch

Lean Meat, Poultry, or Fish, small serving, 2 ozs. cooked, no added fat, or Cheese or Eggs for part or all of the meat
Vegetable, 1 serving } back page
Fruit, 1 serving } for amounts
Bread, 1 slice or alternate
Butter, 1 small pat or alternate
Milk, skim, 1 glass (8 ounce)

Two Sample Menus for Lunch

Sandwich—
Luncheon Meat, 1 slice
American Cheese, 1 slice
Bread, 1 slice
Butter, 1 small pat
Raw Carrot, 1
Apple, 1 small
Milk, skim, 1 glass

Salad—
Cottage Cheese, ¼ cup
Tomato, 1
Lettuce
Saltines, 4, 2″ square
Butter, 2 small pats
Peach, 1 medium
Milk, skim, 1 glass

Dinner

Lean Meat, Poultry, or Fish, generous serving, 4 ounces cooked, no added fat
Vegetables, 2 servings
1 raw and 1 cooked
Fruit, 1 serving
Milk, skim, 1 glass (8 ounce)

Two Sample Menus for Dinner

Broiled Beef Patty, 1 large
Asparagus, ½ cup
Tossed Green Salad, Vinegar Dressing, ½ to 1 cup
Pineapple, ½ cup
Milk, skim, 1 glass

Roast Chicken, leg and thigh
Green String Beans, ½ cup
Head Lettuce, wedge, with lemon
Strawberries, 1 cup
Milk, skim, 1 glass

Snacks, if desired

Bouillon or consomme, tomato juice, raw vegetables, coffee or tea, or food may be saved from meals.

Note: *Extra food will mean extra calories. Sugars or sirups, added butter, other fats or oils, or larger servings of the foods listed would be "extras." Coffee or tea, without sugar or cream, may be used as desired.*

Alternates in Dairy Foods

1 glass of whole milk (170 Calories) may replace...

1 glass skim milk (85 Calories) by omitting
2 small pats of butter (70 Calories)

Alternates in Fats

The Calories of each average about 35.

- 1 teaspoon butter
- 1 teaspoon oil or cooking fat
- 1 teaspoon mayonnaise
- 1 tablespoon sour cream
- 1 tablespoon light cream
- 1 slice crisp bacon

Alternates in Meat, Poultry, or Fish

These are about equal in protein...the Calories vary slightly, but average about 70 for each.

- 1 ounce cooked meat (with visible fat removed), poultry, or fish...1 ounce is 1 thin slice, about 2″ by 1½″ by ½″, or 1 small, thin lamb chop
- 1 egg
- 1 ounce slice cheese, American or Swiss
- 2 tablespoons creamed cottage cheese

Alternates in Bread

The Calories of each average about 60.

bread	1 slice	vegetables, cooked:	
cereal:		beans, lima,	
cooked	½ cup	fresh	½ cup
flake or puff		corn, sweet	½ cup
ready-to-eat	¾ cup	parsnips	½ cup
crackers:		potato	1 small
graham	2, 2½″ sq.	(2″ diameter, or	
oysterettes	½ cup	diced, ½ cup)	
saltines	4, 2″ sq.		
soda	3, 2½″ sq.		

Choices in Vegetables and Fruits

Vegetables—

Use as much raw vegetables as desired. If cooked limit amount to ½ cup...(under 25 calories)

asparagus
beans, string
broccoli
cabbage
carrots
cauliflower
celery
cucumber
greens
kale
lettuce
squash, summer
spinach
tomatoes
turnips

Fruits (fresh or unsweetened)—

The Calories of each average about 45.

½ cup applesauce
blackberries
blueberries
cherries
*grapefruit juice
grapes
*orange juice
pineapple
pineapple juice
raspberries

apple	1 small
apricots, fresh	2 medium
dried	4 halves
banana	½ medium
*cantaloupe	½ medium
grapefruit	½ small
honeydew	2″ wedge
nectarine	1 large
*orange	1 small
peach	1 medium
pear	1 small
plum	1 medium
*strawberries	1 cup
tangerine	1 medium
watermelon	½ slice, 1″

*These are high vitamin C, and can be used interchangeably. Use one daily.

Aids to Varying Meals...

Bake, broil or roast meats. For flavor, add herbs to meats and vegetables—use ¼ teaspoon of dry herbs for each six servings of vegetables, meat or fish.

Try basil, rosemary, garlic salt, dry mustard or curry with beef or lamb; rosemary or savory with veal, chicken or fish.

Season tomatoes with curry, basil, or oregano; turnips with rosemary; cabbage with savory; zucchini or carrots with thyme or nutmeg; cauliflower with dill; spinach with marjoram or nutmeg.

Vinegar Dressing: ¼ cup vinegar, 2 tablespoons water, 1 clove garlic, sliced, ⅛ teaspoon salt, ⅛ teaspoon paprika, ¼ teaspoon non-caloric liquid sweetener, ½ teaspoon dill or tarragon, or a combination of rosemary or thyme. Store in refrigerator. Makes ⅓ cup.

For another tasty Salad Dressing: combine ½ cup tomato juice, ½ teaspoon dry mustard, ½ teaspoon salt, ⅛ to ¼ teaspoon non-caloric liquid sweetener, ½ teaspoon lemon juice, and a dash of chili powder and hot pepper sauce. Store in refrigerator. Makes ½ cup.

Scramble eggs in a double boiler with 1 tablespoon of milk for one egg. Add salt and pepper and for variety a dash of curry, or chopped chives, green onion or parsley.

Flavor milk with instant coffee, vanilla or spices.

Michigan, Texas Fight Venereal Disease

Two state medical societies are starting statewide educational programs to control venereal disease, The AMA News has reported.

In Michigan, the Congress of Parents and Teachers (PTA) and Michigan State Medical Society are cooperating in a program to educate students, teachers and parents about the threat of VD and ways to control it.

The project is believed to be the first of its kind in the nation, says the National PTA headquarters in Chicago.

The Texas Medical Association is preparing a similar campaign which may become the most extensive such program in the nation.

A Conference on Venereal Disease will be sponsored January 28 by the TMA, Texas Department of Health, Texas Junior Chamber of Commerce, and Texas PTA.

Attending will be key officers and committee chairmen of 11 state organizations interested in VD control programs.

Statistics furnished by the City of Houston Health Department indicate the need for the program. The department estimates 140,000 Texans are infected by VD each year; that for the four-month period July to October 1966, Texas was one of only two states showing a significant increase in reported infectious syphilis and that Texas reported the second highest number of infectious syphilis cases and the largest number of venereally infected teenagers.

During this same four-month period, Michigan had the second largest increase in reported cases of infectious syphilis.

About 1,000 Michigan PTA groups have been contacted by the Michigan State Medical Society, and urged to contact local medical societies for assistance in presenting VD programs at their meetings and to help conduct VD assembly meetings in schools.

The state's 55 local medical societies have been urged by the MSMS to cooperate in the programs. Physician-speakers have been provided for youth groups, and a seminar on VD was held at the state society's annual meeting.

The National Congress of Parents and Teachers, at its national meeting in May, approved a resolution calling for a "sound and adequate program of venereal disease education in every school system in the United States, beginning at least by the eighth grade . . ."

The American Medical Association, at its annual meeting in June, gave renewed emphasis to its nationwide VD control program.

See Page 76 of the advertising section to make your hotel reservation for the 1967 CMA Annual Session.

DIARRHEA
MUCOUS COLITIS
DIVERTICULITIS
SPASTIC URETERITIS
BLADDER SPASM

are relieved with.....

Trocinate®

BRAND THIPHENAMIL HCl

Minimum dosage 400 mg., q. 4 h. until relief is constant, adjust maintenance dosage.

A therapeutic blood level cannot be obtained with small dosage. Trocinate is metabolized and eliminated in the urine as harmless degradation products—a safety factor. Sixteen years of clinical usage with the absence of untoward effects establishes the safety of Trocinate. The autonomic nervous system is not involved in its prompt action.

NOW AVAILABLE IN 2 STRENGTHS,
100 mg. and 400 mg.
PINK SUGAR-COATED TABLETS

Literature and samples available.

WM. P. POYTHRESS & CO., INC.
RICHMOND, VIRGINIA 23217

Manufacturers of ethical pharmaceuticals since 1856

Especially...

When the bronchitis→emphysema syndrome is complicated by hypertension or heart disease—

ORAL ELIXOPHYLLIN®

Each 15 cc. contains theophylline 80 mg.; alcohol 3 cc.

. . . has consistently provided rapid, sustained bronchodilation[1] without the hazards of epinephrine-like drugs.

Used in recommended t.i.d. dosage "[Elixophyllin] . . . rapidly produces an effective blood level comparable to that produced when the drug is administered intravenously."[2] Such serum levels have been correlated[3] with a proven significant increase in pulmonary function. By maintaining effective bronchodilation with Elixophyllin, the incidence of acute attacks may be reduced, increasing damage to bronchial tissue arrested, functioning tissue preserved, and progression toward intractable emphysema retarded.

Unusually well tolerated—Unlike many available compounds, the theophylline in Elixophyllin is not precipitated in the presence of gastric acid, and thereby avoids gastric irritation. Moreover, by remaining in solution, Elixophyllin is carried without delay into the blood stream for *predictable bronchodilative action.*

Simple, precise oral medication—minimizes danger of self-overmedication:

Adults, 45 cc. (3 tablespoonfuls) before breakfast, at 3 P.M., and before retiring. After two days, 30 cc. (2 tablespoonfuls) t.i.d. Does not contain epinephrine-like drugs; alcoholic content is non-grain, hypo-allergenic. May be contraindicated in peptic ulcer and gout.

1. Corroborated by 40 published studies.
2. Miller, W. F.: Postgrad. Med. *39*:230-239 (Mar.) 1966.
3. Jackson, R. H., et al.: Dis. Chest *45*:75-85 (Jan.) 1964.

Sherman Laboratories

DETROIT, MICHIGAN

help
keep her
this way

Specifically designed
for estrogen
replacement in the
menopause
...and later years

Premarin®

BRAND OF

CONJUGATED ESTROGENS (equine)

PREMARIN (conjugated estrogens – equine) is a naturally occurring, orally active, water-soluble estrogen presented as the complete equine estrogen complex. CAUTION: *In the female:* To avoid continuous stimulation of breast and uterus, cyclic therapy is recommended (3 week regimen with 1 week rest period – Withdrawal bleeding may occur during this 1 week rest period). *In the male:* Continuous therapy over prolonged periods of time may produce gynecomastia, loss of libido, and testicular atrophy. AVAILABLE in 4 potencies for individualized treatment: *Tablets* – 2.5 mg. (purple), 1.25 mg. (yellow), 0.625 mg. (red), 0.3 mg. (green); bottles of 100 and 1,000. 2.5 mg. tablets also available in 20's.

6703

AYERST LABORATORIES New York, N.Y. 10017 • Montreal, Canada

Officers OF THE CALIFORNIA MEDICAL ASSOCIATION

General Officers

President: JAMES C. MACLAGGAN, 525 Hawthorn Street, San Diego 92101. 714 233-6519
President-Elect: JOHN G. MORRISON, 333 Estudillo, San Leandro 94577. 415 483-1332
Past President: RALPH C. TEALL, 2626 L Street, Sacramento 95816. 916 448-1851
Speaker of the House of Delegates: WILLIAM F. QUINN, 1401 S. Hope Street, Los Angeles 90015. 213 749-0181
Vice-Speaker of the House of Delegates: JOSEPH W. TELFORD, 3255 Fourth Avenue, San Diego 92103. 714 295-5151
Chairman of the Council: CARL E. ANDERSON, 1150 Montgomery Drive, Santa Rosa 95405. 707 546-1922
Secretary: HELEN B. WEYRAUCH, 450 Sutter Street, San Francisco 94108. 415 392-6909
Editor: DWIGHT L. WILBUR, 655 Sutter Street, San Francisco 94102. 415 673-4080

Executive Director: HOWARD HASSARD, 693 Sutter Street, San Francisco 94102. 415 776-9400
General Counsel: PEART, HASSARD, SMITH and BONNINGTON, 44 Montgomery Street, San Francisco 94104. 415 982-8585

* * *

District Councilors

FIRST DISTRICT—Roger C. Isenhour, (1967), 2881 Fourth Avenue, San Diego 92103. San Diego County. 714 295-2137
SECOND DISTRICT—Imperial, Inyo, Mono, Orange, Riverside and San Bernardino Counties—
  Office No. 1—Henry V. Eastman (1967), 661 W. First Street, Tustin 92680. 714 544-4560
  Office No. 2—Frank C. Melone (1968), 527 N. Palm Avenue, Ontario 91761. 714 986-1104
THIRD DISTRICT—Los Angeles County—
  Office No. 1—Malcolm C. Todd (1968), 2865 Atlantic Avenue, Long Beach 90806. 213 426-1355
  Office No. 2—Elmer F. Gooel (1969), 465 N. Roxbury Drive, Beverly Hills 90210. 213 271-5745
  Office No. 3—Richard L. Taw (1967), 3741 Stocker Street, Los Angeles 90008. 213 293-7157
  Office No. 4—Lewis T. Bullock (1968), 3875 Wilshire Boulevard, Los Angeles 90005. 213 382-2209
  Office No. 5—Joseph P. O'Connor (1969), 595 E. Colorado Boulevard, Pasadena 91101. 213 792-2154
  Office No. 6—Franklin F. Ham (1967), 14556 Hamlin Street, Van Nuys 91401. 213 786-5711
  Office No. 7—Wilbur G. Rogers (1968), 1122 N. Brand Boulevard, Glendale 91202. 213 241-3125
  Office No. 8—Jean F. Crum (1969), 8355 E. 3rd Street, Downey 90241. 213 869-1091
  Office No. 9—Joseph F. Boyle (1967), 1136 W. Sixth Street, Los Angeles 90017. 213 482-9891
FOURTH DISTRICT—Joseph F. Maguire (1967), 2755 Loma Vista Road, Ventura 93003. San Luis Obispo, Santa Barbara and Ventura Counties. 805 648-2567
FIFTH DISTRICT—Ralph W. Burnett (1969), 515 Truxton Avenue, Bakersfield 93301. Calaveras, Fresno, Kern, Kings, Madera, Mariposa, Merced, San Joaquin, Stanislaus, Tulare and Tuolumne Counties. 805 327-4411
SIXTH DISTRICT—Monterey, San Benito, San Mateo, Santa Clara and Santa Cruz Counties.
  Office No. 1—Richard S. Wilbur (1967), 300 Homer Avenue, Palo Alto 94301. 415 321-4121
  Office No. 2—Albert G. Miller (1968), 77 San Mateo Drive, San Mateo 94401. 415 343-9074
SEVENTH DISTRICT—San Francisco County—
  Office No. 1—Malcolm S. M. Watts (1969), 909 Hyde Street, San Francisco 94109. 415 776-3838
  Office No. 2—Roberta F. Fenlon (1967), 490 Post Street, San Francisco 94102. 415 986-1391
EIGHTH DISTRICT—Alameda and Contra Costa Counties—
  Office No. 1—Harold Kay (1969), 411 Thirtieth Street, Oakland 94609. 415 832-4822
  Office No. 2—William F. Kaiser (1967), 3000 Colby Street, Berkeley 94705. 415 843-6515
NINTH DISTRICT—Carl E. Anderson (1967), 1150 Montgomery Drive, Santa Rosa 95405. Del Norte, Humboldt, Lake, Marin, Mendocino, Napa, Solano and Sonoma Counties. 707 546-1922
TENTH DISTRICT—James H. Yant (1968), 2600 Capitol Avenue, Sacramento 95816. Alpine, Amador, Butte, Colusa, El Dorado, Glenn, Lassen, Modoc, Nevada, Placer, Plumas, Sacramento, Shasta, Sierra, Siskiyou, Sutter, Tehama, Trinity, Yolo and Yuba Counties. 916 446-5278
ELEVENTH DISTRICT—Forest J. Grunigen (1967), 3919 Beverly Boulevard, Los Angeles 90004. Forty First Medical Society. 213 663-1161

SCIENTIFIC BOARD REPRESENTATIVE—William P. Longmire, Jr. (1967), UCLA Department of Surgery, Los Angeles 90024. 213 272-8911

* * *

COMMISSION ON *Medical Services*

Ralph Burnett, Bakersfield
Elmer F. Gooel, Beverly Hills (Secty.)
Arthur F. Howard, Fresno (Chmn.)
Charles B. Hudson, Oakland
Homer C. Pheasant, Los Angeles
Milo A. Youel, San Diego.

Committee on Fees

H. Dean Hoskins, Oakland	1967
Wallace A. Gerrie, Jr., Newport Beach	1967
Robert L. Watson, Jr., Los Angeles	1967
Roger C. Isenhour, San Diego	1968
Alfred J. Murrieta, Jr., Los Angeles	1968
Maurice S. Salomon, San Francisco	1968
Homer C. Pheasant, Los Angeles (Chmn.)	1969
Glenn A. Pope, Sacramento	1969
Leo Bell, San Mateo	1969
Joseph F. Boyle, Los Angeles	1969

CONSULTANT: William H. Thompson, San Mateo.

Committee on Federal Medical Care Programs

Charles B. Canby, Van Nuys	1967
Merlin A. Hendrickson, Rialto	1967
Ralph L. Hoffman, San Diego	1967
John S. McKee, Jr., San Diego	1968
Arthur Howard, Fresno (Chmn.)	1968

Committee on Veterans Affairs

Charles B. Hudson, Oakland (Chmn.)
Ben D. A. Miano, San Bernardino
Robert L. Watson, Los Angeles
CONSULTANT: Lloyd E. Gould, Napa.

Committee on Mediation

Eli A. Layon, Brawley	1967
Milo A. Youel, San Diego (Chmn.)	1967
Richard L. Taw, Los Angeles	1968
Albert G. Clark, San Francisco	1969
George Babbin, Sacramento	1969

Committee on Insurance and Prepayment

Oscar W. Hills, San Mateo	1967
Dave F. Dozier, Sacramento	1967
Ralph W. Burnett, Bakersfield (Chmn.)	1968
Stanley J. Murasky, Reseda	1968
Ivan Neubauer, San Jose	1969
Thayer A. Smith, Norwalk	1969

* * *

COMMISSION ON *Public Agencies*

Lewis T. Bullock, Los Angeles
Josepb P. Cosentino, Sacramento (Secty.)
William F. Kaiser, Berkeley
Howard W. Lindsey, Jr., San Mateo
Albert Miller, San Mateo (Chmn.)
Stanley A. Skillicorn, San Jose
Howell E. Wiggins, San Diego
Joseph P. O'Connor, Pasadena
CONSULTANT: Malcolm C. Todd, Long Beach.

Committee on Public Health

Robert L. Garrett, Vallejo	1967
Karl L. Schaupp, Jr., San Francisco	1967
Allen F. Sterling, San Bernardino	1967
Austin W. Matthis, Sacramento	1967
Albert G. Miller, San Mateo	1968
Stanley A. Skillicorn, San Jose (Chmn.)	1968
Alfred F. Kandlbinder, Monterey	1969
Robert Watson, Jr., Los Angeles	1969
Samuel S. Woolington, Long Beach	1969

Committee on Mental Health

Emmanuel Silver, Palo Alto	1967
Ira H. Degenhardt, San Rafael	1967
Rudolph B. Toller, Stockton	1967
John A. Stroud, III, Sacramento	1967
Alfred Auerback, San Francisco	1968
David H. Payne, Los Angeles	1968
Jack B. Lomas, Beverly Hills	1968
Joseph J. Furlong, San Francisco	1969
Stuart C. Knox, Los Angeles (Chmn.)	1969

Committee on Welfare Medical Care Programs

Clyde L. Boice, Palo Alto	1967
Edgar Rosen, Oakland	1967
Thomas N. Elmendorf, Willows	1967
Vincent P. Carroll, Laguna Beach	1967
Orrin S. Cook, Jr., Sacramento	1968
Robert F. Schell, San Rafael	1968
William F. Quinn, Los Angeles	1969
James Dalton, Santa Barbara	1969
Arthur F. Howard, Fresno	1969
Howard W. Lindsey, Jr., San Mateo (Chmn.)	1969

SUBCOMMITTEE ON ADOPTIONS: Walter Beckh, San Francisco; Fred M. Kay, Fullerton; Robert J. McNeil, Los Angeles, (Chmn.); Donald Minkler, Berkeley; Dale W. Ritter, Chico; Ben J. Siebenthal, Sacramento.

Committee on Occupational Health and Rehabilitation

John H. Leimbach, Jr., San Francisco	1967
Herbert A. Holden, San Leandro	1967
Howell E. Wiggins, San Diego (Chmn.)	1967
Edward Zaik, Los Angeles	1968
Packard Thurber, Jr., Los Angeles	1968
Thomas Gucker, III, Los Angeles	1968
Herbert A. Lints, Oakland	1969
Joseph N. Vizzard, Los Gatos	1969
Patrick J. Maloney, Merced	1969

* * *

COMMISSION ON *Community Health Services*

Wayne P. Chesbro, Berkeley
Charles G. Hutter, Jr., Hollywood
Gerald G. Jampolsky, Tiburon (Secty.)
Elmer R. Jennings, Long Beach
Harold Kay, Oakland (Chmn.)
George A. Martin, Redding
Pierre Salmon, San Mateo
Leopold J. Snyder, Fresno
Beverly G. Stewart, Bakersfield

Committee on Rural Health

Mitry P. Ajalat, Calexico	1967
Richard W. Johnson, Arbuckle	1967
Leopold J. Snyder, Fresno (Chmn.)	1968
Erwin P. Brauner, Visalia	1968
Carrol W. Goss, Lamont	1968
Franklin L. Murphy, Oroville	1969
Thomas Rossitto, Auburn	1969
M. Marlin Clark, Delano	1969

Committee on School Health

B. Otis Cobb, Martinez	1967
Gerald G. Jampolsky, Tiburon (Chmn.)	1967
Byron H. Demorest, Sacramento	1967
William A. Leovy, La Mesa	1968
Tom W. Robinson, Newport Beach	1968
Glenn Austin, Los Altos	1968
Rutb A. Frary, Watsonville	1968
Leslie M. Holve, Los Angeles	1969
Henry B. Bruyn, Berkeley	1969

Committee on Health Care of the Aged

Franklin F. Ham, Van Nuys	1967
Pierre Salmon, San Mateo (Chmn.)	1967
Charles E. Schoff, Jr., Sacramento	1968
Raymond D. Manchester, Visalia	1968
Leo A. Wagner, Novato	1969
Lloyd E. Gould, Napa	1969
Walter Batcbelder, San Mateo	1969

Committee on Disaster Medical Care

Charles C. Henderson, San Mateo	196
Donald E. Barker, Van Nuys	196
Wayne P. Cbesbro, Berkeley (Chmn.)	196
Michael J. Hitchko, Eureka	196
J. T. McNally, Stockton	196
John Heald, San Francisco	196

CONSULTANTS: Charles N. Earl, Arcata; Frank F Schade, Los Angeles; Robert S. Jobnson, Chico Stephen Bruny, Fresno.

Committee on Automotive and Traffic Safety

George C. Snively, Sacramento	196
Beverly G. Stewart, Bakersfield (Chmn.)	196
Arnold R. Friesen, Pasadena	196
Joseph F. Maguire, Ventura	196
Thomas W. Lyons, La Mesa	196
Robert L. Tour, San Francisco	196

CONSULTANTS: Julian A. Waller, Berkeley; Julia M. Sether, Los Angeles.

Committee on Medical Aspects Of Sports and Physical Fitness

John F. Blinn, Stockton	19
Robert K. Kerlan, Los Angeles	19
George S. Buehler, Whittier	19
Philip H. McFarland, Fullerton	19
Tom W. Robinson, Newport Beach	19
Charles G. Hutter, Jr., Hollywood (Chmn.)	19
Arnold A. Nutting, San Rafael	19

Committee on Environmental Health

George A. Martin, Redding	19
Robert L. Hippen, San Diego	19
Grace M. Talbott, San Francisco	19
Francis M. Pottenger, Jr., Monrovia	19
Reginald H. Smart, Los Angeles	19
Douglas Campbell, San Francisco	19
John R. Lee, Mill Valley	19

Committee on Blood Banks

Robert W. Purvis, Modesto	19
Osman W. Hull, Monterey	19
James H. Yant, Sacramento	19
Elmer Jennings, Long Beach (Chmn.)	19

CONSULTANT: Melvin L. Sommer, Beverly H

COMMISSION ON *Communications*

Frederick Ackerman, Pleasant Hill
L. Morgan Boyers, Sacramento (Secty.)
John A. E. Bullis, Los Angeles
Roberta F. Fenlon, San Francisco
Elmer F. Gooel, Beverly Hills
Walter W. Hopps, Los Angeles
George Schwartz, Beverly Hills
Malcolm C. Todd, Long Beach (Chmn.)
Richard S. Wilbur, Palo Alto
EX-OFFICIO: James C. MacLaggan, San Diego; John G. Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Scientific Board

David A. Rytand, Palo Alto	1967
William W. Belford, San Diego	1967
Jerome W. Bettman, San Francisco	1967
John B. Dillon, Los Angeles	1967
Justin J. Stein, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
R. Kirklin Ashley, San Francisco	1967
Victor Richards, San Francisco	1967
David Rubin, Los Angeles	1967
Keith P. Russell, Los Angeles	1967
Edward J. Zaik, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Seymour J. Brockman, Los Angeles	1968
S. Austin Jones, Los Angeles	1968
Hobart M. Kelly, Riverside	1968
Saul J. Robinson, San Francisco	1968
Eugene Ziskind, Los Angeles	1968
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. deC. M. Saunders, San Francisco	1968
Herbert A. Holden, San Leandro	1968
David A. Wood, San Francisco	1968
Arthur A. Clinco, Los Angeles	1968
R. Theodore Bergman, Los Angeles	1968
Leland B. Blanchard, San Jose	1969
L. S. Goerke, Los Angeles	1969
Bert L. Halter, San Francisco	1969
Ellis Jones, Pasadena	1969
Carl McCandless, San Francisco	1969
J. Blair Pace, Oceanside	1969
Donald W. Petit, Los Angeles	1969
Glenn Pope, Sacramento	1969
Robert J. Prentiss, San Diego	1969
Edward B. Shaw, San Francisco	1969
Ralph E. Worden, Los Angeles	1969

COUNCIL REPRESENTATIVES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.
EX-OFFICIO: Dwight L. Wilbur, San Francisco.

Executive Committee

William W. Belford, San Diego	1967
Victor Richards, San Francisco	1967
Justin J. Stein, Los Angeles	1967
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. Dillon, Los Angeles	1969
Donald Petit, Los Angeles	1969

COUNCIL DESIGNEES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.

Committee on Nominations

Justin J. Stein, Los Angeles (Chmn.)	1967
Edward B. Shaw, San Francisco	1969
Robert J. Prentiss, San Diego	1969

Committee on Scientific Assemblies

John B. Dillon, Los Angeles (Chmn.)	1967
Victor Richards, San Francisco	1967
David A. Rytand, Palo Alto	1967
John B. deC. M. Saunders, San Francisco	1968
Ellis Jones, Pasadena	1969
Donald Petit, Los Angeles	1969

CONSULTANT: Claron Oakley, Los Angeles

Committee on Continuing Medical Education

John B. Dillon, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
Saul J. Robinson, San Francisco	1968
Donald W. Petit, Los Angeles (Chmn.)	1969
Bert L. Halter, San Francisco	1969
J. Blair Pace, Oceanside	1969

Committee on CALIFORNIA MEDICINE

David Rubin, Los Angeles	1967
R. Kirklin Ashley, San Francisco	1967

Committee on Scientific Information

Justin J. Stein, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Arthur A. Clinco, Los Angeles	1968
Edward B. Shaw, San Francisco (Chmn.)	1969
L. S. Goerke, Los Angeles	1969

Committee on Maternal and Child Care

Russell W. Mapes, Beverly Hills	1967
Keith P. Russell, Los Angeles (Chmn.)	1967
Forrest H. Howard, Garden Grove	1967
Charles M. Blumenfeld, Sacramento	1968
Herbert A. Holden, San Leandro	1968
Hobart M. Kelley, Riverside	1968
Leland Blanchard, San Jose	1969

Committee on Cancer

John W. Cline, San Francisco	1967
Robert W. Jamplis, Palo Alto	1967
David A. Wood, San Francisco (Chmn.)	1967
John G. Walsh, Sacramento	1968
Lewis W. Guiss, Los Angeles	1969
Robert Prentiss, San Diego	1969

SUBCOMMITTEE ON CANCER EDUCATION: Sol R. Baker, Beverly Hills; Robert W. Jamplis, Palo Alto (Chmn.); Maurice Galante, San Francisco; Justin J. Stein, Los Angeles.
SUBCOMMITTEE ON TUMOR TISSUE REGISTRY: John W. Cline, San Francisco (Chmn.); Lewis W. Guiss, Los Angeles; James E. Kahler, Los Angeles; Carl M. McCandless, San Francisco; Weldon K. Bullock, Los Angeles.
SUBCOMMITTEE ON CONSULTATIVE TUMOR BOARDS: Bert L. Halter, San Francisco; Carl L. Moore, Bakersfield; Victor Richards, San Francisco; John G. Walsh, Sacramento (Chmn.).

Committee on Adverse Drug Reactions

Edward R. Bloomquist, Glendale	1967
Ralph O. Wallerstein, San Francisco	1967
John F. Murray, Fresno (Chmn.)	1968
Russell J. Tat, San Francisco	1969
William W. Thayer, Stockton	1969

CONSULTANT: John W. Brown, Berkeley; Ralph W. Weilerstein, Berkeley.
SUBCOMMITTEE ON DANGEROUS DRUGS: Edward Bloomquist, Glendale; Norman A. Gale, San Diego; Edward T. Jewett, Santa Rosa; William F. Quinn, Los Angeles (Chmn.); Helen B. Weyrauch, San Francisco.
CONSULTANT: Ralph W. Weilerstein, Berkeley.

Committee on Cardiovascular Diseases

Harold I. Griffeath, San Francisco (Chmn.); Lewis T. Bullock, Los Angeles; David J. New, Modesto; George C. Griffith, Los Angeles.
CONSULTANT: John E. Connolly, Los Angeles.

* * *

BUREAU OF *Research and Planning*

J. Brandon Bassett, Oakland
John Sheehy, Riverside
Samuel Sherman, San Francisco (Chmn.)
H. Russell Fisher, Glendale
G. Roger Ellison, Pomona
EX-OFFICIO: Malcolm S. M. Watts, San Francisco; Ralph C. Teall, Sacramento.
CONSULTANT: Gerald W. Shaw, Santa Monica.

* * *

COMMISSION ON *Professional Welfare*

Bert L. Halter, San Francisco
George K. Herzog, Jr., San Francisco (Chmn.)
Francis E. West, San Diego
James H. Yant, Sacramento (Secty.)

Committee on Physicians Group Insurance

Robert J. Wescott, El Centro	1967
Robert X. DeMangus, Glendale	1967
John B. Long, Sacramento	1967
George K. Herzog, Jr., San Francisco (Chmn.)	1968
John Hogan, San Diego	1969
Homer C. Pheasant, Los Angeles	1969

Liaison Committee to Medical Schools

Samuel R. Irvine, Beverly Hills	1967
Francis E. West, San Diego	1967
Frank J. Novak, Menlo Park	1968
Stanley Cowell, Los Angeles	1968
Bert L. Halter, San Francisco (Chmn.)	1969
Alonzo Neufeld, Los Angeles	1969
Herbert Jenkins, Sacramento	1969

EX-OFFICIO: James C. MacLaggan, San Diego; John G. Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Medical Review and Advisory Committee

Eugene M. Webb, San Francisco	1967
James H. Yant, Sacramento (Chmn.)	1967
John B. Dillon, Los Angeles	1968
Carl Goetsch, Berkeley	1968
Donald Donisthorpe, Los Angeles	1969

CONSULTANT: Don Harper Mills, Long Beach.

Liaison Committee to State Bar of California

David Rubsamen, Berkeley	1967
John V. Fiore, Westchester	1967
Francis E. West, San Diego (Chmn.)	1968
Carl Goetsch, Berkeley	1968
Robert J. McNeil, Los Angeles	1969
Henry A. Brown, San Mateo	1969

* * *

COMMISSION ON *Allied Health Professions and Services*

Roger W. Barnes, Los Angeles (Secty.)
Forest J. Grunigen, Los Angeles (Chmn.)
Frank C. Melone, Ontario
Thomas W. Ledwich, Napa
C. Gerald Scarborough, San Jose
Richard H. Schug, Long Beach

Committee on Paramedical Personnel

Frank C. Melone, Ontario (Chmn.)	1967
Gregory Bard, San Francisco	1968
Elmer R. Jennings, Long Beach	1968
Gerson R. Biskind, San Francisco	1969
Joseph Schaefer, Santa Rosa	1969
Leonard M. Asher, Los Angeles	1969
Richard H. Schug, Long Beach (Chmn.)	1969

Committee on Other Professions

George S. Buehler, Whittier	1967
Burt L. Davis, Palo Alto	1967
C. Gerald Scarborough, San Jose (Chmn.)	1968
William D. Evans, Los Angeles	1968
J. Gordon Epperson, Oakland	1969
Lewis T. Bullock, Los Angeles	1969

Liaison Committee to California Medical Assistants Association

Leon O. Burke, Sacramento	1967
John C. Brennan, Pico Rivera	1967
H. Russell Irwin, San Diego	1967
Peary Benn Berger, Inglewood	1968
Judith D. Smith, Vallejo	1969
Sanford E. Feldman, San Francisco	1969
Richard H. Schug, Long Beach (Chmn.)	1969

CONSULTANTS: Leland W. Bonar, Redding; Henry Bodner, Van Nuys.

Committee on Medicine and Religion

Albert E. Long, Daly City	1967
Roger W. Barnes, Los Angeles (Chmn.)	1967
Max Nelson, National City	1968
Frank J. Glassy, Sacramento	1969
Richard J. Lescoe, Torrance	1969

Committee on Voluntary Health Agencies

H. Mason Hohl, Beverly Hills	1967
Thomas W. Ledwich, Napa (Chmn.)	1968
Harvey May Cordua, Jr., San Diego	1969
J. Brandon Bassett, Oakland	1969

* * *

Judicial COMMISSION

Donald A. Charnock, Los Angeles	1967
Albert E. Long, Daly City	1967
Donald Cass, Hollywood	1967
Floyd O. Due, Oakland	1968
Sidney J. Shipman, San Francisco	1968
Francis E. West, San Diego (Chmn.)	1968
Douglas G. Campbell, San Francisco	1969
W. Philip Corr, Riverside (Secty.)	1969
L. E. Wilson, Anaheim	1969

* * *

COMMISSION ON *Hospital Affairs*

William L. Argo, Fresno (Secty.)
James C. Doyle, Beverly Hills
Bert L. Halter, San Francisco (Chmn.)
John C. Lungren, Long Beach
Glenn Pope, Sacramento
John T. Saidy, San Mateo
Joseph W. Telford, San Diego
Frederic N. Tyroler, Los Angeles

Medical Staff Survey Committee

Robert M. Adams, San Leandro
W. L. Argo, Fresno
Daniel W. Black, Castro Valley
Rush M. Blodget, Jr., Redding
Bert L. Halter, San Francisco (Chmn.)
J. Ralph Hughes, San Diego
Wm. Kenneth Jennings, Santa Barbara
Donald O. Lagerlof, Los Angeles
John C. Lungren, Long Beach
William A. Newsom, San Francisco
James L. Rhee, Oakland
John T. Saidy, San Mateo
Wilfred J. Snodgrass, Santa Monica
Alexander Hatoff, Oakland
Shepard F. Palitz, Bakersfield
William L. Sumption, Santa Monica
A. Jerome Hurter, Pasadena
Ernest R. Barnett, Alhambra
Richard L. Taw, Los Angeles
Richard H. McDonald, Garden Grove
Robert M. Zweig, Riverside
L. Morgan Boyers, Sacramento
James O. Farley, Sacramento
Joseph W. Telford, San Diego
Paul Deeb, Redlands
Maxwell Goldstein, Fontana
Ray Seavers, Jr., San Bernardino
William Soldmann, La Mesa
Lloyd Milburn, San Francisco
Frank Solomon, San Francisco
Sanford Feldman, San Francisco
John Bazzano, San Francisco
Kenneth Allen, San Mateo
Martin Kohn, San Bruno
William Stephenson, Santa Barbara
Merlin Hendrickson, Rialto
Andrew D. Lucine, Campbell
Fred Haven Draper, San Jose
Wayne O. Buck, San Jose
William A. Reeves, Mt. View
A. A. Thurlow, Jr., Santa Rosa
John A. Carollo, Sabastopol
William Rector, Sacramento
Gerald W. Shaw, Santa Monica
Charles L. Lear, Norwalk
John R. Heckman, Marysville
Robert Buckley, Santa Monica
Woodrow W. Burgess, Sacramento
Ira H. Degenhardt, San Rafael
Arthur E. Gladman, Oakland
Norman Graff, San Mateo
Frank V. Hoffman, Huntington Beach
Robert E. James, Belmont
Clement O. Juul, Oakland
Seymour Kolko, San Mateo
Melvin M. Lipsett, Berkeley
Jack B. Lomas, Los Angeles
Edward A. Macklin, San Francisco
D. Stuart MacRobbie, Berkeley
Felix H. Ocko, Berkeley
William R. Sheehy, Berkeley
John A. Stroud, Sacramento
R. J. Sutherland, Oakland
Rudolph B. Toller, Stockton
Albert T. Voris, Belmont
G. Cameron Cray, Palo Alto

(Continued on Page 62)

Officers of Scientific Sessions

Members who contemplate presentation of scien tific papers should promptly address the secretar of the proper section at the address shown belou

Allergy

Leo N. Meleyco............Chairman
2040 Forest Avenue, San Jose 95128

Ernest J. Saslow............Secretary
2011-18th Street, Bakersfield 93301

Samuel Ross ....Assistant Secretary
2946 Fresno Street, Fresno 93706

Anesthesiology

Thomas W. McIntosh............Chairman
686 East Union St., Pasadena 91101

Donald B. Dose............Secretary
5625 Linfield Ave., San Diego 92120

Donald A. Walker............Assistant Secretary
35 Spring Lane, Tiburon 94920

Dermatology

Robert G. Walton ....Chairman
1700 McHenry Village Way, Modesto 95350

Samuel Ayres, III............Secretary
405 N. Bedford Rd., Beverly Hills 90210

George T. Wilson............Assistant Secretary
Welch and Pasteur Rd., Stanford Medical Plaza
Palo Alto 94304

General Practice

J Blair Pace............Chairman
408 Cassidy St., Oceanside 92054

Alfred F. Kandlbinder............Secretary
302 Via Paraiso St., Monterey 93940

Allen F. Sterling............Assistant Secretary
1285 D St., San Bernardino 92045

General Surgery

Seibert Pearson ....Chairman
2865 Atlantic Ave., Long Beach 90807

Albert G. Clark............Secretary
450 Sutter St., San Francisco 94108

Robert T. Hood, Jr............Assistant Secretary
2010 Wilshire Blvd., Los Angeles 90057

Industrial Medicine and Surgery

Leon R. Rudnick............Chairman
15921 E. 14th St., San Leandro 94578

Raymond O. Oja ....Secretary
525 Golden Gate Ave., San Francisco 94102

James G. Forgerson............Assistant Secretary
Metropolitan Life Insurance Company
600 Stockton St., San Francisco 94108

Internal Medicine

Robert L. Paver............Chairman
384 Post St., San Francisco 94108

John L. Benton, Jr............Secretary
1216 Wilshire Blvd., Los Angeles 90017

Edgar Rosen ....Assistant Secretary
1340 K St., Sacramento 95814

Obstetrics and Gynecology

Lester T. Hibbard............Chairma
142 S. Windsor Blvd., Los Angeles 90004

Warren E. Jones............Secretar
1380-45th Street, Sacramento 95819

James W. Ravenscroft............Assistant Secretar
2330 First Ave., San Diego 92101

Ophthalmology

Victor G. Fellows............Chairma
595 Buckingham Way, San Francisco 94117

Wendell C. Irvine ....Secretar
3875 Wilshire Blvd., Los Angeles 90005

Robert E. Hopkins............Assistant Secretar
4255 Pacific Ave., Stockton 95207

Orthopedics

David C. Monsen............Chairma
2300 S. Hope St., Los Angeles 90007

Arthur R. Hartwig............Secretar
490 Post St., San Francisco 94102

Vernon L. Smythe............Assistant Secretar
3875 Wilshire Blvd., Los Angeles 90005

Otolaryngology

G. Howard Gottschalk ....Chairma
8618 S. Sepulveda Blvd., Los Angeles 90045

Charles P. Lebo ....Secretary
490 Post St., San Francisco 94102

Irving L. White............Assistant Secretary
911 San Antonio Dr., Long Beach 90807

Pathology

Frank R. Dutra............Chairman
20103 Lake Chabot Rd., Castro Valley 94546

William H. Kern............Secretary
679 S. Westlake Ave., Los Angeles 90057

Donald L. Bittner............Assistant Secretary
Bay Area Laboratories, 2245 Post St.,
San Francisco 94115

Pediatrics

Richard L. Anderson............Chairman
636 Harris St., Eureka 95501

Sherrod C. Swift............Secretary
14619 Whittier Blvd., Whittier 90605

Robert J. Harvey............Assistant Secretary
909 Hyde St., San Francisco 94109

Physical Medicine

Rene Cailliet ....Chairman
4900 Sunset Blvd., Los Angeles 90027

Anton A. Tratar............Secretary
2360 Clay St., San Francisco 94115

Elizabeth Austin ....Assistant Secretary
Los Angeles County Hospital
1200 N. State St., Los Angeles 90033

Preventive Medicine

Henrik L. Blum............Chairman
P. O. Box 871, Martinez 94554

Maxwell Rosenblatt ....Secretary
Los Angeles County Health Department,
220 N. Broadway, Los Angeles 90012

Russell B. Watson............Assistant Secretary
Orange County Health Department, Santa Ana
92701

Psychiatry and Neurology

Werner M. Mendel............Chairman
1934 Hospital Place, Los Angeles 90033

Ames Fischer ....Secretary
Langley Porter Inst., 401 Parnassus Ave.,
San Francisco 94122

Howard S. Barrows............Assistant Secretary
2025 Zonal Ave., Los Angeles 90033

Radiology

Victor G. Mikity............Chairman
1200 State St., Los Angeles 90033

Vernon R. Gee............Secretary
2020 Court St., Redding 96001

Richard P. Storrs............Assistant Secretary
2131 West 3rd St., Los Angeles 90057

Urology

Carl K. Pearlman............Chairman
1518 N. Sycamore, Santa Ana 92701

James L. Goebel............Secretary
51 Loma Linda, Ross 94957

Robert O. Pearman............Assistant Secretary
5353 Balboa Blvd., Encino 91316

Delegates and Alternates

TO THE AMERICAN MEDICAL ASSOCIATION

DELEGATES		ALTERNATES
Francis E. West, San Diego	(1966-1967)	Vincent P. Carroll,* Laguna Beach
Samuel R. Sherman, San Francisco	(1966-1967)	Albert G. Miller, San Mateo
Henry Gibbons, III, San Francisco	(1966-1967)	A. B. Sirbu, San Francisco
John M. Rumsey, San Diego	(1966-1967)	Carl E. Anderson, Santa Rosa
Eugene F. Hoffman, Los Angeles	(1966-1967)	Joseph F. Boyle, Los Angeles
Warren L. Bostick, Los Angeles	(1966-1967)	Walter H. Brignoli, St. Helena
J. B. Price, Santa Ana	(1966-1967)	Donald C. Dodds, Oakland
Ralph C. Teall, Sacramento	(1966-1967)	Robb Smith, Orange Cove
James C. Doyle, Beverly Hills	(1966-1967)	Herman H. Stone, Riverside
Wilbur G. Rogers, Glendale	(1966-1967)	Carl M. Hadley, San Bernardino
Charles B. Hudson, Oakland	(1966-1967)	Robert L. Watson, Jr., Los Angeles
John G. Morrison, San Leandro	(1967-1968)	Frederick Ackerman, Pleasant Hills
William F. Quinn, Los Angeles	(1967-1968)	Dudley M. Cobb, Los Angeles
Robert C. Combs, San Francisco	(1967-1968)	Emmett Rixford, San Francisco
Homer C. Pheasant, Los Angeles	(1967-1968)	James C. MacLaggan, San Diego
Alfred J. Murrieta, Los Angeles	(1967-1968)	Harold Kay, Oakland
Burt L. Davis, Palo Alto	(1967-1968)	Leon P. Fox, San Jose
Arlo A. Morrison, Ventura	(1967-1968)	Henry Brown, San Mateo
James E. Feldmayer, Exeter	(1967-1968)	Charles Grayson, Sacramento
O. W. Wheeler, Riverside	(1967-1968)	Wilbur Bailey, Los Angeles
Malcolm C. Todd, Long Beach	(1967-1968)	Neil C. Hamel, Encino
J. E. Vaughan, Bakersfield	(1967-1968)	Joseph P. O'Connor, Pasadena

(Delegates and Alternates to the AMA are elected for terms of two calendar years. Each office is scheduled for election at the Annual Session of the second year shown above.) * *Council appointee.*

Other Organizations and Medical Schools

Board of Medical Examiners of the State of California

Secretary............Shelby M. Hicks, M.D.
1021 O Street, Room A202, Sacramento 95814
San Francisco............30 Van Ness Ave., 94102
Room 2100
Los Angeles............107 South Broadway 90012
Sacramento............1021 O Street, Room A547 95814

The Public Health League of California

Executive Secretary............Ben H. Read
955 South Western Ave., Los Angeles 90006
Assistant Executive Secretary............Al Paul Putnam
693 Sutter Street, San Francisco 94102
San Francisco............693 Sutter Street 94102
415 474-1061
Los Angeles............955 South Western Ave. 90006
213 731-6397
Sacramento............1127-11th Street, 95814
916 444-7496

Department of Public Health of the State of California

Director............Lester Breslow, M.D.
2151 Berkeley Way, Berkeley 94704
Berkeley............2151 Berkeley Way 94704
415 843-7900
Sacramento............1927-13th Street 95814
916 445-2264
Los Angeles............417 South Hill Street 90013
213 620-2900

Medical Schools in California

University of California School of Medicine, Medical Center, San Francisco 94122. Dean: Stuart C. Cullen, M.D.

Stanford University School of Medicine, 300 Pasteur Drive, Palo Alto 94304. Dean: Robert J. Glaser, M.D.

University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 90033. Dean: Roger O. Egeberg, M.D.

Loma Linda University School of Medicine, Loma Linda 92354. Dean: David Burdg Hinshaw, M.D.

University of California at Los Angeles, School of Medicine, Hilgard Avenue, Los Angeles 90024. Dean: Sherman M. Mellinkoff, M.D.

University of California, California College of Medicine, 1721 Griffin Avenue, Los Angeles 90031. Dean: Warren L. Bostick, M.D.

University of California, San Diego, School of Medicine, La Jolla 92038. Acting Dean: Robert D. Tschirgi, M.D.

University of California, Davis, School of Medicine, Davis 95616. Dean: Charles John Tupper, M.D.

California Medical Education and Research Foundation

Directors: Ralph C. Teall, M.D., President; Carl E. Anderson, M.D., Vice-President; Howard Hassard, Secretary; James C. MacLaggan, M.D.; Harold Kay, M.D.; William F. Quinn, M.D.; E. B. Shaw, M.D.

Roster — CALIFORNIA COMPONENT MEDICAL SOCIETIES

Society secretaries are requested to notify California Medicine promptly when changes are indicated in their roster information

ALAMEDA-CONTRA COSTA Medical Assn., 6230 Claremont Ave., Oakland 94618. Meets quarterly.
Frederick W. Ackerman........President
2100 Monument Blvd., Pleasant Hill 93542
Herbert A. Holden........Secretary
1436 Clarke St., San Leandro 94577

BUTTE-GLENN Medical Society, P. O. Box 1008, Chico 95927. Meets Fourth Thursday.
Walter S. Mazen........President
1617 Esplanade, Chico 95926
John B. Copeland........Secretary
1423 Magnolia, Chico 95926

FORTY FIRST Medical Society, Suite 821, 1515 N. Vermont Ave., Los Angeles 90027
Paul D. Yates........President
844 Hermosa Ave., Hermosa Beach 90254
Jordan M. Phillips........Secretary
11239 S. Lakewood Blvd., Downey 90241

FRESNO County Medical Society, 1118 Divisadero St., Fresno 93721. Meets Second Tuesday, 6:30 p.m., California Hotel.
Theodore Steinberg........President
155 N. Thesta St., Fresno 93701
Jack Schiff........Secretary
167 N. Thesta St., Fresno 93701

HUMBOLDT-DEL NORTE County Medical Society. Meets Second Thursday.
Wells C. Carey........President
2822 E St., Eureka 95501
Max Todd........Secretary
616-14th St., Arcata 95521

IMPERIAL County Medical Society, P. O. Box 518, Imperial 92251. Meets Second Tuesday, 8 p.m., Pioneer Memorial Hospital, Brawley.
O. G. Fais........President
1745 S. Imperial Ave., El Centro 92243
Elmer C. Werner........Secretary
1745 S. Imperial Ave., El Centro 92243

INYO-MONO County Medical Society. Meets Fourth Tuesday except December, January, February.
Milton R. Jones, Jr........President
Box 38, Lone Pine 93545
Robert W Denton........Secretary
611 W. Line St., Bishop 93514

KERN County Medical Society, 2603 G Street, Bakersfield 93301. Meets Third Tuesday, 7:30 p.m., Society Office, 2603 G Street, except June, July, August.
Keith Spaulding........President
2521 "G" St., Bakersfield 93301
James Friend........Secretary
1921-18th St., Bakersfield 93301

KINGS County Medical Society, P. O. Box 1003, Hanford 93230. Meets Second Thursday, 7:00 p.m. Place to be announced.
James Dean........President
912 N. Douty St., Hanford 93230
Willard S. Bridwell........Secretary
1507 N. 11th Ave., Hanford 93230

LASSEN-PLUMAS-MODOC-SIERRA County Medical Society Meets on call.
Kenneth G. Korver........President
50 N. Roop St., Susanville 96130
James McCullough........Secretary
50 N. McDow St., Susanville 96130

LOS ANGELES County Medical Assn., 1925 Wilshire Blvd., Los Angeles 90057.
Joseph F. Boyle........President
1136 W. Sixth St., Los Angeles 90017
Homer C. Pheasant........Secretary
2300 S. Hope St., Suite 300,
Los Angeles 90007

MARIN Medical Society, 1601 Second St., Suite 106, San Rafael 94901. Meets First Thursday, 7:00 p.m.
Robert Mills........President
711 D St., San Rafael 94901
William A. Galeno........Secretary
712 D St., San Rafael 94901

MENDOCINO-LAKE County Medical Society.
Frank H. Dailey........President
719 S. Dora, Ukiah 95482
Patrick R. Allanson........Secretary
765 S. Dora St., Ukiah 95482

MERCED-MARIPOSA Medical Society, P. O. Box 586, Merced 95340. Meets Fourth Thursday; meeting place changes.
Patrick J. Maloney........President
P. O. Box 586, Merced 95340
Robert J. Schiffler........Secretary
P. O. Box 586, Merced 95340

MONTEREY County Medical Society, P. O. Box 308, Salinas 93903. Meets First Thursday.
Joseph E. Turner........President
1010 Cass St., Monterey 93940
Thomas S. Elliott........Secretary
101 Maple St., Greenfield 93927

NAPA County Medical Society, P. O. Box 2158, Napa 94559. Meets Second Wednesday.
Richard W. Neil........President
1375 Main St., Helena 94574
Victor J. Bouillon........Secretary
3020 Beard Rd., Napa 94558

ORANGE County Medical Association, 300 S. Flower, Orange 92666. Meets First Tuesday, 7:00 p.m.
Wallace A. Gerrie........President
1901 Westcliff Dr., Newport Beach 92660
David I. Nielsen........Secretary
400 Westminster Blvd., Newport Beach 92660

PLACER-NEVADA County Medical Society. Meets Second Wednesday.
F. Harold Johnson........President
305 Oak Ridge Dr., Roseville 95678
Arthur R. Weaver........Secretary
1212 High St., Suite 106, Auburn 95603

RIVERSIDE County Medical Association, 4175 Brockton Ave., Riverside 92501. Meets Second Monday, 7:30 p.m., Spanish Arts Gallery, Mission Inn.
Roger W. Ridley........President
6969 Brockton Ave., Riverside 92506
Richard W. Trotter........Secretary
6876 Magnolia Ave., Riverside 92506

SACRAMENTO County Medical Society, 5380 Elvas Ave., Sacramento 95819. Meets Third Tuesday, 8:30 p.m. Location of meetings varies.
James W. Martin........President
2811 L St., Sacramento 95816
James O. Farley........Secretary
3428 Watt Ave., Sacramento 95821

SAN BENITO County Medical Society. Meets once a month, except July and August. Time and place to be announced.
Robert D. Quinn........President
930 Sunset Dr., Hollister 95023
Roderick L. Yip........Secretary
891 Sunset Dr., Hollister 95023

SAN BERNARDINO County Medical Society, 1875 N. D St., San Bernardino 92405. Meets First Tuesday, 7:30 p.m., Harold's Club, Fontana.
Harold Hill........President
219 Cajon Ave., Redlands 92373
Nicholas P. Krikes........Secretary
25060 Base Line, San Bernardino 92410

SAN DIEGO County Medical Society, 3427-4th Ave., San Diego 92103. Meets Second Tuesday. Location of meetings varies.
John Wanless........President
3427 Fourth Ave., San Diego 92103
W. C. Herrick........Secretary
233 A St., San Diego 92101

SAN FRANCISCO Medical Society, 250 Masonic Ave., San Francisco 94118.
Albert G. Clark........President
450 Sutter St., San Francisco 94108
Harry W. Sawyer........Secretary
909 Hyde St., San Francisco 94109

SAN JOAQUIN County Medical Society, 445 W. Acacia St., P. O. Box 230, Stockton 95201. Meets First Thursday, 8:15 p.m.
Thomas Huff........President
512 W. Harding Way, Stockton 95204
Dora A. Lee........Secretary
2420 N. California St., Stockton 95204

SAN LUIS OBISPO County Medical Society, P.O. Box 319, San Luis Obispo 93402. Meets Third Saturday, 7:00 p.m., Madonna Inn, San Luis Obispo 93402.
Robert Buck........President
P. O. Box 319, San Luis Obispo 93402
Billy W. Mounts........Secretary
P. O. Box 319, San Luis Obispo 93402

SAN MATEO County Medical Society, 122 El Camino Real, San Mateo 94401. Meets Third Tuesday.
Stanford B. Rossiter........President
Sequoia Hospital, Redwood City 94602
Eldon E. Ellis........Secretary
2900 Whipple Ave., Redwood City 94602

SANTA BARBARA County Medical Society, 300 W. Pueblo St., Santa Barbara 93105. Meets Second Monday of the month, University Club, 1332 Santa Barbara St., Santa Barbara.
T. K. Hill........President
1421 State St., Santa Barbara 93105
Casimir Domz........Secretary
317 W. Pueblo St., Santa Barbara 93105

SANTA CLARA County Medical Society, 700 Empey Way, San Jose 95128. Meets First Monday.
Gerald Besson........President
700 Empey Way, San Jose 95128
Alexander MacKenzie........Secretary
700 Empey Way, San Jose 95128

SANTA CRUZ County Medical Society. Meets every Second Month, Second Tuesday. Time, place to be announced.
W. W. Hoskins........President
30 Brennan St., Watsonville 95076
William Blade........Secretary
1510 Seabright Ave., Santa Cruz 95062

SHASTA-TRINITY County Medical Society. Meets Second Monday of the month. Place to be announced.
Joseph T. DeWitte........President
1129 Hartnell Ave., Redding 96001
Eugene A. Nutley........Secretary
2625 Edith Ave., Redding 96001

SISKIYOU County Medical Society. Meets Sunday on call.
Robert L. Anderson........President
504 Hillcrest Dr., Yreka 96097
John P. Geyman........Secretary
705 S. A St., Mt. Shasta 96067

SOLANO County Medical Society, 631 Tennessee St., Vallejo 94599.
Tuesday. 8:00 p.m., at different meeting places.
Robert L. Garrett........President
1512 Napa St., Vallejo 94590
Keith E. Vincent........Secretary
2121 Redwood St., Vallejo 94594

SONOMA County Medical Society, 1625 Franklin Ave., Santa Rosa 95404. Meets Second Thursday.
John S. Dunn........President
576 B St., Santa Rosa 95401
Elizabeth Buckley........Secretary
1140 Sonoma Ave., Santa Rosa 95405

STANISLAUS County Medical Society, 1125-13th St., Modesto 95354. Meets Third Tuesday of the month, 7 p.m., Hotel Covell, Modesto.
Howard Kuiil........President
1700 McHenry Ave., Modesto 95350
Jeanne I. Miller........Secretary
709-18th St., Modesto 95354

TEHAMA County Medical Society. Meets at call of President.
W. Scott Davis........President
P. O. Box 118, Los Molinos 96055
M. Blair McGee........Secretary
217 Corona Ave., Corning 96021

TULARE County Medical Society, 1640 West Mineral King. P. O. Box 16, Visalia 93278.
Irving M. Schor........President
P. O. Box 1151, Visalia 93277
George Tiss........Secretary
P. O. Box 7, Visalia 93277

VENTURA County Medical Society, 3291 Loma Vista Rd., Room 40, Ventura 93003. Meets Second Tuesday each month, 7:30 p.m., Los Posas Country Club, Camarillo.
Joseph F. Gstettenbauer........President
3003 Loma Vista Rd., Ventura 93003
Franklin K. Helbling........Secretary
3291 Loma Vista Rd., Room 40, Ventura 93003

YOLO County Medical Society. Meets First Wednesday.
Benjamin R. Robinson........President
650 Third St., Woodland 95695
Corbin Young........Secretary
101 Main St., Winters 95694

YUBA-SUTTER-COLUSA County Medical Society.
John R. Heckman........President
714-14th St., Marysville 95901
Norman K. Noordhoff........Secretary
320 G St., Marysville 95901

Officers — CALIFORNIA PHYSICIANS' SERVICE — 1966-67
BLUE SHIELD

Officers:

Chairman of the Board	Richard S. Wilbur
Vice-Chairman of the Board	Milo A. Youel
Secretary	Wilbur G. Rogers
Treasurer	Bert L. Halter

BOARD OF *Trustees:*

Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Roberta F. Fenlon*	San Francisco
Donald R. Fitch	Glendale
Mr. Thomas Hadfield	San Francisco
Bert L. Halter	San Francisco
Donald C. Harrington	Stockton
M. M. Haskell	Long Beach
Carl Goetsch	Berkeley
Mr. Philip S. Magruder	Flintridge
Gregory C. Murray	Los Angeles
Wilbur G. Rogers*	Glendale
Mr. Paul Schrade	Los Angeles
William H. Thompson	San Mateo
Richard S. Wilbur*	Palo Alto
Milo A. Youel	San Diego

*Appointed by CMA Council

BOARD COMMITTEES:

Executive Committee

Richard S. Wilbur, Chairman	Palo Alto
Carl Goetsch	Berkeley
Bert L. Halter	San Francisco
Mr. Philip S. Magruder	Flintridge
Wilbur G. Rogers	Glendale
William H. Thompson	San Mateo
Milo A. Youel	San Diego

Finance Committee

Bert L. Halter, Chairman	San Francisco
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Donald R. Fitch	Glendale
Mr. Philip S. Magruder	Flintridge

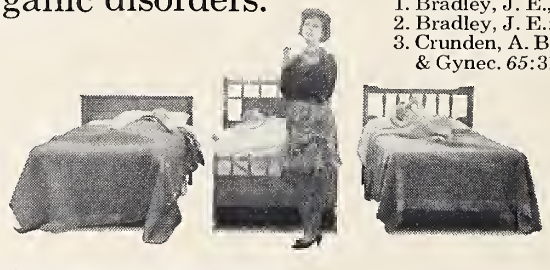
Medical Policy Committee

Wilbur G. Rogers, Chairman	Glendale
Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Roberta F. Fenlon	San Francisco
Carl Goetsch	Berkeley
Mr. Thomas Hadfield	San Francisco
M. M. Haskell	Long Beach
Donald C. Harrington	Stockton
Gregory C. Murray	Los Angeles

Togetherness....

...can be rough when epidemics of nausea and vomiting strike a family. Emetrol offers prompt, safe relief. It is free from toxicity[1] or side effects[2,3] and will not mask symptoms of serious organic disorders.

1. Bradley, J. E., *et al.*: J. Pediat. *38*:41 (Jan.) 1951.
2. Bradley, J. E.: Mod. Med. *20*:71 (Oct. 15) 1952.
3. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. *65*:311 (Feb.) 1953.



RORER

WILLIAM H. RORER, INC.
Fort Washington, Pa.

Emetrol®
phosphorated carbohydrate solution
emesis control

BOOKS RECEIVED

Books received by CALIFORNIA MEDICINE *are acknowledged in this column. Selections will be made for more extensive review in the interest of readers as space permits.*

THE ART OF PREDICTIVE MEDICINE—The Early Detection of Deteriorative Trends (Proceedings of a Symposium)—Compiled and edited by Webster L. Marxer, M.D., Director, Diagnostic Survey Department, Beverly Hills Medical Clinic, Beverly Hills, California, and George R. Cowgill, Ph.D., Sc.D. (Hon.), Professor Emeritus of Nutrition, Yale University; Adjunct Professor of Biochemistry and Nutrition, University of Southern California, Los Angeles, California; Beverly Hills Medical Clinic, Beverly Hills, California. Program Committee, Wendell H. Griffith, Webster L. Marxer and Laurence E. Morehouse. Charles C Thomas, Publisher, Springfield, Illinois, 1967. 358 pages, $15.75.

CARDIAC SURGERY—By John C. Norman, M.D., Associate in Surgery, Harvard Medical School; Boston City Hospital and Children's Hospital Medical Center, Boston, Massachusetts, and 56 contributors. Foreword by Francis D. Moore, M.D., Appleton-Century-Crofts (Division of Meredith Publishing Company), New York, N.Y., 1967. 603 pages, $15 cloth; $9.75 paperback.

CLINICAL OBSTETRICS AND GYNECOLOGY—Volume 9, Number 4, December 1966—Toxemia of Pregnancy, edited by Frederick P. Zuspan, M.D., and Management of Genital Organ Prolapse, edited by R. B. Durfee, M.D. Published quarterly by Hoeber Medical Division, Harper & Row, Publishers, 49 East 33rd Street, New York, N. Y., 10016, about 1,200 pages per year. Subscription: $18.00 per year.

COMPLICATIONS IN SURGERY AND THEIR MANAGEMENT—Second Edition, with contributions by 52 authorities—Edited by Curtis P. Artz, M.D., F.A.C.S., Professor of Surgery and Chairman of the Department, Medical College of South Carolina; and James D. Hardy, M.D., F.A.C.S., Professor of Surgery and Chairman of the Department, University of Mississippi School of Medicine. W. B. Saunders Company, Philadelphia and London, 1967. 888 pages, $24.00.

DILEMMAS IN DRUG THERAPY—By Harry Beckman, M.D., Houghton Fellow in Clinical Research and Consulting Physician, Columbia Hospital, Milwaukee; Consulting Physician, Milwaukee County General Hospital; Honorary Physician, Saint Mary's Hospital, Milwaukee; Editor, Year Book of Drug Therapy; Professor (Emeritus) of Pharmacology, Marquette University School of Medicine. W. B. Saunders Company, Philadelphia and London, 1967. 404 pages, $11.50.

DRUGS IN CURRENT USE—1967—Edited by Walter Modell, M.D., F.A.C.P., Associate Professor, Clinical Pharmacology, Cornell University Medical College. Springer Publishing Company, Inc., 200 Park Ave., South, New York, 1966. 152 pages, $2.75 (Paperback).

DRUNK'S DIARY—By Theodore Van Dorn—Philosophical Library, Publishers, New York 16, N.Y., 1966. 116 pages. $4.00.

DISORDERS OF THE RESPIRATORY TRACT IN CHILDREN (by 29 Authorities)—Edited by Edwin L. Kendig, Jr., M.D., Professor of Pediatrics, Medical College of Virginia; Director of Child Chest Clinics, Medical College of Virginia Hospitals, Richmond. W. B. Saunders Company, Philadelphia and London, 1967. 834 pages, $26.00.

HUMAN NUTRITION AND DIETETICS—Third Edition—By Sir Stanley Davidson, B.A. Cantab., M.D., F.R.C.P., Edin., F.R.C.P. London, F.R.S.E., M.D. (Hon. Oslo), LL.D. (Edin.), Extra Physician to Her Majesty The Queen in Scotland; Regius Professor of Medicine, University of Aberdeen, 1930-1938; and R. Passmore, M.A., D.M. Oxon., Reader in Physiology, University of Edinburgh; formerly Lieutenant-Colonel, Indian Medical Service. Foreword by Lord Boyd Orr. The Williams and Wilkins Company, Baltimore, 1966. 864 pages, $19.50.

LABORATORY TESTS IN COMMON USE—Fourth Edition—By Solomon Garb, M.D., F.A.C.P., Professor of Pharmacology; Associate Professor of Community Health and Medical Practice, University of Missouri School of Medicine. Springer Publishing Company, Inc., New York, 1966. 192 pages, $2.85 (Paperback).

THE STOMACH—Including Related Areas in the Esophagus and Duodenum—The Thirteenth Hahnemann Symposium—Edited by Charles Thompson, M.D., Professor of Medicine and Head of Section of Gastroenterology, Hahnemann Medical College and Hospital, Philadelphia, Pa.; Donald Berkowitz, M.D., Associate Professor of Medicine, Temple University Medical School; Attending in Gastroenterology, Albert Einstein and Sidney Hillman Medical Centers, Philadelphia; and Edwin Polish, M.D., Associate Professor of Medicine, Hahnemann Medical College and Hospital, Philadelphia, Pa. Consulting Editor, John H. Moyer, M.D., Professor and Chairman, Department of Medicine, Hahnemann Medical College and Hospital, Philadelphia, Pa. Grune & Stratton, Inc., New York and London, 1967. 474 pages, $23.50.

PROGRESS IN HEMATOLOGY—VOLUME V—Edited by Elmer B. Brown, M.D., and Carl V. Moore, M.D., with 21 contributors. Grune & Stratton, Inc., New York and London, 1966. 399 pages, $16.75.

PROGRESS IN NEUROLOGY AND PSYCHIATRY—An Annual Review, Volume XXI—Edited by E. A. Spiegel, M.D., Professor and Head of the Department of Experimental Neurology, Temple University School of Medicine, Philadelphia. Grune & Stratton, Inc., New York and London, 1966. 657 pages, $17.50.

SCIENCE AND PSYCHOANALYSIS, VOLUME X—Sexuality of Women—Scientific Proceedings of the Tenth Annual Spring Meeting of the American Academy of Psychoanalysis—Edited by Jules H. Masserman, M.D., Professor and Co-Chairman of Psychiatry, Northwestern University, Chicago, Illinois. Grune & Stratton, Inc., New York and London, 1966.

YEAR BOOK OF ANESTHESIA (1966-1967 Year Book Series)—Edited by Stuart C. Cullen, M.D., Professor of Anesthesia and Dean, School of Medicine, University of California Medical Center, San Francisco. Year Book Medical Publishers, Incorporated, 35 East Wacker Drive, Chicago, 1966. 397 pages, $8.50.

Finer ALLERGENIC *Extracts*

- *specialized allergy service*
- *diagnostic and therapeutic extracts*

PROMPT • PERSONAL SERVICE

Frees you from details and delays.
Provides you with ready-to-use extracts.

Descriptive literature sent on request.

BERKELEY BIOLOGICALS
Second & Hearst Sts., Berkeley, Calif. 94710
2275 W. Lincoln Ave., Anaheim, Calif. 92801

STACEY'S for any Medical Book in Print
Retail Stores in Palo Alto and San Francisco

NEW BOOK

HANDBOOK OF POISONING: Diagnosis and Treatment. By Robert H. Dreisbach, M.D., Professor of Pharmacology, Stanford University School of Medicine, Palo Alto, California. 5th ed. 483 pages. 1966. Lange. $5 (paper).

Stacey's
326-9955
2575 HANOVER STREET
PALO ALTO, CALIFORNIA 94304

Please send me on approval:____________

Name____________
Street____________
City____________ State______

Conference on Narcotics Studies Liaison of Medicine, Law

Medicine and the law moved toward closer understanding and liaison in dealing with narcotics addicts at a national conference in New York City.

More than 100 physicians, law enforcement authorities and others concerned with the problem of addiction from throughout the nation gathered for a two-day workshop (January 16 and 17) to discuss how to improve the control of narcotics and the management of narcotic-dependent persons (addicts).

Participants in the conference agreed that it is important that state and local bodies be created to strengthen liaison with law enforcement authorities to help resolve questions of control and management.

The conference was called by the American Medical Association, with the joint sponsorship of the Medical Society of the State of New York and the County Medical Societies of New York City. Presiding were Lindsay E. Beaton, M.D., of Tucson, Ariz., Chairman of the AMA Council on Mental Health, and Dale C. Cameron, M.D., Chairman of the AMA Committee on Alcoholism and Addiction. Dr. Cameron, Superintendent of St. Elizabeth's Hospital, Washington, D.C., also is Chairman of the Committee on Problems of Drug Dependence of the National Academy of Sciences-National Research Council.

In attendance were national and selected state representatives of the medical profession, medical and pharmacy boards of licensure, federal and state law enforcement agencies, jurists, legislators, sociologists, psychologists and the clergy.

"Narcotic addiction is a sociological and medical problem. It also is a legal problem. Through the American Medical Association, the medical profession has long recognized the need for cooperation with law enforcement," said Henry L. Giordano, Commissioner of Narcotics of the Bureau of Narcotics of the Treasury Department, and the federal government's chief narcotics law enforcement officer.

"The increasing interest of the medical profession in exploring medical solutions to the addiction problem is heartening. Narcotic addiction has received much long-awaited attention in the last several years. The addict is definitely in the picture these days," Mr. Giordano said.

Mr. Giordano outlined the provisions of the new federal Narcotic Addict Rehabilitation Act of 1966, to become effective next month. In so doing he pointed out that "it helps to attack the roots of addiction by applying more flexible tools of medicine and psychiatry, rehabilitation and aftercare

(Continued on Page 36)

mudrane®

for

- EMPHYSEMA
- ASTHMA
- CHRONIC BRONCHITIS
- BRONCHIECTASIS

The fast-disintegrating uncoated tablet gives relief in 15 minutes

Each tablet contains:

Potassium Iodide	195 mg.
Aminophylline	130 mg.
Phenobarbital, Caution: May be habit forming	21 mg.
Ephedrine HCl	16 mg.

FEDERAL LAW PROHIBITS
DISPENSING WITHOUT PRESCRIPTION

Precautions: Usual for aminophylline-ephedrine-phenobarbital. Iodides may cause nausea, long use may cause goiter. Discontinue if symptoms of iodism develop.

Iodide contraindications: tuberculosis, pregnancy.

DOSAGE

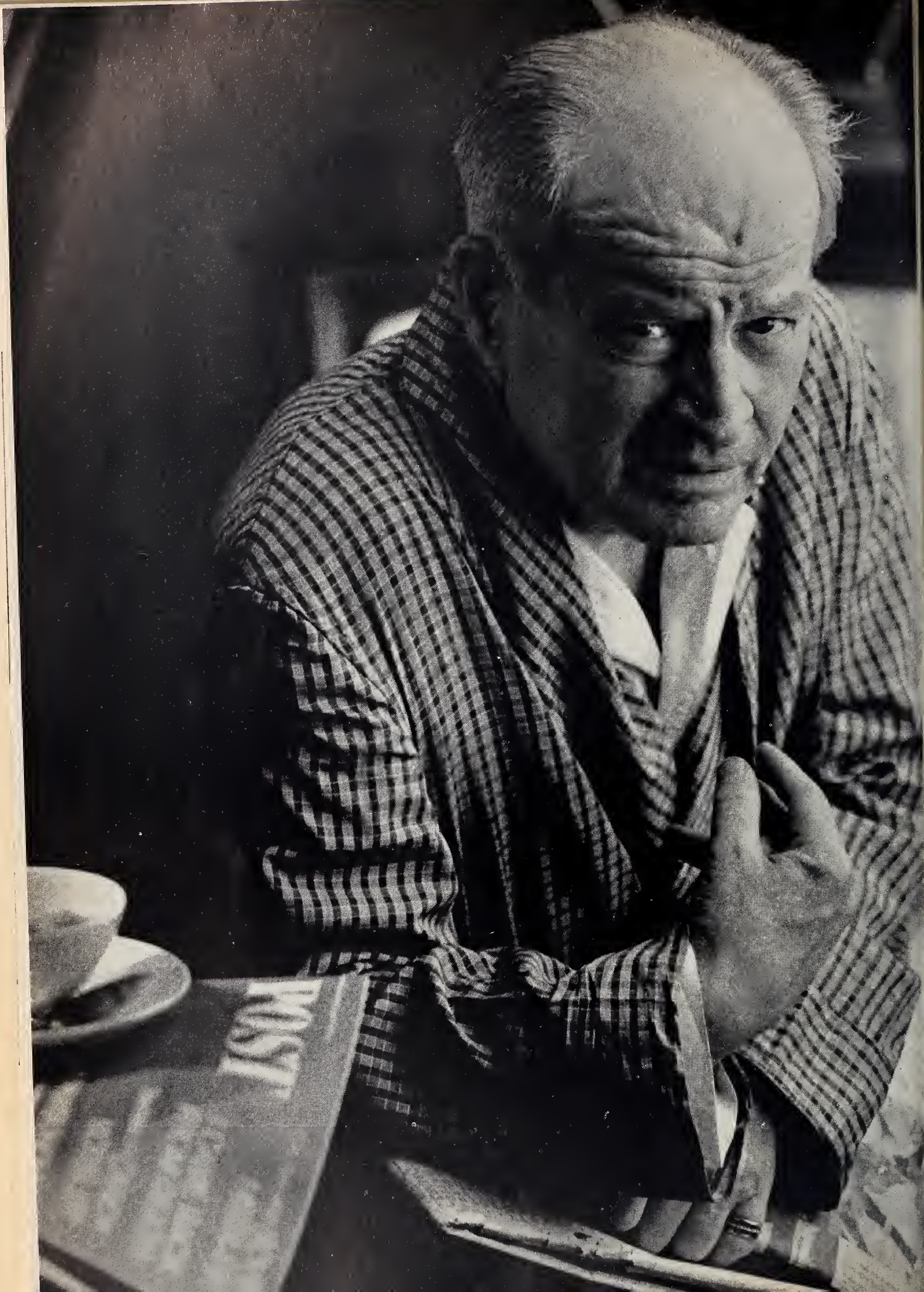
One tablet, with full glass of water, 3 or 4 times daily.

Dispensed in bottles of 100 and 1000 tablets.

MUDRANE GG—Formula, dosage and package identical to Mudrane—*except*—100 mg. glyceryl guaiacolate replaces the potassium iodide. The value of Mudrane cannot be enjoyed by a small group in which K.I. is contraindicated. Mudrane GG is prepared for this group.

MUDRANE GG ELIXIR—Four 5 cc teaspoonfuls is equivalent to one Mudrane GG tablet. Dosage adjusted to age and weight of child. Mudrane GG Elixir is for pediatric patients and those who think they cannot swallow tablets. Dispensed in pint and half gallon bottles.

WM. P. POYTHRESS & CO., INC.
RICHMOND, VIRGINIA 23217
Manufacturers of ethical pharmaceuticals since 1856



Worldwide clinical experience confirms the predictable therapeutic potential of Synalar

It is particularly gratifying that the promise of the advanced chemical design and high order of bioassay activity of Synalar (fluocinolone acetonide) has been confirmed by widespread therapeutic application. Indeed, the impressive clinical response rate of Synalar has been documented in no fewer than 232 papers from 22 countries.

Representative Clinical Results with Synalar*

Efficacy Documented in over 4,000 Patients

Condition	Number of Publications	Number of Patients	Significant Improvement†
Contact Dermatitis	27	750	713
Eczematous Dermatitis	21	472	409
Seborrheic Dermatitis	18	442	426
Atopic Dermatitis	24	460	426
Psoriasis	36	1,699	1,510
Neurodermatitis	18	351	324
Total	144	4,174	3,808

*Complete bibliography on request.

†Expressed by the authors as excellent, very good, good, complete remission of inflammation, etc.

PRESCRIBING INFORMATION
For initiation of therapy: Cream 0.025%,
5 and 15 Gm. tubes, 425 Gm. jars; *for*
emollient effect: Ointment 0.025%, 15
Gm. tubes; *for maintenance therapy:*
Cream 0.01%, 15 and 45 Gm. tubes, 120
Gm. jars; *for intertriginous or hairy*
ites: Solution 0.01%, 20 cc. and 60 cc.
plastic squeeze bottles; *for infected in-*
lammatory dermatoses: Neo-Synalar®
Cream (0.025% fluocinolone acetonide,
eomycin sulfate, equivalent to 0.35%
eomycin base), 5 and 15 Gm. tubes.
CONTRAINDICATIONS: Tuberculous, fungal,
nd most viral lesions of the skin, (in-
luding herpes simplex, vaccinia, and
aricella). Not for ophthalmic use. Con-
raindicated in individuals with a his-
ory of hypersensitivity to any of the
omponents. PRECAUTIONS: Synalar prep-
rations are virtually nonsensitizing and
onirritating. However, the solution may
roduce burning or stinging when ap-
ied to denuded or fissured areas. In
me patients with dry lesions, the solu-
on may increase dryness, scaling or
ching. While topical steroids have not
een reported to have an adverse effect
n pregnancy, the safety of their use on
egnant females has not absolutely
en established. Therefore, they should
t be used extensively on pregnant pa-
ents, in large amounts, or for prolonged periods of time. Prolonged use of any antibiotic may result in overgrowth of nonsusceptible organisms; if this occurs, appropriate therapy should be instituted. When severe local infection or systemic infection exists, the use of systemic antibiotics should be considered, based on susceptibility testing. SIDE EFFECTS: Side effects are not ordinarily encountered with topically applied corticosteroids. As with all drugs, however, a few patients may react unfavorably to Synalar under certain conditions. The neomycin in Neo-Synalar Cream rarely produces allergic reactions.

REFERENCES: 1. Lerner, L. J., Bianchi, A., Turkheimer, A. R., Singer, F. M., and Borman, A.: Anti-inflammatory steroids: potency, duration and modification of activities. Ann NY Acad Sci 116:1071 (Aug. 27) 1964. 2. Idem: Comparison of anti-granuloma, thymolytic and glucocorticoid activities of anti-inflammatory steroids. Proc Soc Exp Biol Med 116:385 (June) 1964. 3. Ringler, A.: Activities of adrenocorticosteroids in experimental animals and man, in Dorfman, R. I.: Methods of hormone research, New York, Academic Press, 1964. vol. III. pp. 234-280. 4. Gubersky, V. R.: To be published.

fluocinolone acetonide—an original steroid from
SYNTEX
LABORATORIES INC., PALO ALTO, CALIF.

For inflammatory dermatoses...
by any measure
a topical corticosteroid
of choice

Synalar®
(fluocinolone acetonide)

Milligram for milligram one of the most active topical corticosteroids available

Rapid and predictable in antiinflammatory and antipruritic activity

Results often comparable to those of systemic corticosteroids with fewer hazards

...but doctor,
I eat like a bird!

IN WEIGHT CONTROL
OBETROL®

Each OBETROL-10 tablet contains: Methamphetamine Saccharate 2.5 mg., Methamphetamine Hydrochloride 2.5 mg., Amphetamine Sulfate 2.5 mg., Dextro-amphetamine Sulfate 2.5 mg. (OBETROL-20 tablets contain twice this potency) Pat. #2748052.

This combination of amphetamines may be useful as an adjunct in the management of certain forms of obesity where an appetite depressant is indicated.

CONTRAINDICATIONS: Hypertension, advanced arteriosclerosis, coronary artery disease, cardiac arrhythmias, peripheral vascular disease, states of undue restlessness, anxiety, excitement, agitated depression, hyperthyroidism, idiosyncrasy to amphetamine, concomitant administration of a monoamine oxidase inhibitor. PRECAUTIONS: Use with caution in individuals with anorexia, insomnia, vasomotor instability, asthenia, psychopathic personality, a history of homicidal or suicidal tendencies, and individuals who are known to be hyperreactive to sympathomimetic agents, or emotionally unstable individuals who are known to be susceptible to drug abuse. Certain monoamine oxidase inhibitors may potentiate the action of Obetrol. SIDE EFFECTS: The most common side effects attended with the use of amphetamines include nervousness, excitability, euphoria, insomnia, dryness of mouth, nausea, vertigo, constipation, and headache. DOSAGE AND ADMINISTRATION: Initial adult dose is one-half to one 'Obetrol-10' tablet daily, preferably one-half to one hour before meals. This may be gradually increased to one 'Obetrol-10' or 'Obetrol-20' tablet one to three times daily as indicated. SUPPLIED: Tablets scored, in bottles of 100, 500, and 1000.

REQUEST SAMPLES AND LITERATURE

OBETROL PHARMACEUTICALS
Div. of Rexar Pharmacal Corp. Brooklyn, N.Y. 11207

OBETROL PHARMACEUTICALS • BROOKLYN, N.Y. 11207
Dr. ____________________
Address ____________________
City ____________ State ____________ Zip ______

Conference on Narcotics Studies Liaison of Medicine, Law

(Continued from Page 17)

supervision so that the addicts may return to society as useful members."

Anthony A. Lapham, Executive Assistant to David C. Acheson, Special Assistant for Enforcement to the Secretary of the Treasury, urged that organized medicine form committees on a regional or state basis to work with federal and state enforcement officials in dealing with addiction.

"An important step in the right direction," said Mr. Lapham, "was taken when members of the Committee on Alcoholism and Addiction of the AMA Council on Mental Health and members of the Committee on Problems of Drug Dependence of the National Academy of Sciences-National Research Council agreed to act jointly in an advisory capacity to the Commissioner of Narcotics," Mr. Lapham said. "It would be very helpful as a second step if medical bodies could be organized on a regional or state basis," he said.

Jerome H. Jaffe, M.D., University of Chicago psychiatrist, in discussing developments in treatment of addicts, pointed out that "A comprehensive treatment should attempt to help the opiate-dependent individual become a mature, stable, responsible, productive and nondrug-dependent member of his own community."

"It is apparent," said Dr. Jaffe, "that a growing segment of both the medical and legal professions are refusing to be bound either to old stereotypes of 'the addict' or to outmoded treatment models. We must develop skills to select the right treatment for each patient."

Lawrence C. Kolb, M.D., Director of the New York State Psychiatric Institute, pointed out that the physician sometimes faces a difficult problem in long-term treatment of an addict. The physician must establish a strong emotional bond with the patient to gain his confidence and thus carry out effective therapy. To do this, it sometimes is necessary for the physician to prescribe drugs to maintain addiction for a period of time while he is gaining the confidence of the patient, in order that effective therapy can be carried out.

Dr. Kolb pointed out that local liaison committees of physicians and law enforcement authorities are needed to counsel with the physician in practice to help him make proper decisions in such instances.

Particular attention was paid to the importance of developing improved programs in the five states having the greatest drug dependence problem. These are Illinois, New York, California, Michigan and the District of Columbia. Representatives of these states met toward the end of the conference to study ways and means of improving programs and liaison in their respective states.

Especially...

When the bronchitis→emphysema syndrome is complicated by hypertension or heart disease—

ORAL ELIXOPHYLLIN®

Each 15 cc. contains theophylline 80 mg.; alcohol 3 cc.

. . . has consistently provided rapid, sustained bronchodilation[1] without the hazards of epinephrine-like drugs.

Used in recommended t.i.d. dosage "[Elixophyllin] . . . rapidly produces an effective blood level comparable to that produced when the drug is administered intravenously."[2] Such serum levels have been correlated[3] with a proven significant increase in pulmonary function. By maintaining effective bronchodilation with Elixophyllin, the incidence of acute attacks may be reduced, increasing damage to bronchial tissue arrested, functioning tissue preserved, and progression toward intractable emphysema retarded.

Unusually well tolerated—Unlike many available compounds, the theophylline in Elixophyllin is not precipitated in the presence of gastric acid, and thereby avoids gastric irritation. Moreover, by remaining in solution, Elixophyllin is carried without delay into the blood stream for *predictable bronchodilative action.*

Simple, precise oral medication—minimizes danger of self-overmedication:

Adults, 45 cc. (3 tablespoonfuls) before breakfast, at 3 P.M., and before retiring. After two days, 30 cc. (2 tablespoonfuls) t.i.d. Does not contain epinephrine-like drugs; alcoholic content is non-grain, hypo-allergenic. May be contraindicated in peptic ulcer and gout.

Sherman Laboratories

DETROIT, MICHIGAN

1. Corroborated by 40 published studies.
2. Miller, W. F.: Postgrad. Med. *39*:230-239 (Mar.) 1966.
3. Jackson, R. H., et al.: Dis. Chest *45*:75-85 (Jan.) 1964.

★

Specialized Service
IN
PROFESSIONAL LIABILITY INSURANCE

is a high mark of distinction

THE
MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA
Professional Protection Exclusively since 1899

NORTHERN CALIFORNIA OFFICE:
Gordon C. Jones and John K. Galloway, Representatives
1518 Fifth Avenue, San Rafael, Telephone: 415-453-5140
Mailing Address: P.O. Box 1079, San Rafael 94902
SOUTHERN CALIFORNIA OFFICE:
Davis S. Spencer and Grady T. Oyer, Representatives
Suite 3, 33 East Huntington Drive, Arcadia, Tel. 213-681-5239
Mailing Address: P.O. Box 543, Arcadia 91008

Easy on the Budget... Easy on the Mother

Tablets & Elixir

For Iron Deficiency Anemia

FAMOUS
Fergon®
brand of FERROUS GLUCONATE

C.M.A.P. CODE # 5261

BREON LABORATORIES INC.
Subsidiary of Sterling Drug Inc.
90 Park Avenue, New York, N.Y. 10016

Scientific Data Lacking On Cancer "Vaccine"

Two Cleveland medical leaders say in the January 30 *Journal of the American Medical Association* that administration of a so-called cancer "vaccine" to humans is completely unwarranted until scientific tests prove the safety and effectiveness of the product.

So far, there are no scientific data to justify experimentation on humans with a "vaccine" developed by the Rand Corporation of Cleveland, say David Fishman, M.D., president of the Cleveland Academy of Medicine, and Austin S. Weisberger, M.D., chairman of the Department of Medicine of Western Reserve University.

Their report on the vaccine, the publicity it has received, and an investigation of the product by a committee of the Cleveland Academy of Medicine, appear in the Question and Answer section of the *Journal.*

Their report is in answer to a question by a Barron, Wis., physician regarding the usefulness of the product, one of several similar ones received by the *Journal* in recent months.

An editor's note after the answer announces that directors of the Cleveland Academy of Medicine have declared it unethical for any Academy member to use the Rand vaccine for new patients. This policy, effective January 10, 1967, will stand "until investigative and research data are available warranting such human experimentation," the Academy said.

Publicity about the so-called vaccine a few months ago caused the Cleveland Academy of Medicine to invite officials of the Rand Corporation to "provide . . . more factual information."

Said Drs. Weisberger and Fishman: "The following points were elicited: (1) There have been no experiments with animal tumors to test the efficacy of the 'vaccine.' There have been no presentations at scientific assemblies, no publications, and no critical review or evaluation of data.

"In fact, it has not even been established that the Rand Corporation has, at this time, essential data establishing the specificity of the tumor antigens being employed, the production of specific antibodies against the tumor antigens, and the lack of cross-reaction with normal tissue antigens.

"(2) Experiments for toxic or other side effects have been performed on one horse, 12 rabbits, and 40 mice for a 'minimum of three injections.'

"(3) The human experiments being performed cannot be expected to demonstrate the efficacy of the 'vaccine.' No controls or double-blind (statistical) studies are being employed. The material is being released to any licensed physician and osteopath requesting supplies of the 'vaccine' in accordance with Ohio law. However, this is being done without regard for the clinician's experience in

(Continued on Page 65)

California
Medicine

MEDICAL PROGRESS

The Hypothalamic-Pituitary-Adrenocortical System

Clinical Evaluation by Pharmacologic Techniques

GRANT GWINUP, M.D., *Los Angeles*

■ *The hypothalamic-pituitary-adrenocortical system is a complex negative feed-back control mechanism. Under non-stress conditions it functions to maintain a circadian rhythm of adrenal steroid production which is secondary to a circadian fluctuation in the sensitivity to suppression of* ACTH *by hydrocortisone. Any stressful stimulus causes an augmentation of steroid production which is due in part to a decrease in sensitivity of* ACTH *to suppression. Aldosterone production is not primarily controlled by* ACTH.

The increased understanding of this system and of its pathologic alterations has led to the development of a number of pharmacologic techniques which have proved most valuable in evaluating its integrity. Hypothalamic-pituitary function can be assessed by the response to the administration of dexamethasone, methopyrapone, bacterial pyrogen and vasopressin. Adrenal cortical function can be assessed by the response to ACTH, *dexamethasone and spironolactone.*

FOR MANY YEARS, it has been known that there is a fundamental "push-pull" relationship between the pituitary gland and the adrenal gland which serves to control the output of corticosteroids from the adrenal. Only recently, however, has the complexity of this relationship come to be appreciated. New information has given new insights and has made possible additional diagnostic techniques which have proved to be most helpful in diagnosing abnormalities of this system. Many of the newer diagnostic tests measure the response to the administration of a pharmacologic agent. The purpose of this review is to consider the controlling mechanisms and to discuss pharmacologic techniques used to determine their integrity.

Controlling Mechanisms

The essence of the pituitary-adrenal relationship is a negative feedback system. If the concentration of circulating adrenal steroids in the plasma tends to fall, ACTH is released by the pituitary gland in increased quantities. ACTH stimulates the adrenal

---

From the Department of Medicine, Division of Endocrinology, University of California, California College of Medicine, Los Angeles.

Reprint requests to: Department of Medicine, University of California. College of Medicine, 1721 Griffin Avenue, Los Angeles 90031.

gland to elaborate a greater quantity of steroids so that equilibrium is reestablished. However, as the production of adrenal steroids continues, further release of ACTH by the pituitary is inhibited and this serves to avoid overcompensation by an excessive production of steroids.

It is appreciated that such a simple feedback control system would function to keep the concentration of adrenal corticosteroids relatively constant in the blood at all times. However, if one measures the concentration of steroids in the plasma, one finds that the level does not remain constant, but fluctuates through a cycle of 24 hours' duration. The greatest adrenal activity occurs in the very early hours of the morning and it diminishes slowly and progressively until there is only minimal activity in the late hours of the evening, resulting in what has come to be called the circadian rhythm of steroid secretion. (*Circadian* means "about the day" and appears to be a desirable replacement for the term *diurnal,* which has approximately the same meaning but which unfortunately sounds more like a urologic term.) Recent studies indicate that the circadian steroid secretion pattern is secondary to a circadian secretion pattern of ACTH, or more properly to a circadian fluctuation in the sensitivity to suppression of ACTH by hydrocortisone.[15]

The nature of the stimuli which initiate this basic rhythm are not completely understood. That the circadian rhythm is not simply secondary to the ocular stimulus of daylight is indicated by the fact that blind persons evidence the same rhythmic output of corticosteroids as do sighted individuals. Since persons who work night shifts appear to have a pattern of steroid secretion similar to those who work day shifts, it would seem that physical activity is not the initiating stimulus. However, if a person moves from one part of the world to another, his rhythmic pattern of steroid secretion (after a short period of adjustment) assumes the cycle of others living at the same longitude.

It should be mentioned that of the various adrenal secretory products, only hydrocortisone, the important glucocorticoid secreted by the human adrenal cortex, is responsible for suppressing ACTH secretion. It is normally produced in quantities of approximately 20 mg a day. All other naturally-occurring steroids are relatively ineffective in producing such suppression. All of the potent synthetic steroids do effectively suppress ACTH. Aldosterone, the important mineralocorticoid of the human adrenal cortex, is as potent, milligram for milligram, as hydrocortisone in suppressing ACTH; but since it is only produced in quantities of approximately 100 mcg per day, it is biologically unimportant in this regard.

To this point we have considered only the production of adrenal steroids under normal nonstress conditions. It is well known, however, that under circumstances of maximum stress the production of hydrocortisone by the adrenal gland can reach levels 10 times the normal baseline output. This response to stress would seem to be caused, in part at least, by a stress-induced change in the sensitivity of ACTH suppressibility. It has been demonstrated, for instance, that if a person undergoing a relatively minor surgical procedure such as a hemorrhoidectomy (it should be noted that this is not often considered a minor procedure by the patient) is given an amount of hydrocortisone slightly in excess of that which he would normally produce in such circumstances, there is no increase in ACTH production and consequently no increase in endogenous hydrocortisone production. This indicates that during stress of this order, ACTH can still be suppressed; however, the circulating levels of hydrocortisone necessary to effect suppression in the stressed state are much higher than those normally circulating in the unstressed state. If, however, a person undergoes an operation such as a gastrectomy (considered by all to be a major surgical procedure), then it has been shown that the administration of hydrocortisone in quantities greatly in excess of those which would normally be produced under such circumstances fail completely to suppress ACTH elaboration.

Formerly it was thought that suppression of ACTH by hydrocortisone as well as its increased production secondary to stress was mediated by the pituitary gland. There is now a great deal of evidence from a number of sources to indicate that both of these effects on the pituitary take place through the hypothalamus, which acts as an essential intermediate in a sort of double-play combination consisting of adrenal-to-hypothalamus-to-pituitary.

It will be recalled that the blood supply to the anterior pituitary gland is somewhat different from the blood supply to most of the tissues of the body. The anterior pituitary receives the predominate portion of its blood from a large venous channel which courses down the pituitary stalk. This is a true portal system, in that this blood comes directly from a capillary network originating in the area of the hypothalamus. There is convincing evidence that specific peptides called "corticotropin-releasing factors," produced by cells in the hypothalamus, are carried by the "portal system" to the pituitary gland and play a major role in controlling the release of ACTH. One of these peptides has been particularly well characterized and has been found to be very similar in its molecular composition to antidiuretic hormone or vasopressin.[6]

It should be mentioned in passing, that the production of aldosterone is influenced, but not primarily controlled through ACTH action; rather it is controlled through an increased generation of angiotensin acting on the adrenal cortex.[2]

With this framework of the structure of the system controlling adrenal cortical function in mind, it is possible to understand some of the pharmacologic procedures used by endocrinologists in the clinical evaluation of the integrity of the system.

Assessing Hypothalamic-Pituitary Function

The detection of abnormalities in the ability of the pituitary gland to properly produce ACTH has been compromised, to say the least, by the lack of a simple assay procedure with sufficient sensitivity to demonstrate subnormal levels of ACTH. Without such an assay it has been necessary to rely heavily on pharmacologic methods of eliciting an ACTH response and to measure an increase in adrenal secretory products in the patient's blood or urine as a reflection of that response.

In 1959, Liddle[10] and his associates demonstrated that pituitary function could be tested by the administration of methopyrapone or SU-4885. The latter term, which was the manufacturer's code designation for the compound, unfortunately has persisted despite the fact that it sounds very much like a telephone number and is approximately as difficult to remember. Methopyrapone blocks adrenal synthesis of hydrocortisone, but does not block the production of other steroids. In a normal subject, if hydrocortisone cannot be produced, ACTH secretion is not inhibited through the negative feedback mechanism. Under the influence of increased ACTH secretion there is an increased production of adrenal steroids other than hydrocortisone, and these are measured by the commonly used urinary steroid methods. In normal persons therefore, the administration of methopyrapone produces an increase in urinary steroid secretion, but in persons with disruption of the hypothalamic-pituitary relay system such an increase does not occur. The methopyrapone test is a means for determining the integrity of hypothalamic-pituitary unit in regard to its function in the negative feedback control system. The normal response to the oral administration of methopyrapone, 500 mg every six hours for two or three days, is a two-fold to three-fold increase in urinary steroids. The failure for such a response to occur in a patient who has intact adrenals as demonstrated by a suitable response to ACTH administration, is presumptive evidence of hypothalamic-pituitary dysfunction.[10]

Another method for assessing the ability of the hypothalamic-pituitary unit to produce an increase in ACTH is to measure the urinary steroid response to the stressful stimulus provided by an injection of bacterial pyrogen. Unfortunately, certain untoward symptoms must be provoked in order to achieve a definite adrenal response, and for this reason this test does not have wide clinical acceptance.

Our studies of the adrenal response to the injection of a pharmacologic dose of synthetic vasopressin suggested that such a response might be due to the similarity between vasopressin and of corticotropin-releasing factors,[8] and that vasopressin might serve as a suitable stand-in for corticotropin-releasing factors (until these factors are available for clinical use) in assessing the ability of the pituitary to release ACTH. We found that in normal subjects, the intramuscular injection of 10 units of synthetic vasopressin consistently produced a two-fold increase in the concentration of plasma hydrocortisone one hour following the injection while patients with pituitary tumors had a diminished response or none at all.[9] In a limited number of patients we found a similar response when commercially available vasopressin was used instead of synthetic vasopressin.

Detecting Adrenal Insufficiency

Since ACTH became commercially available, testing the integrity of the adrenal cortex has been a relatively easy task. The standard procedure consists of the intravenous administration of ACTH.[4] The increased production of hydrocortisone is usually assessed by measuring either urinary 17-hydroxy steroids or urinary ketogenic steroids, and the increased production of adrenal androgen by measuring urinary 17-ketosteroids. The adrenal production of androgens as well as hydrocortisone is controlled to a large extent by ACTH. In males, of course, under normal circumstances, approximately one-third of the urinary 17-ketosteroids are derived from androgen produced in the testicle, which is not under the control of ACTH. With the standard infusion test, ACTH is administered intravenously over either six or eight hours daily for four or five days. Although a definite response can generally be appreciated by the first or second day of stimulation, it is sometimes advisable to continue for a full five days in order to rule out the presence of "relative" adrenal insufficiency. In such a case, urinary steroids may increase for the first day or two, but subsequently, as the reserve capacity of the adrenal is exceeded, their secretion may diminish again in the latter days of the test.[17] A normal adrenal gland should continue to respond to the challenge and to increase progressively its output of steroids each day for five days.

It is not generally appreciated that the number of units of ACTH used in the infusion is greatly in excess of the maximal effective dose. More than 2 units over an eight-hour period gives no increased adrenal response, and is added only to serve as a most generous safety factor or out of deference to convention. The time over which the infusion is given is important, however. Each additional hour of ACTH administration produces a greater response.

Since a quantitative response is not generally necessary in establishing a clinical diagnosis of adrenal insufficiency, the procedure for stimulating the adrenal has been modified in order to avoid the necessity of intravenous infusions. Under such circumstances, a slowly absorbed preparation of ACTH, such as corticotropin-zinc, is injected intramuscularly every 12 hours for four or five days and 24-hour urinary specimens are assayed for adrenal steroids. When ACTH is given intramuscularly, it is generally necessary to use larger quantities of ACTH (such as 20 units every 12 hours) since it is not as active when it is administered by this route.

In 1965 we suggested a simplified screening test for adrenal insufficiency[12] which obviated the necessity of intravenous infusion and urine collections. In this test, 25 units of aqueous ACTH is administered intramuscularly and, one hour after injection, the plasma-hydrocortisone level is assayed fluorometrically and compared with the plasma-hydrocortisone level immediately preceding the injection. Normally, the post-injection concentration is more than double the pre-injection concentration. This has proved in our experience to be a reliable screening procedure with the obvious advantage of the very short duration of the test and elimination of the need for collecting an accurate 24-hour urine specimen, which is always difficult.

If, however, there is any doubt about the interpretation of the results of either test in which ACTH is administered by the intramuscular route, it is advisable to repeat the procedure, using daily intravenous infusions of ACTH.

Any patient in whom a diagnosis of adrenal insufficiency is under serious consideration should be given a small daily dose of a potent synthetic glucocorticoid such as dexamethasone, 1.0 mg on the day before and the same amount on each day of ACTH administration. This does not influence in any way the results of the test and appears to offer protection against the untoward serious reactions to ACTH which may occur in patients with adrenal insufficiency.

Demonstrating Adrenal Hyperplasia and Tumors

The excessive production of adrenal steroids may result from either of two conditions: An adrenal tumor (benign or malignant), or bilateral adrenal hyperplasia.

An adrenal tumor producing excessive quantities of hydrocortisone and/or androgens is by definition autonomous and not under the control of the hypothalamic-pituitary system. In other words, the tumor does not stop producing steroids when ACTH levels fall, and, in point of fact, if the tumor makes hydrocortisone excessively (some tumors secrete mainly androgens) then ACTH is continuously suppressed. Such adrenal tumors generally produce high 24-hour urinary steroid levels which are not lowered by the administration of large dosages of dexamethasone (2 mg every six hours) which assures complete suppression of ACTH.[11] Therefore, in any case in which the administration of dexamethasone in such dosage for several days fails to bring about a decided reduction in urinary steroids, the presence of an adrenal tumor is suspected.

Other adrenal tumors produce aldosterone as their only important secretory product. These tumors cause high blood pressure and although they have been previously regarded as an unusual cause of hypertension, Conn[3] recently produced evidence indicating that such tumors are responsible for a significant proportion of cases of so-called "essential" hypertension. Conn showed that such patients may have perfectly normal serum electrolytes and can therefore not be distinguished from other persons with hypertension by simple electrolyte determination. The detection of patients with this curable form of hypertension has now become a difficult and demanding problem.

The diagnosis of an aldosterone-producing tumor in a hypertensive patient with normal electrolytes can be made by demonstrating depressed serum renin activity following the application of appropriate physiologic stimuli. Such procedures, however, can only be accomplished in a few research laboratories.[3] We have recently shown that patients with aldosterone-producing tumors can be more easily detected by a simple pharmacologic test.[7] We have found that, in such patients, normalization of blood pressure is predictable after several weeks of treatment with spironolactone, 50 mg four times daily. The striking response in blood pressure to spironolactone contrasts decidedly with the minimal effect on blood pressure of hydrochlorthiazide, 50 mg twice daily, administered during a subsequent four-week control period. Patients with hypertension not caused by an adrenal tumor characteristically show only a moderate response to either spironolactone or hydrochlorothiazide, and the greater effect is usually produced by the thiazide.

Bilateral adrenal hyperplasia (Cushing's syndrome) is the result of an excessive or inappropriate secretion of ACTH.[5,18] Occasionally it may result from the production of an ACTH-like substance by a malignant neoplasm of diverse origin.[1,13] Such cases will not be considered in this review. Nelson[14] and his associates have shown that in early or mild cases of Cushing's syndrome there is only a loss of the increased sensitivity of hypothalamic-pituitary suppressibility to hydrocortisone which normally occurs in the latter part of each day so that the circadian rhythm of steroid secretion is lost. The concentration of serum hydrocortisone in such cases may not be outside of normal limits in the early hours, but the expected fall later in the day does not occur.[2] In more advanced cases, the resistance of the hypothalamus and pituitary to suppression is even further increased and the concentration of serum hydrocortisone is consistently above normal levels. The "thermostat" is set too high but it is still operating, and although the resistance of ACTH to suppression is increased, suppression can still be achieved with a higher than normal concentration of hydrocortisone. This is similar to the way in which the heat can be turned off in an overheated room by burning a candle under the thermostat. Liddle[11] and coworkers defined this relationship and devised a clinical application for candle-burning which has proved to be a most valuable pharmacologic test for distinguishing patients with Cushing's syndrome from normal or obese subjects (obese subjects characteristically have higher urinary steroids and they are continually being considered as possible cases of a "glandular disorder") and from patients with hydrocortisone producing adrenal tumors.

Liddle showed that the administration of dexamethasone, 0.5 mg every six hours for several days, consistently caused a pronounced decrease in urinary steroid secretion in normal persons but did not produce a significant reduction in the urinary steroids of patients with Cushing's syndrome. The subsequent increase of the dosage of dexamethasone to 2.0 mg every six hours, however, did cause suppression of steroid secretion in patients with adrenal hyperplasia, but not in patients with adrenal tumors.

More recently, Pavatos, Smilo and Forsham[16] suggested a similar but abbreviated test designed simply to separate patients with adrenal hyperplasia or tumors from normal and obese subjects. They showed that the administration of a single dose of dexamethasone, 1.0 mg orally at 11:00 p.m., will cause a predictable depression of the concentration of plasma 17-hydroxycorticosteroids the following morning in normal and obese subjects. Patients with adrenal hyperplasia or tumor are resistant to suppression when this procedure is used.

In the future our ability to evaluate the integrity of the hypothalamic-pituitary-adrenocortical system will be greatly improved by the development of assays for ACTH which are more sensitive and, it is to be hoped, more simple. Similar assays for corticotropin-releasing factors, and angiotensin would be most welcome additions to our diagnostic box of tools. The availability of synthetic corticotropin-releasing factors would lead to the development of other pharmacologic techniques which might revolutionize our ability to evaluate ACTH production. In the meantime, the correlation of the results of the various available pharmacologic techniques may help us to better localize the functional lesion in individual patients and lead to an increased understanding of the function of the hypothalamic-pituitary-adrenocortical system in man.

REFERENCES

1. Christy, N. P.: Adrenocorticotropin activity in the plasma of patients with Cushing's syndrome associated with pulmonary neoplasms, Lancet, 1:85-86, 14 January 1961.

2. Conn, J. W., Rovner, D. R., and Cohen, E. L.: Normal and altered function of the renin-angiotensin-aldosterone system in man, Ann. Intern. Med., 63:266-284, August 1965.

3. Conn, J. W., Cohen, E. L., Rovner, D. R., and Nesbit, R. M.: Normokalemic primary aldosteronism: A detectable cause of curable "essential" hypertension, J.A.M.A., 193:200-206, 19 July 1965.

4. Eik-nes, K., Sandberg, A. A., Nelson, D. H., Tyler, F. H., and Samuels, L. T.: Changes in plasma levels of 17-hydroxycortico-steroids during the intravenous administration of ACTH I. A test of adrenocortical capacity in the human, J. Clin. Invest., 33:1502-1508, November 1954.

5. Givens, J. R., Ney, R. L., Nicholson, W. E., Graber, A. L., and Liddle, G. W.: Absence of a normal diurnal variation of plasma ACTH in Cushing's disease, Clin. Res., 12:267, April 1954.

6. Guillemin, R.: Hypothalamic polypeptides releasing pituitary hormones, Metabolism, 13:1206, October 1964.

7. Gwinup, G., and Steinberg, T.: The pharmacologic diagnosis and treatment of primary aldosteronism, Manuscript in preparation.

8. Gwinup, G.: Studies on the mechanism of vasopressin-induced steroid secretion in man, Metabolism, 14: 1282-1286, December 1965.

9. Gwinup, G.: A test for pituitary function using vasopressin, Lancet, 2:572-573, 18 September 1965.

10. Liddle, G. W., Estep, H. L., Kendall, J. Williams, W. C., Jr., and Townes, A. S.: Clinical application of a new test of pituitary reserve, J. Clin. Endocr., 19:875-894, August 1959.

11. Liddle, G. W.: Test of pituitary-adrenal supressibility in the diagnosis of Cushing's syndrome, J. Clin. Endocr., 20:1539-1560, 19 December 1960.

12. Maynard, D. E., Folk, R. L., Riley, T. R., Wieland, R. G., Gwinup, G., and Hamwi, G. J.: A rapid test for adrenocortical insufficiency, Ann. Intern. Med., 64:552-556, March 1966.

13. Meador, C. K., Liddle, G. W., Island, D. P., Nicholson, W. E., Lucas, C. P., Nuckton, J. G., and Leutscher, J. A.: Cause of Cushing's syndrome in patients with tumors arising from "nonendocrine" tissue, J. Clin. Endocr., 22:693-703, July 1962.

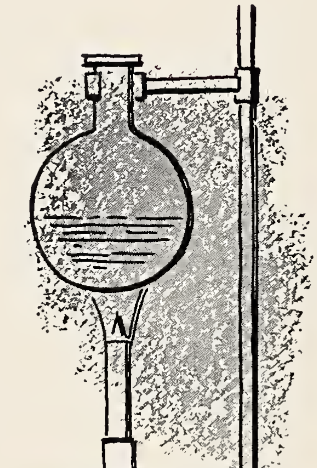
14. Nelson, D. H., Sprunt, J. G., and Mims, R. B.: Plasma ACTH determinations in 58 patients before and after adrenalectomy for Cushing's syndrome, J. Clin. Endocr., 27:722-728, July 1966.

15. Nichols, T., Nugent, C. A., and Tyler, F. H.: Diurnal variation in suppression of adrenal function by glucocorticoids, J. Clin. Endocr., 25:343-349, March 1965.

16. Pavlatos, F. C., Smilo, R. P., and Forsham, P. H.: A rapid screening test for Cushing's syndrome, J.A.M.A., 193:720, 30 August 1965.

17. Smith, H.: Compensated adrenocortical failure, Lancet, L:1077-1078, 18 May 1963.

18. Williams, W. C., Jr., Island, D., Oldfield, R.A.A., Jr., and Liddle, G. W.: Blood corticotropin (ACTH) levels in Cushing's disease, J. Clin. Endocr., 21:426-432, April 1961.



Clinical Use of a New Mitral Disc Valve

JEROME HAROLD KAY, M.D., HAROLD K. TSUJI, M.D., JOHN V. REDINGTON, M.D., YASUNARU KAWASHIMA, M.D., YUZURU KAGAWA, M.D., TAKASHI YAMADA, M.D., PETER CAPONEGRO, M.D., AND ADOLFO MENDEZ, M.D., *Los Angeles*

■ *A disc valve of new design was used successfully for the replacement of the mitral valve in patients with rheumatic mitral valve disease. This valve would appear to have the following advantages over the mitral ball valve prosthesis:*

- *Lower left atrial pressure after replacement.*
- *Elimination of the hazard of left ventricular outflow tract obstruction with mitral valve replacement.*
- *Decreased incidence of thromboembolization.*
- *Abolition of possibility of ventricular septal irritation.*

Despite the better outlook for this valve compared with the ball valve for mitral valve substitution, the mitral valve should always be repaired whenever feasible. Repair is possible in the majority of patients.

USUALLY THE BEST treatment for a stenotic or insufficient mitral valve is surgical repair, but there are some patients in whom prosthetic valvular replacement is necessary—patients with moderate to pronounced calcification of the valve or decided fibrotic deformity. In patients with mitral insufficiency we have found repair to be of permanent value and free of the serious complication of peripheral embolization which may occur following replacement with a ball valve.

Because of the high incidence of peripheral embolization, the possible occurrence of left ventricular outflow tract obstruction, and the arrhythmia due to ventricular septal irritation that may occur with mitral ball valve replacement, we developed a disc valve (Figure 1). Experimentally in dogs this valve appeared to have important advantages over the ball valve. Following good experimental results, the valve was used clinically for mitral valve substitution in 14 patients with rheumatic mitral valve disease in whom repair of the valve was not possible. Between the time of the first and the fourteenth replacement operation using the disc valve a total of 73 patients were operated upon for mitral valve disease, 59 of them having had repair of the existing valve. In the following case, prosthetic replacement was carried out.

Report of a Case

The patient, a woman born in 1933, had rheumatic fever at the age of nine years and had had shortness of breath since the age of 13. When she consulted us in October 1962, she had pronounced symptoms of congestive failure, including paroxysmal nocturnal dyspnea, chronic cough and inability

---

From the Department of Surgery of the University of Southern California School of Medicine, the Los Angeles County General Hospital, and the Saint Vincent's Hospital.

Aided by grants HE 09223-02 and HE 09883-01 from the United States Public Health Service, grants 354 and 373 from the Los Angeles County Heart Association, a grant from the Children's Heart Foundation of Southern California, and a grant from the Los Angeles Thoracic and Cardiovascular Foundation.

Submitted 9 September 1966.

Reprint requests to: 318 South Alvarado Street, Los Angeles 90057 (Dr. Kay).

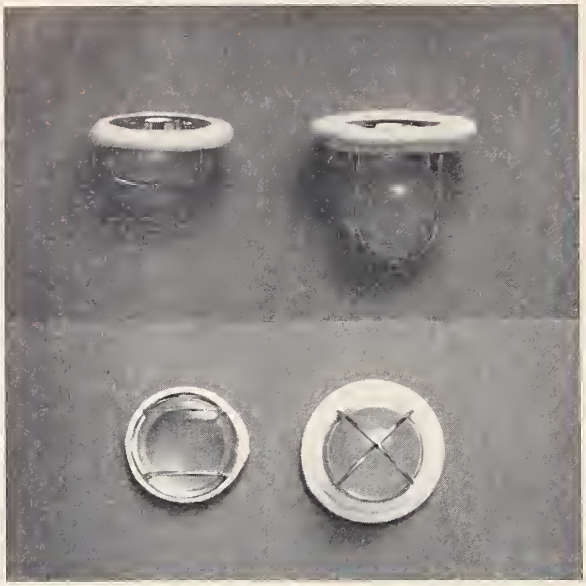


Figure 1.—On the right is the Starr-Edwards ball valve and on the left the new Kay-Shiley disc valve. Note that for the same cross sectional area, there is a pronounced decrease in the length of the cage, as well as the size of the disc compared with the ball. Note also the parallel rather than perpendicular struts.

to be out of bed because of dyspnea and fatigue. She was on a low salt diet and was taking digitalis and diuretics. A benign tumor of the breast had been removed in 1958 and a hysterectomy and partial removal of the bladder was performed in 1960 for a carcinoma.

On physical examination, blood pressure was 95/60 mm of mercury. The pulse had a sinus rhythm. The cardiac apex was at the left sixth intercostal space at the anterior axillary line. There was a grade V/VI apical diastolic rumble, an opening snap and a loud M_1. There was a grade IV/VI systolic murmur immediately to the left of the sternum at the fourth intercostal space. No rales were noted and there was no leg edema or hepatomegaly.

An electrocardiogram was consistent with right and left atrial hypertrophy as well as right ventricular hypertrophy. There was sinus rhythm with occasional ventricular premature contractions.

X-ray studies showed grade IV/VI cardiac enlargement. This included grade III/VI enlargement of the right and left atria and grade II/VI enlargement of the right and left ventricles. The changes described were indicative of combined mitral stenosis and insufficiency as well as tricuspid insufficiency.

Operation was carried out 30 November 1962, with extracorporeal circulation through a heart-lung machine. The mitral valve was found to be heavily calcified and stenotic and there was pronounced calcification of the aortic leaflet. The mural leaflet was rigid and thickened. There was insufficiency at the center of the valve. The valve orifice admitted one finger. A knife and finger fracture technique was used to open the anterolateral commissure. The chordae tendinae were shortened and fused. The posteromedial commissure was also opened through a great deal of calcium. Posteromedial annuloplasty was performed.[8] This valve was obviously a very poor one, but because this was early in the history of valve replacement, it was deemed wise to repair rather than replace the valve. At the end of the repair procedure, the valve was rigid but admitted two fingers and was no longer insufficient. Examination of the tricuspid valve revealed stenosis rather than insufficiency. The valve admitted one to one and one-half fingers and there was fusion of all three commissures. The commissure between the anterior and the septal leaflet was opened with a knife and the tricuspid valve then easily admitted two fingers without evidence of tricuspid insufficiency.[13]

A tracheostomy was performed. The patient's postoperative course was prolonged and she left the hospital on the 29th postoperative day. She did well at home and continued to improve. After a time she was able to carry on with her household duties and look after her small children, but in February 1965 she began to notice dyspnea on exertion and she had difficulty with housework. She had no orthopnea nor paroxysmal nocturnal dyspnea. At that time she was taking digitalis and was following a low salt diet.

When the patient was examined on 9 September 1965, the blood pressure in the right arm (recumbent) was 120/70 mm of mercury with a sinus rhythm. She had a chronic cough and rales were noted at the right base posteriorly. There was one plus edema. The liver was not tender but the edge was palpable two to three fingerbreadths below the costal margin.

The apex of the heart was at the fifth intercostal space midway between the midclavicular line and the anterior axillary line. There was a moderate right ventricular lift. A grade III/VI apical diastolic rumble was present and there was a grade II/VI apical systolic murmur, as well as a grade IV/VI systolic murmur just to the left of the sternum at the fourth intercostal space.

The answering of questions and undressing and dressing caused pronounced dyspnea.

Right and left heart catheterization were performed 19 October and the pulmonary artery pressure was 42/22 mm of mercury, rising to 70/42 with exercise. Pulmonary wedge pressure was 25/15 mm. Right atrial pressure was 9/-3. The cardiac index was 2.3 liters per minute at rest and 3.8 liters with exercise.

Operation was carried out 18 November. Through a median sternotomy incision, grade III/VI enlargement of the right atrium was noted and there was III/VI systolic thrill in the right atrium. The left atrium was enlarged. There was a grade III/VI diastolic thrill in the left ventricle. A catheter was inserted into the right internal mammary artery to measure pressures, and another was inserted into the ascending aorta to return oxygenated blood to the patient.[7] On palpation of the tricuspid valve before circulatory by-pass was begun, there was found to be no evidence of tricuspid stenosis, but instead there was secondary tricuspid insufficiency, grade III/VI, with a valve and annulus that admitted three to three and a half fingers.

After by-pass was in effect, examination revealed calcification of the mitral valve. There was pronounced calcification of the anterior leaflet as well as the area of the posteromedial commissure. The mural leaflet was again found to be fibrotic, thickened and rigid. The valve was removed and a No. 6 Kay-Shiley disc valve* (identical diameter and cross sectional area to the Starr-Edwards No. 3 ball valve) was sutured in place with interrupted mattress sutures in such a fashion that the 3 millimeters of the remaining mitral valve covered the prosthetic device. Therefore, the disc valve was subvalvular and in the ventricle.[11]

On examination of the tricuspid valve under direct vision, no evidence of stenosis was seen but there was secondary tricuspid insufficiency. Two figure-of-eight sutures were placed in the annulus of the tricuspid valve in such a way that the posterior leaflet was almost completely obliterated. These sutures were placed in the annulus from the area of the commissure between the septal and posterior leaflet toward the commissure between the anterior and posterior leaflet. When the sutures were tied, the tricuspid annulus was narrowed to two fingerbreadths.[8]

The patient did well postoperatively and a tracheostomy was not necessary. Catheterization was performed in April 1966 (five months after operation), and the pulmonary artery pressure was 35/15 mm of mercury, rising to 50/20 with exercise. Pulmonary wedge pressure was 16/5 mm and the right atrial pressure was 5/0 mm. The cardiac index was 2.5 liters per minute at rest and 3.3 liters with exercise.

Discussion

The importance of repairing the valve rather than replacing it with a prosthetic device has been stressed in the past.[3,6,9,10] During the past eight years we have become convinced of the necessity of reconstructing valves whenever possible rather than replacing them. Starr noted an incidence of 27 emboli in 65 patients who were observed a minimum of three months after ball valve replacement of the mitral valve.[4]

Peripheral embolization is not a complication of mitral valve repair and the majority of patients with mitral insufficiency can have repair of the insufficient mitral valve with annuloplasty. The valve most amenable to correction is the one with gross dilatation and pliable leaflets, with or without torn chordae tendineae. However, even a valve with combined mitral stenosis and insufficiency can be repaired in some patients, provided there is not too much deformity or calcification. The only valves that cannot be repaired are those with moderate to heavy calcification or with severe scarring and fibrosis.

Clinically, there have been two serious problems with mitral ball valve replacement. One is the high incidence of peripheral embolization and the other is outflow obstruction from the left ventricle. Byron[1] mentioned irritation of the ventricular septum, with arrhythmia and death, as another complication.

Cross, Ferein and Jones[2] and Hufnagel and Conrad[5] have reported on other types of disc valves.

Because of our problems with the ball valve, we also developed a disc valve. It has been used experimentally in our research laboratory for the past two years and during that time it was compared with the conventional ball valve in the mitral and tricuspid area of dogs.[11] The following observations were noted in comparing these two valves. There was a lowering of the incidence of thrombosis with the disc valve compared with the ball valve. The left atrial pressure was significantly lower in the dogs with the disc valve in the mitral position. Recent studies by Morrow, Harrison and Braunwald[12] indicated that although hemodynamic abnormalities regress after use of the ball valve, a diastolic gradient that increases during exercise persists across the valve. In our experiments, it was noted that with mitral ball valve replacement there was a gradient at the outflow area of the left ventricle. This gradient was 25 mm of mercury or more in five of eight dogs. This either did not occur with the mitral disc valve or else was minimal compared with that of the ball valve.

---

*Manufactured by Shiley Laboratories, 13431 Eton Place, Santa Ana, Calif.

In our patients with ball valve replacement, we most commonly used a No. 2 or No. 3 Starr-Edwards ball valve, approximately half of the patients having the smaller size and half the larger. It was rare for a patient to have insertion of the largest (No. 4) ball valve or the smallest (No. 1). For the 14 patients with disc valve replacement (Table 1) either a No. 6 or a No. 7 was used, these sizes having the same inside diameter and cross sectional area as the No. 3 and No. 4 ball valves. Therefore, these patients had a larger valve inserted than was possible with the ball valve. Larger sized disc valves have been manufactured and will be inserted in patients with large left ventricles. The size of the valve inserted is probably important in decreasing the left atrial pressure and also in decreasing left atrial stagnation. Since the disc valve cage is only 40 per cent as long as the ball valve, the disc valve can be inserted beneath the remaining 3-millimeter cuff of the patient's valve tissue.[12] This allows for a shield of valve tissue over the disc valve, except for the 1-millimeter ring of Stellite.®* This technique cannot be used routinely for

TABLE 1.—*Data on 14 Cases in which Cardiac Valves were Replaced with Disc Valves*

	Diagnosis	*Procedure*	*Result*
1.	(a) Calcified valve with mitral insufficiency (b) Tricuspid insufficiency	(a) Replacement of mitral valve with a No. 6 disc valve (b) Tricuspid valvuloplasty	Asymptomatic
2.	Mitral stenosis and insufficiency	Mitral valve replaced with a No. 7 disc valve	Asymptomatic
3.	Mitral stenosis and insufficiency	Mitral valve replaced with a No. 6 disc valve	Asymptomatic
4.	Mitral stenosis and insufficiency	Mitral valve replaced with a No. 7 disc valve	Asymptomatic
5.	Mitral stenosis and insufficiency	Mitral valve replaced with a No. 6 disc valve	Embolus three months after surgery resulted in aphasia and hemiplegia (prothrombin time 50 per cent at that time)
6.	(a) Calcific mitral stenosis and insufficiency (b) Tricuspid insufficiency	(a) Mitral valve replaced with a No. 6 disc valve (b) Tricuspid annuloplasty	Asymptomatic (See paper for summary)
7.	Mitral stenosis and insufficiency	Mitral valve replaced with a No. 7 disc valve	Asymptomatic
8.	Calcific mitral stenosis	Mitral valve replaced with a No. 5 disc valve	Asymptomatic
9.	(a) Calcific mitral stenosis (b) Aortic stenosis and insufficiency	(a) Mitral valve replaced with a No. 6 disc valve (b) Aortic valvuloplasty	Asymptomatic
10.	Mitral stenosis	Mitral valve replaced with a No. 6 disc valve	Died in low cardiac output
11.	Mitral insufficiency	Mitral valve replaced with a No. 9 disc valve	Asymptomatic
12.	(a) Mitral stenosis and insufficiency (b) Tricuspid insufficiency	(a) Mitral valve replaced with a No. 6 disc valve (b) Tricuspid annuloplasty	Died of aspergillus endocarditis
13.	(a) Mitral stenosis (b) Tricuspid stenosis and insufficiency (c) Aortic stenosis	(a) Mitral valve replaced with a No. 6 disc valve (b) Tricuspid valve replaced with a No. 8 disc valve (c) Aortic valvuloplasty	Asymptomatic
14.	Mitral insufficiency	Mitral valve replaced with a No. 7 disc valve	Asymptomatic

---

*A cobalt base metal similar to vitalium, made by Union Carbide Corp.

ball valve substitution, since it lowers the valve into the left ventricle and therefore (with the ball valve) increases the risk of left ventricular outflow tract obstruction and causes irritation of the ventricular septum. The disc valve has been laboratory-tested for the equivalent of 35 years without any evidence of wear. However, with the ball or disc valve machined correctly, this is not an important factor as compared with moving leaflet valves, which wear out rather rapidly. The disc valve that we used has been machined in such a fashion that disc sticking is impossible.

Four of the 14 patients in whom the mitral valve was replaced with a disc valve had tricuspid valve involvement as well. In three patients the tricuspid valve was repaired but in the fourth it was necessary to replace the valve with a disc valve. In two patients there was concomitant aortic valve disease and in both of these patients the valve was repaired. There were two deaths in this group of 14 patients. In one patient, death was due to aspergillus endocarditis; in the other, to low cardiac output.

There was one peripheral embolus in these 14 patients. It occurred three months after insertion of the mitral disc valve, and the patient's prothrombin time was 50 per cent at the time of embolization rather than the desired 20 per cent.

Anticoagulant therapy should be employed in all patients with prosthetic valves.

ADDENDUM: At present 18 more patients have had mitral valve replacement with the Kay-Shiley disc valve and the results have been comparable.

REFERENCES

1. Byron, F. X.: Discussion of paper by Young, W. P., Gott, V. L., Rowe, G. D.: Open-heart surgery for mitral valve disease, with special references to a new prosthetic valve, J. Thoracic & Cardiovasc. Surg., 50:827, December 1965.

2. Cross, F. S., Ferein, A. N., and Jones, R. O.: Evaluation of two prostheses for total replacement of the mitral valve, J. Thoracic & Cardiovasc. Surg., 46:719, December 1963.

3. Egerton, W. S., and Kay, J. H.: Repair of mitral insufficiency associated with ruptured chordae tendineae, Ann. Surg., 157:351, March 1963.

4. Herr, R., Starr, A., McCord, C. W., and Wood, J. A.: Special problems following valve replacement, Ann. Thoracic Surg., 1:403, July 1965.

5. Hufnagel, C. A., and Conrad, P. W.: Comparative study of some prosthetic valves for aortic and mitral replacement, J. Thoracic & Cardiovasc. Surg., 46:719, May 1965.

6. Kay, J. H., Egerton, W. S., and Zubiate, P.: The Surgical treatment of mitral insufficiency and combined mitral stenosis and insufficiency with use of the heart-lung machine, Surgery, 50:67, July 1961.

7. Kay, J. H., Dykstra, P. C., and Tsuji, H. K.: Retrograde ilioaortic dissection—A complication of common femoral artery perfusion during open heart surgery, Am. J. Surg., 3:464, March 1966.

8. Kay, J. H., Maselli-Campagna, G., and Tsuji, H. K.: The surgical treatment of tricuspid insufficiency, Ann. Surg., 162:53, July 1965.

9. Kay, J. H., Magidson, O., and Meihaus, J. E.: The surgical treatment of mitral insufficiency and combined mitral stenosis and insufficiency using the heart-lung machine, Am. J. Cardiol., 9:300, February 1962.

10. Kay, J. H., Tsuji, H. K., and Redington, J. V.: The surgical treatment of mitral insufficiency associated with torn chordae tendineae, Ann. Thoracic Surg., 1:269, May 1965.

11. Kay, J. H., Kawashima, Y., Kagawa, Y., Tsuji, H. K., Redington, J. V.: Experimental mitral valve replacement with a new disc valve, (To be published in Ann. Thoracic Surg., 1966).

12. Morrow, A. G., Clark, W. D., Harrison, D. C., Braunwald, E.: Prosthetic replacement of mitral valve: Operative method and results of preoperative and postoperative hemodynamic assessments, Circulation, 29: (Supplement 1, p. 2), April 1964.

13. Zubiate, P., and Kay, J. H.: Tricuspid stenosis—Surgical treatment, Supplement to Circulation, 29:95, April 1964.

Dermatopathic Lymphadenopathy

A Clinicopathologic Analysis of Lymph Node Biopsy Over a Fifteen-Year Period

ROBERT A. COOPER, M.D., AND PETER J. DAWSON, M.D., *Portland, Oregon,*
AND OSCAR N. RAMBO, M.D., *San Francisco*

■ *Forty cases of dermatopathic lymphadenopathy were found in a series of 906 consecutive lymph node biopsies (4.8 per cent).*

The histologic development and progression of the disease was correlated with the clinical state of the patient.

In 35 of 40 cases the patients had active skin disease at the time of the biopsy; one of the remaining five patients had Hodgkin's disease, one had multiple myeloma and one had secondary syphilis. In the other two, no organic cause was found.

In nine cases (22.5 per cent), the histological pattern typical of dermatopathic lymphadenopathy was associated with malignant lymphoma. Except for two biopsies, which showed coexisting malignant lymphoma and dermatopathic lymphadenopathy, no histologic features were found which distinguished patients with malignant lymphoma from the remainder.

While the pathogenesis of the lymph node changes remains obscure, the histologic features suggest that it is at least in part an immune response, although the nature of the responsible antigen is unknown.

LIPO-MELANOTIC RETICULOSIS,* although first described by Wise,[16] was established by the work of Pautrier and Woringer.[13,14] Over the years reports of several series of cases† have been published as well as reports of individual cases.

While the entity is now well known, three problems remain: (1) Is this histologic pattern found only in association with skin disease; (2) what is the pathogenesis of the lymph node changes; and (3) what is the relationship to malignant lymphoma. In the present report, dermatopathic lymph nodes selected from a large series of consecutive biopsies were studied in an effort to resolve these problems.

Materials and Methods

All diagnostic lymph node biopsies, exclusive of those containing metastatic tumor or included with radical surgical specimens, obtained at the University of California Medical School for the period 1943-1957 inclusive were reviewed. In addition to lymph nodes with the generally recognized microscopic features of dermatopathic lymphadenopathy, a careful search was made for possible early

---

From the Departments of Pathology, University of Oregon Medical School, Portland, Oregon, and the University of California School of Medicine, San Francisco, California.

Submitted 24 June 1966.

Reprint requests to: Department of Pathology, University of Oregon Medical School, 3181 S.W. Sam Jackson Park Road, Portland, Oregon 97201 (Dr. Cooper).

*Called also *dermatopathic lymphadenitis* or, better, since it is not an inflammation, *dermatopathic lymphadenopathy.*

†Reference Nos. 1, 5, 6, 7, 8, 11, 12.

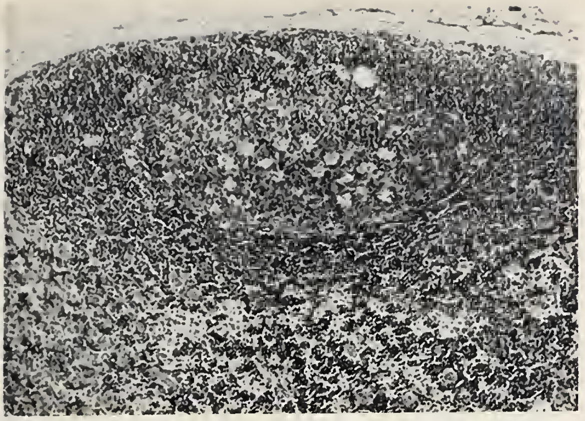


Figure 1.—Early dermatopathic lymphadenopathy. The cortical sinuses just deep to a germinal follicle contain small aggregates of pale histiocytes. Pigment, not evident at this magnification, was present in both cortical sinus and germinal center histiocytes. Hematoxylin and eosin, ×150.

lesions characterized by focal histiocytic aggregates and lipid or pigment deposits.

Formalin, Zenker's or Bouin's fixed blocks were recut and stained with hematoxylin-eosin, with para-aminosalycilic acid (PAS) before and after digestion, by Sudan black B for lipid, by Lillie's silver oxide method for reticulin, by the Masson silver and Schmorl ferric-ferricyanide methods for melanin and by the Prussian blue method for iron. The slides were studied separately by each author; and, without knowledge of the histologic results, clinical data were abstracted from each patient's chart. Thirty-three skin biopsies from 27 patients were also reviewed. Only after the microscopic and clinical analyses had been completed were the results compared and clinicopathologic correlations made.

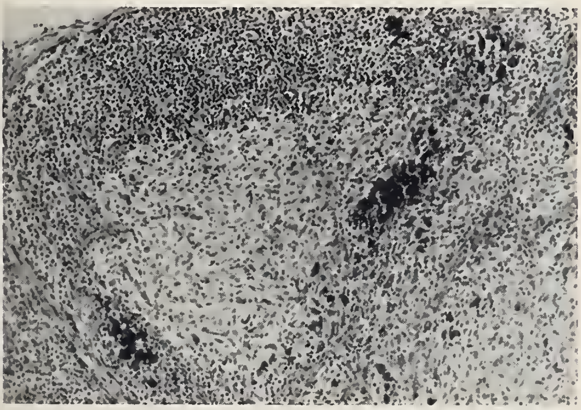


Figure 2.—Dermatopathic lymphadenopathy, intermediate stage. The central portion of a tertiary cortical nodule is replaced by an accumulation of histiocytes. Abundant melanin pigment is present at the borders of the nodule. Hematoxylin and eosin, ×150.

Results

Of 906 lymph node biopsies examined, 44 showed changes of dermatopathic lymphadenopathy, an incidence of 4.8 per cent in a series from which nodes containing metastatic tumor or forming part of radical procedures had been excluded. Two cases were discarded because of lack of clinical information. This left 42 biopsies from 40 patients, which formed the basis of this study.

Males were affected nearly twice as often as females. Although occurring at any age, the condition was most common in the fifth and sixth decades. Clinically, the axillary nodes were most commonly affected (in 27 of 40 patients), generally in combination with either the cervical or inguinal nodes. In three cases the affected nodes were not enlarged clinically, but in each of these cases the nodes drained an area of diseased skin.

The clinical diagnoses are given in Table 1. Thirty-five patients had skin lesions at the time of

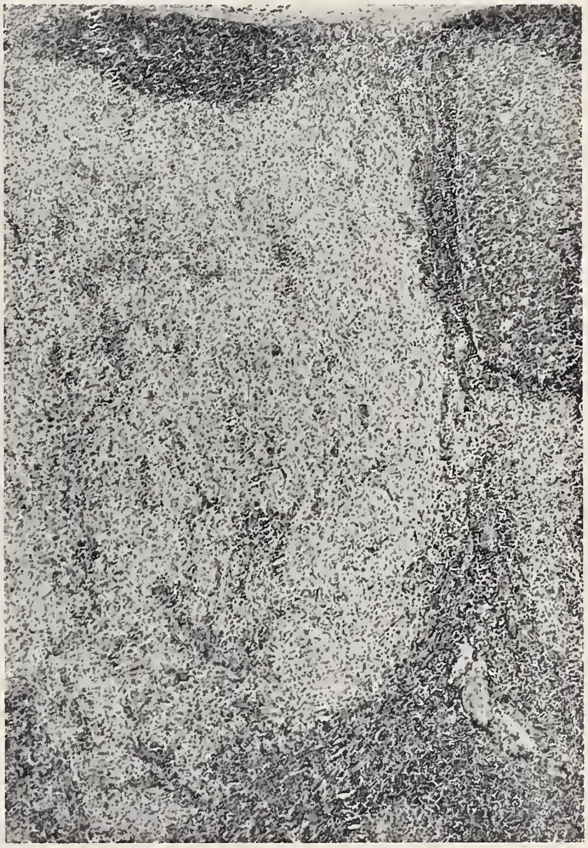


Figure 3.—Dermatopathic lymphadenopathy, advanced stage. A tertiary cortical nodule is extensively replaced by histiocytes with only a small subcapsular lymphocytic zone remaining. Note the circumscribed character of the histiocytic infiltrate and its abrupt termination at the medulla. The germinal follicle belongs to a second tertiary nodule. Hematoxylin and eosin, ×60.

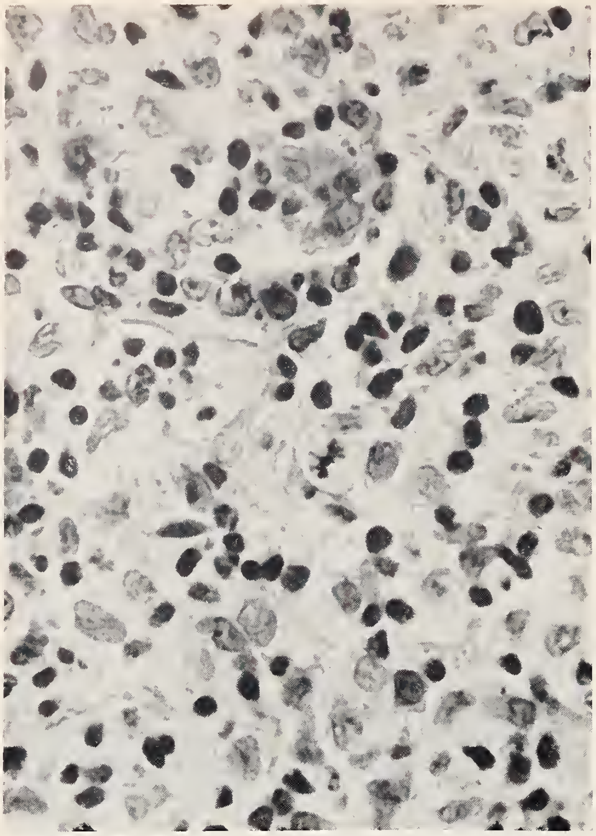


Figure 4.—Portion of a cortical nodule. The histiocytic cells have pale vesicular nuclei and moderately abundant, weakly acidophil cytoplasm. Note the apparent decrease in cortical lymphocytes and the histiocytic mitosis. Hematoxylin and eosin, ×825.

lymph node biopsy. A generalized rash was present in 26 patients and localized skin lesions in nine. Of the five patients without skin disease, three had systemic disease, namely Hodgkin's disease, multiple myeloma and secondary syphilis. Of the remainder, one had a severe prolonged anxiety neurosis, and the other severe pruritus of undetermined cause. Thus, 12.5 per cent of the patients did not have clinical skin disease. Pruritus was present in only 30 cases. On at least one occasion the absolute eosinophil count was significantly raised in 14 patients. Nine of 40 biopsies (22.5 per cent) came from patients with malignant lymphoma or mycosis fungoides.

Grossly, the lymph nodes removed at biopsy varied in diameter from 0.7 to 5.0 cm and were generally soft and tan, although five nodes contained black pigment. Microscopically, the characteristic feature was the presence in the cortex of nodular aggregates of histiocytes with pale staining cytoplasm, vesicular nuclei and light eosinophilic nucleoli (Figures 1, 2 and 3). These nodules overshadowed the presence of moderate numbers of cortical germinal centers.

Small numbers of normal mitoses were seen in the cortical histiocytes in 23 biopsies, and they were fairly numerous in two. In 12 biopsies these histiocytic aggregates occupied more than three-fourths of the total cortical area, while in seven the involved area was very much smaller and accounted for less than one-quarter of the cortex. Admixed with the histiocytes were small numbers of lymphocytes (Figure 4).

Eosinophils were present in 27 biopsies and were moderately numerous in 10. Reticulin stains showed that although the nodal architecture was essentially normal, there was dilatation of the cortical sinuses with a resultant apparent diminution in the overall density of reticulin fibers.

The medulla showed striking changes. The medullary sinuses were dilated and populated by approximately equal numbers of lymphocytes and histiocytes; a few of the latter contained phagocytized debris. The medullary cords were the site of a moderate or pronounced plasmacytic infiltrate (Figure 5) accompanied by the formation of Russell bodies. This plasmacytic response appeared to be an integral part of the histologic picture and was absent from only three biopsies, two from infants and one from a patient with Hodgkin's disease. In 36 biopsies stained with hematoxylin and eosin, golden-brown, 1-micron pigment granules were found, characteristically around the edges of the nodular histiocytic aggregates. However, in sections stained with Masson's silver stain, pigment was present in every biopsy. Perls' stain showed that this pigment did not contain iron, although variable amounts of hemosiderin were present in six biopsies, principally in the medullary sinus his-

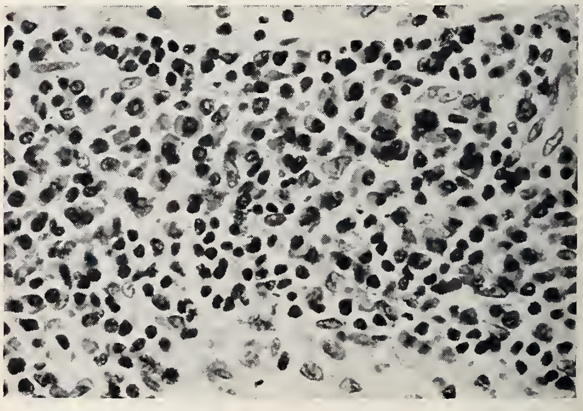


Figure 5.—Medulla in dermatopathic lymphadenopathy. An intersinusoidal medullary cord is distended with large numbers of plasma cells. Hematoxylin and eosin, ×600.

tiocytes. Collections of large histiocytes with coarsely vacuolated cytoplasms (foam cells) were present in 19 biopsies and were characteristically situated at the periphery of the histiocytic nodules (Figure 6). Staining with Sudan black B revealed phospholipid in 37 biopsies.

From histologic comparison of the whole series, the earliest change appeared to be the accumulation of large pale histiocytes at the centers of the tertiary nodules between the germinal centers, which themselves gradually enlarged (Figure 1). Small amounts of pigment (best seen with Masson's silver stain) were present at the corticomedullary junction and within germinal centers. Enlargement of the histiocytic nodules followed, due in part at least to local mitotic activity, and was associated with progressive accumulation of pigment and increase in size and number of the germinal follicles (Figure 2).

With the advent of well established follicular activity, there was concomitant vigorous plasmacytic response within the medullary cords. In advanced cases, the cortex was largely replaced by the histiocytic aggregates (Figure 3).

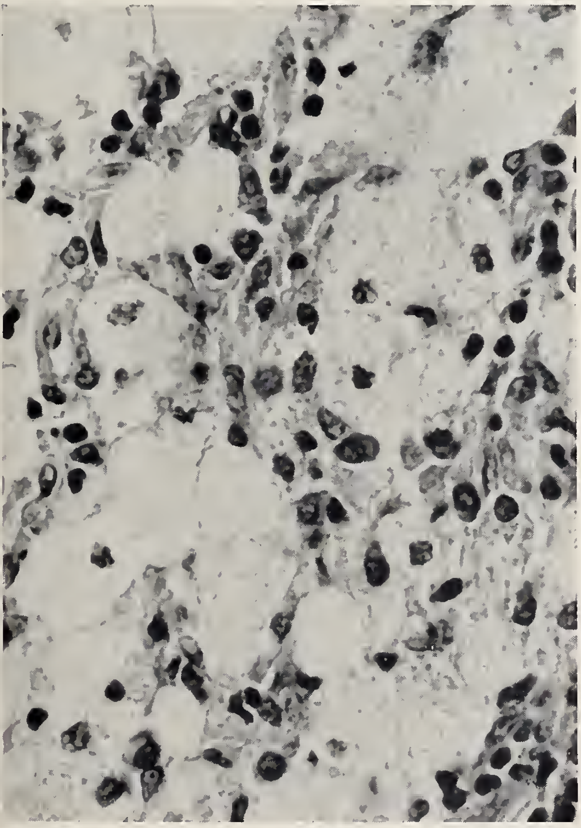


Figure 6.—Collection of lipid-laden histiocytes located in sinuses at the corticomedullary junction. Hematoxylin and eosin, ×825.

While there was clinical evidence for regression of the lymphadenopathic condition, we cannot be sure of the ultimate sequence of histologic events. Presumably the histiocytes gradually find their way into the medullary sinuses and efferent lymphatics, and the plasmacytic response in the medullary cords gradually subsides. We have never seen scarring even in the most advanced examples of dermatopathic lymphadenopathy; hence we believe that clinical regression is associated with pathologic regression and a return to a nearly normal histologic state.

Because dermatopathic lymphadenopathy was associated with malignant lymphoma in nearly 25 per cent of cases, a special attempt was made to find histologic features that might distinguish these cases from the remainder. Except in two biopsies where the changes of lymphosarcoma and Hodgkin's disease were seen coexisting in the same or adjacent nodes, no such distinguishing features could be found, even in retrospect.

Discussion

Of the many who published reports on the subject, Hurwitt[5] and Möller[11] were the only observers to attempt to establish the specificity of the association of the histologic features in the lymph node with skin disease. In both series, comprising 12 and nine patients respectively, this association was constant. In the present larger series, however, there were five exceptions. Pautrier and Woringer[13,14] and many other investigators expressed belief that pruritis was the common denominator, but pruritus was absent in one-quarter of our series.

While all the earlier reports stressed the importance of not only the histiocytic accumulations but also the presence of melanin and lipid, it was only the former that was a consistent histologic feature. In six of the biopsies in the series here reported, melanin was present in amounts too small to be seen without special stains, and its absence in otherwise typical cases has also been reported by Laymon and Jackson.[8] It is important to note that small amounts of melanin may be found in otherwise normal nodes.[3,7] Foam cells (lipid histiocytes) were not present in 23 biopsies, and lipid stainable with Sudan black B was not present in five.

In contrast, the plasmacytopoiesis which was present in all but three of the biopsies in the series has excited little comment in the literature. The proliferation of plasma cells in the medullary cords and the enlargement of the secondary (germinal) follicles seen in dermatopathic lymphadenopathy closely resembles that described in lymph nodes actively engaged in antibody production.[9,15] Further, histologic features resembling those of dermatopathic lymphadenopathy have been produced in guinea pigs by 1,2,4-dinitrochlorbenzine,[10] which is a potent allergen. Similar histologic features may also be produced in rats by subcutaneous or intraperitoneal injection of melanin from the choroid of cows.[4]

These results, coupled with the histologic features, suggest that the changes in dermatopathic lymphadenopathy may be at least partially on an allergic basis. For the present, the nature of the antigen and its source remain unknown. This does not, however, explain the only constant histologic feature, namely, histiocytic proliferation and aggregation. It is possible that another factor besides the antigen is responsible for the accumulation of histiocytes, although it might also be the result of other properties of the antigen.

TABLE 1.—*Clinical Diagnosis in 40 Patients with Dermatopathic Lymphadenopathy*

MALIGNANT LYMPHOMA (including mycosis fungoides)	Mycosis fungoides	5
	Probably mycosis fungoides	1*
	Hodgkin's disease	1†
	Lymphosarcoma	1
	Multiple myeloma	1†
		9
RETICULOSES	Histiocytosis X	1
DERMATOSES	Exfoliative dermatitis	11
	Atopic dermatitis	3
	Exfoliative psoriasis	2
	Dermatitis herpetiformis	1
	Polymorphous light eruption	1
	Seborrheic dermatitis	1
	Pyoderma	1
	Angioneurotic edema	1
	Secondary lues	1†
	Nonspecific chronic dermatitis	3
		25
COLLAGEN DISEASES	Dermatomyositis	1
	Polyarteritis nodosa	1
	Idiopathic thrombocytopenic purpura	1
		3
IDIOPATHIC	Idiopathic pruritus	1†
	Anxiety state	1†
		2

---

*Skin biopsy consistent with mycosis fungoides, but patient lost to follow-up.

†No skin lesions.

Save for the two cases where evidence of malignant lymphoma was present elsewhere in the same biopsy, either in the involved node or in one adjacent to it, we could not tell microscopically which nodes were associated with malignant lymphoma elsewhere. The association of dermatopathic lymphadenopathy with mycosis fungoides has been known from the descriptions of Pautrier and Woringer.[13,14] Certainly, there is no microscopic evidence to support either the view of Bluefarb and Webster[2] that dermatopathic lymphadenopathy is a form of follicular lymphoma, or the belief expressed by Nairn and Anderson[12] that direct transition to malignant lymphoma occurs in involved nodes. Rather it seems that mycosis fungoides and other dermal lymphomas are potent initiators of the chain of events that results in the reaction in the regional nodes that is characteristic of dermatologic lymphadenopathy. Nevertheless this association is well established, and in any case in which these lymph node features are observed, the patient should be carefully examined for signs of a malignant lymphoma elsewhere.

Little has been written on the subsequent course of events in nodes affected by dermatopathic lymphadenopathy. Indirect evidence suggests that virtually complete resolution occurs if the inciting cause is removed. Focal involvement in seven of the 42 biopsies in the present series suggests that the disease may exist without clinical lymph node enlargement. Hence, dermatopathic lymphadenopathy may be much more prevalent than published reports would indicate.

REFERENCES

1. Auger, C., Grandbois, J., and Gaumond, E.: Dermatopathic lipomelanotic reticular cell hyperplasia of lymph nodes, Canad. Med. Assn. J., 66:245-248, 1952.

2. Bluefarb, S. M., and Webster, J. R.: Lipomelanotic reticulosis. Report of two cases in which the condition developed into mycosis fungoides, Arch. Derm. (Chicago), 61:830-841, 1950.

3. Gail, D.: Über das Vorkommen von Melanin and Hämosiderin in peripheren Lymphknoten, Frankfurt Z. Path., 68:64-70, 1957.

4. Hohenadl, L., and dePaola, D.: Über Gewebsreaktionen nach parenteraler Zufuhr von Melanin unter besonderer Berücksichtigung der lipomelanotischen Reticulose, Frankfurt Z. Path., 69:374-383, 1958.

5. Hurwitt, E.: Dermatopathic lymphadenitis. Focal granulomatous lymphadenitis associated with chronic generalized skin disorders, J. Invest. Derm., 5:197-204, 1942.

6. Jarrett, A., and Kellett, H. S.: The association of generalized erythrodermia with superficial lymphadenopathy (lipomelanic reticulosis), Brit. J. Derm., 63:343-362, 1951.

7. Laipply, T. C.: Lipomelanotic reticular hyperplasia of lymph nodes. Report of 6 cases, Arch. Intern. Med., 81:19-36, 1948.

8. Laymon, C. W., and Jackson, R.: Lipomelanic reticulosis, A.M.A. Arch. Derm. (Chicago), 71:303-312, 1955.

9. Leduc, E. H., Coons, A. H., and Connolly, J. M.: Studies on antibody production. II. The primary and secondary responses in the popliteal lymph node of the rabbit, J. Exp. Med., 102:61-72, 1955.

10. Macher, E.: Die Reaktion der regionären Lymphknoten beim tierexperimentellen allergishen Kontaktekzem, Hautartz, 13:18-23, 126-131, 174-179, 1962.

11. Möller, O.: Dermatopathic lymphadenitis, Acta Path. Microbiol. Scand., 28:352-365, 1951.

12. Nairn, R. C., and Anderson, T. E.: Erythrodermia with lipomelanic reticulum-cell hyperplasia of lymph nodes (dermatopathic lymphadenitis), Brit. Med. J., 1: 820-824, 1955.

13. Pautrier, L-M., and Woringer, Fr.: Note préliminaire sur un tableau histologique particulier de lésions ganglionnaires accompagnant des éruptions dermatologiques généralisées, prurigineuses, de types cliniques différents, Bull. Soc. Franc. Derm. Syph., 39:947-955, 1932.

14. Pautrier, L-M., and Woringer, Fr.: A propos d'un aspect histopathologique nouveau du ganglion lymphatique: La réticulose lipomélanique accompagnant certaines dermatoses généralisées, les échanges entre la peau et le ganglion, Ann. Derm. Syph. (Paris), 8 (Series 7): 257-273, 1937.

15. Ringertz, N., and Adamson, C. A.: The lymphnode response to various antigens: An experimental-morphological study, Acta Path. Microbiol. Scand. Suppl., 86:1-69, 1951.

16. Wise, F.: Lymphadenosis cutis universalis, associated with generalized erythrodermia and atrophy of the skin, J. Cut. Dis., 35:669-704, 1917.

The Changing Role of County Hospitals in California

LESTER BRESLOW, M.D., *Berkeley*

WE ARE CELEBRATING TODAY a compact between the County of San Diego and the University of California, San Diego—a compact designed to promote better health care and to enhance medical education. The goals of the County and of the University in this endeavor represent important interests of the people both in the County and in the State. For undertaking it, therefore, you are to be congratulated by all of us.

On the part of the University, the assumption of responsibility for direction of services in the County Hospital is another step toward assuring adequate resources for education of physicians and other health personnel needed in the State. Perhaps even more important, it signifies once again the intent of the University to link itself with the community, to carry out its responsibility for education and research through helping to meet community needs for service. This commitment to public service will improve the quality as well as add to the quantity of physicians and others who will gain their education here.

The compact we are celebrating today will enhance the quality of medical education because teachers and students will be participating in the transformation of a major county hospital into an institution for a new and higher type of health service. San Diego has reason to be proud of the facility it has developed here and the history of its service to people in this County.

But a new day has come to the county hospital in California. This State has again taken the lead in providing for the conversion of county hospitals into community hospitals. About a century ago, the State placed upon the counties in California the responsibility for care of the indigent sick. This responsibility the counties met by building separate hospitals for the poor. Over the years, many of these public hospitals, as in San Diego, have developed a high technical level of health care and have maintained an important resource for graduate medical education. However, it was still segregated care. It carried an element of compulsion because persons who sought care there could not obtain it elsewhere.

Our national policy now calls for desegregation, in health as well as in educational and other endeavors. California is the first major state to extend this policy to include breaking down the segregation of health care based on economic as well as on racial and other grounds. The California Medical Assistance Program, initiated 1 March 1966, provides a new opportunity for most of the poor in this State, although not all of them, who were formerly obliged to seek health care at county hospitals. This is the opportunity to obtain their care from physicians and in hospitals of their own choosing, with the bills to be paid by the State. In this way, the counties are relieved of a big share of the obligation to provide care of the indigent sick. Poor patients who historically filled the county hospitals may henceforth enter any hospital in the community.

The desegregation of health care provided in the California Medical Assistance Program goes both ways: The new law specifically encourages counties, at the option of the Board of Supervisors, to open county hospitals to all patients in the community. These hospitals need no longer restrict their care to the indigent. This has been spoken of as the conversion of county hospitals into community hospitals.

Remarks at Ceremony Inaugurating Operation of the San Diego County-University Hospital, by the University of California, San Diego, 6 July 1966.

The author is Director of the California State Department of Public Health.

Submitted 7 September 1966.

Reprint requests to: Director of Public Health, State Department of Public Health, 2151 Berkeley Way, Berkeley 94704.

In the future, then, county hospitals will begin to serve the entire community and admit patients of all social and economic strata. Many indigent patients are already entering other hospitals in the community; the county hospital will cease being "the hospital for the poor"; patients who pay their own way or whose care is charged to labor-management health and welfare funds, or to the Social Security Fund or other sources for payment will enter the county hospital. Segregated health care for the poor will be a matter of history and any social stigma attaching to the county hospital will vanish.

For the immediate future, the county hospital will still carry responsibility for the care of those who are both poor and doubly unfortunate in not falling into one of the categorical aid groups. For example, adult persons who are poor, but who have no children and are not over 65 years of age or blind or otherwise disabled, are not eligible for the California Medical Assistance Program benefits. If in need of health care, they must continue to obtain it at the county hospital. This limited group will require only a relatively small share of the county hospital services, and it is to be hoped that further social advances will soon enable even these persons to secure health care of their own choice.

In their new role of serving the entire community, the county hospitals in California will continue to offer opportunities for graduate medical education, that is, the training of medical specialists. Several will maintain or will newly establish links with medical schools, as in San Diego, and thus be a resource for undergraduate medical education as well. This will tend to improve even further the quality of care. Every physician knows that one of the most noteworthy signs of excellence in medicine is participation in medical education. County hospitals generally in this State have achieved eminence in this regard and they will surely cling to the superior status and many advantages which affiliation with medical education affords.

However, one major change will occur in the relationship of county hospitals to medical education, and this will be a change for the better. Patients will no longer be known as or treated as "clinical material." Probably nothing in the training of physicians in this country so typifies its greatest defect as the term "clinical material." As used by medical educators it has often implied that patients, and especially patients whose care served as a basis for physician training, were mere biological specimens available for teaching. It is not remarkable that many young physicians who spent years in such an atmosphere acquired attitudes of disrespect which were later resented by patients.

Henceforth patients in California, even those who are poor, will not have to present themselves as "clinical material." Medical educators and graduate and undergraduate medical students, who have not already done so, will rapidly learn that the patient is first of all a human being to be treated with dignity. And people will learn that one reliable sign of good health care is its connection with medical education. Just as physicians know this truth, people generally will learn it. This learning will necessarily be gradual because long-established ideas connecting medical teaching with care of the poor must be overcome. But such prejudices do melt with change in circumstances.

Thus one element in the future of the county hospital will be maintenance of excellence through a relationship to medical education, with patients freely choosing to obtain their care in such a situation.

In addition, county hospitals as centers of high-quality care will serve as the foci for new advances in medical science. Take just one example: Medicine appears to be on the verge of tremendous developments in the transplantation of human tissues and organs, and artificial substitutes for them. Success has already been achieved with blood, arteries, cornea and kidney. How can such achievements be made more generally available to people, and what will be next? County hospitals will be in the midst of the advances involved in answering these questions.

In addition to being involved in these exciting technological developments, county hospitals affiliated with medical schools will have an even more challenging opportunity. That is the opportunity to serve as the base for truly comprehensive care. One of the strongest and among the most justifiable complaints against modern medical care is the complaint against fragmentation. Nowadays, care tends to be episodic rather than continuous. The individual with pain or some other symptom may obtain first-rate diagnostic and therapeutic care for the particular episode of illness. But that is not enough. What is needed, and increasingly expected, is a sense of responsibility by the physician and the rest of the health care team for continuing health care of the individual.

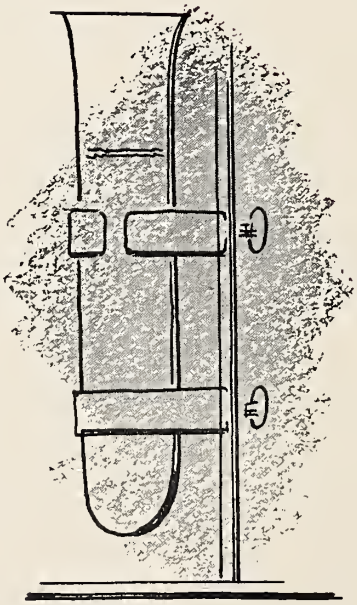
The training of physicians in the recent past has emphasized accurate diagnosis and treatment, but without much regard for continuity of care. Pediatrics has been somewhat of an exception. The future of medicine, with acute illness being relatively well controlled, lies more in the direction of personal health care with the aim of assuring health rather than merely treating disease. To accomplish such a change of direction will require substantial innovation in medical education.

One approach is to have the medical faculty (the staff of a county hospital) and its students, assume total responsibility for health care of a specified population group—perhaps 20,000 to 30,000 persons. Every element of health care for all persons in this population would be provided—at home, in office or clinic, in hospital or other facility. A county hospital serving as the teaching facility for a medical school could offer the entire gamut of services that modern medical science has made available, from minor, routine care to the most complex and difficult. But the important factor is that a new commitment would be established, a commitment to preserve and extend health, not merely to treat disease. For the population being served, the occurrence of a case of measles, now that an effective vaccine is available, or a death from cancer of the cervix, now that means are at hand to avoid such death, or indeed the occurrence of any other disease event that can be prevented, would be regarded properly as a shame. This sense of responsibility for health of an entire population would tend to stimulate in young physicians an attitude of health maintenance going beyond readiness to undertake diagnosis and treatment. Experience in such a situation would yield vastly different physicians from those of the last generation trained in crowded clinics and at the bedside of acutely ill patients only.

Still another implication of the conversion of county hospitals into community hospitals is that the county hospitals may join on an equal footing other hospitals planning for and establishing a national community-wide hospital service system.

Thus county hospitals in California are entering upon the new day in medical care with greater opportunities than ever before. I believe that they will continue to maintain a position of leadership in quality of care and in the training of physicians. Further, they can help to reintroduce both a humanistic attitude and a health orientation which medical education has been lacking during the past few decades.

The San Diego County Hospital by virtue of its history, its first-rate staff, its physical facilities and now its affiliation with the University of California, San Diego Medical School may be a leader in this development.



The Compensable Injury

ALLEN J. ENELOW, M.D., *Los Angeles*

■ *The complexities and interferences in the doctor-patient relationship during the treatment of the compensably-injured workman create an unusually high incidence of delayed recovery, complications, and permanent disability. Many persons, once injured, never return to economic self-sufficiency. Among the factors that may have a bearing in producing this situation are: The psychological context of the injury itself; the personality of the patient; the "reverse incentive" created by reward for illness; third-party interference from attorneys, insurance claims adjusters, and union representatives; consequent hostility of the patient toward a doctor he usually has not chosen; and, too often, hostility of the physician toward his patient. Iatrogenic complications may also be present when ill-advised surgical procedures are used in the presence of psychological complications.*

To improve the situation the adversary system should be removed and outside interferences in treatment should be reduced. Physicians treating patients in such circumstances should learn more about psychiatric diagnosis and management. They should give more attention to remotivation and rehabilitation.

A PATIENT WITH AN accidental injury which makes him eligible for compensation enters a strange and complicated world unlike the corresponding environs of patients of any other type. Attorneys, claims agents and even labor union representatives and judges may have an influential role in the diagnosis and the treatment of his condition. The motivation of the patient is significantly altered and in most instances the attitude of the physician toward the patient is significantly different than toward the average patient. Small wonder, then, that in such cases there is an inordinately high incidence of complications, of delayed recovery and of permanent disability. The aim of this paper will be to examine some of the complexities of the compensable injury problem that create these complications.

In 1963 Nemiah[2] wrote:

> "For most people the enforced dependency of a disabling physical disorder causes no serious difficulty. They are able to accept the dependency and help from other people as a necessary means to recovery, and when the functioning of their physical bodies has returned to its fullest possible capacity, they easily give up their dependent state and return to their habitual adult behavior of activity and self-reliance. There is, however, a group of people who have major emotional conflicts centered around their dependency needs."

Nemiah went on to say that these patients have a great tendency to develop an exaggerated and prolonged pattern of dependency, centered around

---

Presented before a Joint Meeting of the Sections on Physical Medicine and Industrial Medicine and Surgery at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: Department of Psychiatry, USC School of Medicine, 2025 Zonal Avenue, Los Angeles 90033 (Dr. Enelow).

physical complaints, that prevents their return to activity and productive work even when the physical functioning of their bodies has been restored. These are the patients whose disabilities and complaints far exceed the limits one would expect from the findings on physical and laboratory examination. "They appear unduly incapacitated and dependent, make excessive demands on doctors, nurses and family for the smallest service, and become angry, critical and demanding when their wants are not fulfilled." Very often these patients are characterized by a tendency to see themselves as strong, active and self-reliant; transferring the responsibility for their incapacity to the offending organ or the symptoms. These are patients who have had conflicts between wishing to be independent and self-sufficient on the one hand and wishing to be cared for and looked after, on the other; and who have tried to resolve these conflicts by over-reaction in the direction of hard work in an effort to maintain a feeling of independence, an uneasy and unstable solution that has now broken down.

Hirschfeld and Behan,[1] writing in 1963 on "The Accident Process," said that before most accidents occur there is a state of conflict and anxiety within the patient. They studied a number of patients injured on the job in the automotive industry, and came to the conclusion that this conflict and anxiety produces a significantly large number of accidents in the following way: As a result of his emotional condition, the worker finds a self-destructive, injury-producing act, following which he then becomes engaged in going from doctor to doctor for help, thus avoiding the true sources of his original conflict. In other words, an unacceptable problem has been converted into a problem that is acceptable to the patient by virtue of his injury. Hirschfeld and Behan concluded that most accidents are a single event in a continuing dynamic process. The beginning of this process is the state of conflict, tension or anxiety before the event, followed by the catastrophic event itself, and then the new way of life as a patient with an escape from the old conflicts. They found a number of instances in which skilled workers in a state of emotional turmoil made mistakes which a novice would never make. Their conclusion was that the chief difficulty in helping these patients stems from these facts: (1) They are solving life problems through their symptoms, and (2) the symptoms guarantee a legal incapacity which will provide for their continued support.

There appear to be five classes of persons in whom psychiatric complications are most likely to develop when they incur an injury in which compensation is involved. These are:

1. *The hysteric.* There are persons who use avoidance and denial techniques to modify painful reality and who try to block conscious recognition of unacceptable feelings, of impulses and of conflicts, with variable success. Although they maintain some reasonable participation in interpersonal activities, they are incapable of great emotional depth in their relationships with others, making special demands on others in their relationships. They may appear to be warm and responsive, but this warmth is usually found to be superficial and insincere when examined more closely as these patients are quite egocentric and manipulative in their relationships with other people. They are people who make great demands on others and use their symptoms to control the lives of the people upon whom they feel dependent. These are the patients who are most likely to have conversion reactions with non-anatomical paralyses or anesthesias or with excessively widespread disabilities after rather minimal to moderate injuries.

2. *The dependent and immature.* There are persons in their adult years who have never "grown up." Dependent on others and virtually incapable of accepting the responsibility for themselves, they avoid situations which impose responsibility upon them. Their relationships are generally characterized by a clinging quality and by the fact that they openly seek, and tend to find, others who will "take care of them." They often have a history of repeated job changes and of rather brief periods of time spent at any one job. Often they do not marry but continue to remain in the parental home well into adulthood. If they are married, it is usually the spouse who is the strong member of the family. These are persons who, once injured, will settle for a relatively low level of economic subsistence, the injury having provided a concrete "physical" reason to explain their total dependency and lack of ability to take responsibility for themselves.

3. *The over-conscientious hard worker struggling with conflicts over independence and self-sufficiency.* Persons of this order can be considered to have always been one step ahead of their wishes to be dependent and to be cared for by others; who were incapable of accepting such behavior on their own part, and, therefore, worked overly hard

and overly long, resenting it all the while. Once they are injured, they become very much like persons of the type described in Group 2, while protesting that they are active and self-reliant but are prevented from behaving that way by their physical disability.

4. *The depressed patient*. Depression has a twofold effect in increasing the proneness to injury. For one thing, depressed patients often have nearly-conscious or conscious desires to injure themselves, even to kill themselves. They will often quasi-consciously injure themselves in the grip of such self-destructive impulses. Other qualities of the depressed patient are decreased vigilance, attention turned away from the world and inward on the self, reduced psychomotor activity and diminished coordination—all of which make them exceptionally prone to accidents in situations where vigilance and coordination are required. With these patients, the depression is a serious complicating factor in rehabilitation and must be attended to if rehabilitation is to be successful.

5. *The sociopathic exploiter*. The sociopathic person is one who has been unable to assimilate a consistent set of ethical values. His major values are self-seeking ones, and society is viewed as an adversary to be circumvented. The "sociopath" may conform to regulations and laws for much of his life, the responses based on a short range view of immediate consequences rather than any stable set of internal values. When such a person is in a position to manipulate others to his own advantage, as in the instance of an industrial injury, the possibility of economic gain becomes a powerful motive to use dishonest means and to misrepresent his degree of disability. It is in this group that malingering is most often seen. Such persons may appear to have a certain charm, especially if they feel that it is to their advantage to display it. Examination of their history will show a consistent inability to manage relationships with authority figures, frequent job changes, few or no lasting relationships and a history of "using" others to their own advantage.

A complicating factor in every case of industrial injury is the fact that there is an element of reward for illness which militates against the patient's giving up the sick role. In addition, in our litigation-conscious society, the industrially injured patient will frequently consult an attorney before or very soon after, he consults his physician. Thus, almost from the outset, there is a more complicated physician-patient relationship with third party interference than in any other kind of illness. This does not increase the physician's motivation to work with this patient. Some physicians work with such patients only out of a feeling of obligation, which makes them less interested in their industrial injury patients than in any other patient they have. Some physicians feel a great deal of hostility toward the industrial accident patient, and behave as though all such patients were all malingerers and exploiters. For one thing, the care of these patients is considerably less rewarding financially; they are more difficult to treat; and the prognosis is always poorer than with the private patient.

This hostility is matched by the hostility of the patient who does not trust the physician who is paid by the insurance company rather than by him. Each then reacts to the resentment and hostility of the other, which effectively poisons the physician-patient relationship.[3]

The contest atmosphere further complicates the relationship, with both patient and physician being cautious, defensive, suspicious of each other and frequently having their relationship interrupted by court order or by unexpected changes in examining physician arranged by the insurance company or demanded by the patient's attorney.

Finally, the iatrogenic complications themselves complete the compound. Many a patient whose injury has physically cleared up but who for various psychological reasons continues to feel pain and suffers disability, is given braces, or even has surgical procedures performed on him to produce "stability" in a given joint, with a further fixing of symptoms; even developing new disabilities as a result of the surgical procedures. The end result is a medical and psychiatric chaos unlike anything else in medicine.

There is an increasing body of opinion that favors a change in our present system of handling, diagnosing, treating and compensating patients with industrial injuries. It is difficult to see how psychiatric and psychological complications of the compensable injury, as they now occur, can be reduced in frequency until the following changes are brought about:

- Removal of the adversary or contest system from the management of industrial injuries.

- An increased responsibility of the physician toward his patient and an assumption of his proper role as the doctor in charge of the care of his patient, without interference from outside parties.

- An increased awareness on the part of the physician of the psychological factors that predispose to the occurrence of injuries and to the psychiatric complications of injuries. This would include the ability to diagnose and differentiate the hysterical patient from the one with dependency conflicts and to be able to recognize the depressed patient and the "sociopath."

- An increased attention of the physician toward *remotivation,* using the support inherent in the physician-patient relationship to reestablish the patient's desire to care for himself and to reassume his personal and social responsibilities.

REFERENCES

1. Hirschfeld, A. H., and Behan, R. C.: The accident process, J.A.M.A., 186:193-196, 19 October 1963.

2. Nemiah, J. C.: Psychological complications in industrial injuries, Arch. Environ. Health, 7:481-486, October 1963.

3. Stephens, L. L., M.D.: Personal communication. Many of the ideas in this paper were developed and refined during communication with Doctor Stephens who contributed materially to the concepts and formulations contained in it.

Electro-Oculograms in the Early Diagnosis of Chloroquine Retinopathy

MARVIN S. WEINREB, M.D., *Hayward*

■ *Funduscopy, electro-oculography and electroretinography are all valuable in early detection of chloroquine retinopathy, which is reversible if detected early. Simplified instrumentation for electro-oculography was utilized in testing 12 normal controls, one patient with diabetic retinopathy and 15 patients with potential or actual cases of chloroquine retinopathy. Normal controls, and all but one of the patients without clinical evidence of retinopathy, had electro-oculographic ratios above 180. All patients having evidence of retinopathy had ratios below 180.*

IN A RECENT REVIEW of the dermatologic indications for chloroquine and the reactions to the drug Rees and Maibach[36] found the drug to be of definite value in four dermatologic diseases: Discoid lupus erythematosus, desoxyribonucleic acid autosensitivity reactions, polymorphous light eruption and some instances of solar urticaria. They listed 26 other dermatologic conditions in which further verification was necessary. Rheumatologists have used antimalarial drugs in the treatment of certain cases of rheumatoid arthritis, with varying amounts of success.

Although animal studies by Fitzhugh, Nelson and Holland[20] demonstrated chronic toxicity in rats at high dosage levels, only transient, mild toxic effects were found by Alving and coworkers[1] in humans receiving relatively smaller amounts. Millions of doses were administered for malaria suppression and for treatment of the acute disease, with no serious side effects noted. Such side effects as lichenoid skin eruptions, difficulties in ocular accommodation to light, headaches and bleaching of hair were found to be reversible.

The usual dose of chloroquine administered in endemic areas to suppress malaria is 250 mg weekly. A dose of 1,500 mg given in three days is used for acute attacks. The range of dosage given for dermatologic diseases is much higher, going often to 500 mg daily for long periods. Chloroquine keratopathy was found to occur at these higher dosage levels[24]; the condition was reversible and it could be detected before loss in visual acuity occurred. Cambiaggi,[12] in 1957, reported the case of a 37-year-old Negro man with systemic lupus erythematosus in whom narrow, irregular retinal arterioles were noted after chloroquine and hydroxychloroquine therapy, and was followed by macular atrophy surrounded by a zone of pigmentation associated with constricted visual fields and diminished visual acuity. Subsequently, additional case reports of retinopathy caused by chloroquine (Aralen®), hydroxychloroquine (Plaquenil®), and amodiaquine (Camoquine®) were published.* In some of the cases visual acuity diminished progressively in spite of withdrawal of the antimalarial drug.

Henkind and Rothfield[23] studied a group of 45 patients who were receiving chloroquine therapy and found that 18 per cent of them had some de-

---

From the Division of Dermatology, University of California School of Medicine, San Francisco.

Presented before the Section on Dermatology at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: 2457 Grove Way, Hayward 94546.

*Reference Nos. 11, 18, 19, 32, 34, 35, 38, 40, 41, 42.

gree of retinopathy and 44 per cent of those who had been taking the drug for more than three years had retinopathy. Such reports caused Rothermich[37] to sound a note of caution.

Metabolism of Chloroquine

Zvaifler and Rubin[45] utilized spectrophotofluorometric analysis for chloroquine and its N-desethylated metabolite in normal subjects, in patients with rheumatoid arthritis and in patients with non-rheumatoid arthritis. They demonstrated that maximum plasma levels developed much more rapidly in the patients with rheumatoid arthritis than in the other two groups. The groups differed in their excretion patterns, but almost all of the drug administered was accounted for by urinary excretion. Measurable amounts of the drug were present in the urine three and a half years after the last dose had been taken. Studies in rabbits and rats by Bernstein and coworkers[10] showed a rapid and preferential storage of chloroquine in the iris and choroid of pigmented animals. Absence of the drug in all ocular tissues of albino animals was demonstrated. Zvaifler, Bernstein, and Rubin[44] found that in pigmented and albino rats given 250 mg of chloroquine weekly for six or more months, the concentration of chloroquine in ocular tissues of the pigmented animals was equal to or greater than the concentration in any other organ. The concentration of the drug paralleled the degree of pigmentation of the ocular tissue; the iris and choroid had the greatest concentration, the cornea, retina and sclera had less, and the vitreous and lens had none. In the ocular tissues of albino rats, only the retina contained chloroquine. Dale, Parkhill, and Layton[17] noted similar results in rabbits.

More recently, Cohen and Yielding[14] demonstrated the ability of chloroquine to form a complex with DNA and suppress the activity of DNA polymerase. Perez, Mansour, and Zvaifler[33] expressed belief that the polymeric melanin pigment has the physiochemical characteristics of a cation exchange resin and exists as a stable free radical. Both properties are involved in chloroquine binding.

Chloroquine Retinopathy

It was soon realized that the clinical appearance and progression of chloroquine retinopathy differed from that of the other ocular pigment dyscrasias and retinopathy. Okun and coworkers[31] and Henkind, Carr, and Siegel[22] stated that the early features of chloroquine retinopathy included a pericentral scotoma, macular and perimacular pigmentary changes and granularity, often giving a bull's-eye appearance. Later changes consisted of constricted peripheral fields, pronounced pigmentation and depigmentation changes, narrowing of retinal vessels, diminution of visual acuity and changes in physiological tests of retinal function.

Pathology

There is fairly general agreement that late changes consist of irregular degeneration of rod and cone elements with migrations of pigment to the inner and outer nuclear and plexiform layers.[9,10,43]

The most successful animal studies reported to date were performed by Meier-Ruge[29] on cats. The cats were given chloroquine by mouth in amounts of 1.5 to 6.0 mg per kilogram of body weight daily for four to seven weeks, and clinically observable fundus changes developed. From examination of paraffin sections and results of histochemical reaction tests, it was observed that the primary toxic action of chloroquine was on the pigment epithelium, which reacted with extensive enlargement. Later, focal detachments of the retina and desquamations of the pigment epithelium were seen, and finally atrophy of the rods and cones occurred.

Electro-Oculogram Studies

Movement of the eyeballs results in variation of the indirectly measured corneo-fundal standing potential of the eye. When the positively charged cornea is closer to an electrode placed at one canthus, the negatively charged retina is closer to an electrode placed at the other canthus of the same eye. This potential difference can be recorded and can be found to change in amount and polarity as the eye is rotated. A test, the electro-oculogram (EOG), used initially almost exclusively to record eye movements, was thus developed.[25,27]

Arden and his associates, in an extensive series of experiments,[3-8] determined that the potential measured was a function of the metabolism of the pigment epithelium, and that the normal, dark-adapted eye manifested a significant rise in standing potential after illumination. Pigmentary disturbances, including chloroquine retinopathy and other retinal diseases, reduced or eliminated this response. They found considerable variation in the actual voltage present in the eyes tested, but the ratio between the lowest potential reached in the darkened room and the highest potential reached after light stimulation was determined to be the best criterion of retinal function. It was expressed as $\frac{\text{light peak}}{\text{dark trough}} \times 100$. They found that all normal persons had ratios exceeding 185—most of them more than 200. Arden and Fojas[5] found that patients with chloroquine retinopathy had greatly diminished electro-oculogram voltage.

Arden, Friedmann and Kolb,[6] in studying six patients taking chloroquine over periods averaging 18 months, found slight depression of EOG ratio. In one patient the ratio fell from 230 to 165 in six months while taking chloroquine. One month later it was 145. Chloroquine was withdrawn and the ratio was back to 175 in five weeks and back to normal limits in three months. On retrospect he stated that he had noted some blurring of vision just before discontinuing the drug. No changes on fundoscopy were ever noted.

Gouras and Gunkel,[21] performing electro-oculograpy in eight patients with chloroquine retinopathy, found that in five who had mild cases, the ratio was usually within normal limits while the other three, who had severe retinopathy, always had abnormally low ratios. However, in all eight cases the patients had not taken any of the drug for several months, leading the investigators to believe that the test is more sensitive when patients tested are still taking chloroquine. Henkind, Carr and Siegel[22] did follow-up studies of 39 patients in whom retinopathy was demonstrated during chloroquine therapy. They also studied 25 patients who were receiving chloroquine but in whom retinopathy was not present. No single test could detect the earliest cases. Their data suggest that changes in visual acuity, visual fields, color vision and dark adaptation occur relatively late. Funduscopy, electroretinograms and electro-oculograms appeared to give the earliest indications of retinopathy. Continued safe utilization of this useful class of drugs seemed possible after the reversibility of changes was demonstrated in early cases.[16,30] Experiments in electro-oculography, using a simplified clinical apparatus, were suggested to me by Arden.[2] In previous work, complicated and expensive laboratory equipment had been used, with the exception of one case[15] in which tests were done with a modified electrocardiograph machine.

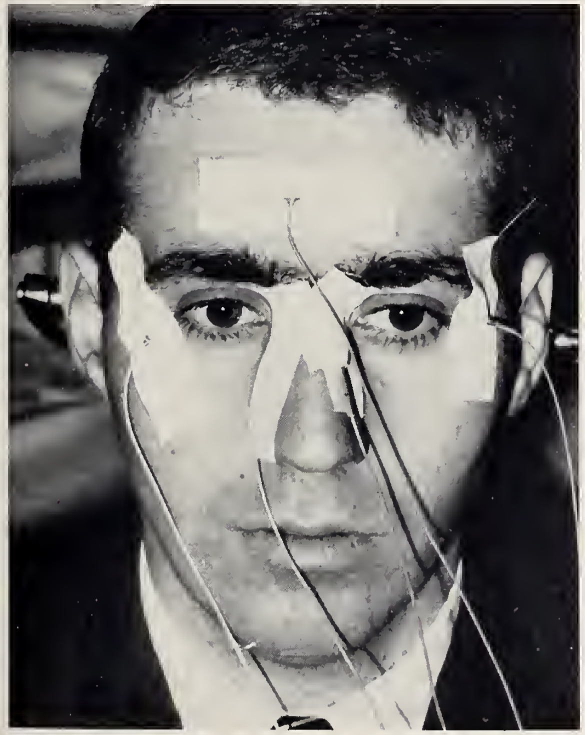


Figure 1.—Electrodes adhesive taped in position for electro-oculogram (EOG) recording.

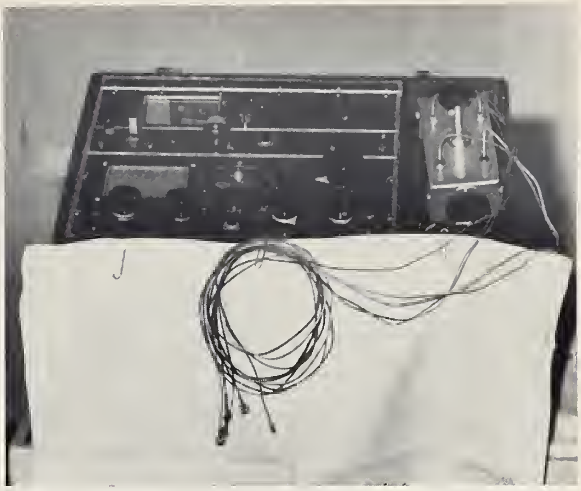


Figure 2.—In front of the modified electrocardiograph are the five leads and electrodes. Junction box with switch on top is visible resting on ECG input cable.

Methods and Materials

Two x-ray view boxes were mounted side by side on the wall with one 7½-watt red fixation light on each side of the combined unit. To increase the intensity of the light source, each box was rewired to accommodate four 15-watt fluorescent tubes instead of the usual two tubes. Separate switches controlled each box and each fixation light. The subject was seated comfortably in a chair equipped with a neck rest. He faced the light source and rotated his eyes comfortably between the fixation lights. A 37° angle was used in these experiments. An older portable electrocardiograph unit (Cardiotron Model PC-2) was modified to reduce the paper speed to 5.0 mm per second. Grass E-1A Durable Disc silver electrodes were chlorided to eliminate polarization and were applied to each canthus with adhesive tape. An indifferent electrode was applied to the subject's forehead (Figure 1). Cambridge electrode jelly at each site assured good contact. The five leads were routed through a fused junction box to the Lead 1 input of the electrocardiograph machine (Figure 2). A switch on the junction box permitted selection of the output of either eye to be amplified and recorded.

The electrodes were attached under normal room illumination and the subject was instructed to look as rapidly as possible from one fixation light to the other and back. Thus, the skin contacts and equipment were tested. Then the room was completely darkened except for the fixation lights,

TABLE 1—*Summary of Clinical Data*

Case	*Sex*	*Age (Years)*	*Disease*	*Drug*	*Total Dose (gm)*	*Time Since Last Dose*	*Retinal Findings*	*EOG Ratio (Average)*
Control	M	20	....	....	....	....	Normal	219
Control	M	27	....	....	....	....	Normal	213
Control	M	29	....	....	....	....	Normal	320
Control	M	37	....	....	....	....	Normal	200
Control	M	41	....	....	....	....	Normal	209
Control	F	18	....	....	....	....	Normal	220
Control	F	27	....	....	....	....	Normal	229
Control	F	20	....	....	....	....	Normal	208
Control	M	23	....	....	....	....	Normal	222
Control	M	21	....	....	....	....	Normal	206
Control	M	35	....	....	....	....	Normal	185
Control	M	22	....	....	....	....	Normal	199
Control	M	51	Diabetic Retinopathy	....	....	....	Grade Z changes with macular degeneration	165
1.	M	36	CDLE	....	....	....	Grade Z changes with macular degeneration	256
2.	F	40	....	....	....	....	Grade Z changes with macular degeneration	211
3.	M	35	CDLE	hydroxychloroquine	22	0	Normal	250
4.	F	50	Scleroderma	chloroquine	52	0	Normal	181
5.	M	38	SLE	hydroxychloroquine	72	0	Normal	211
6.	F	47	SLE	chloroquine	*	3 months	mild pigmentary changes; problem not due to drugs	193
7.	F	53	CDLE	chloroquine	139	3 years	mild bull's-eye macular changes	175
8.	F	36	SLE	chloroquine	255	0	slight mottling of macula	194
				chloroquine	275	0	slight mottling of macula	205
9.	F	42	CDLE	chloroquine	360	3 months	Normal	215
10.	F	59	CDLE	amodiaquine	267	18 months	Normal	159
11.	F	44	Rheumatoid arthritis	chloroquine	*	4 months	early bull's-eye macula	168
12.	F	35	Polymorphic light eruption	chloroquine	710	6 months	Normal	274
13.	F	15	SLE	chloroquine	913	3 months	"haziness" of right macula	130
				chloroquine	913	5 months	"haziness" of right macula	173
14.	F	52	CDLE	amodiaquine	79	....	Normal	206
				amodiaquine	22	....	Normal	......
				hydroxychloroquine	1,500	0	Normal	......

---

*Unknown quantity.

EOG=Electro-oculogram; CDLE=Chronic discoid erythematosus; SLE=Systemic lupus erythematosus.

and a record from each eye was taken every minute. The dark trough was usually reached before 10 minutes passed but some eyes required more time. After 12 minutes the light source was turned on and recording continued for another 12 minutes. The test was then complete and the electrodes were removed from the subject. Measurement of the height of several excursions in each minute's tracing permitted calculation of the ratio $\frac{\text{light peak}}{\text{dark trough}} \times 100$.

The results of these experiments are summarized in Table 1. Ratios of 180 and over appear to be in the normal range. With the exception of Case No. 10, ratios below 180 seem to indicate disease. The presence of subungual pigmentation due to amodiaquine in Case No. 10 may be significant in this regard. Also, Maguire and Kolb[26] showed that treatment with amodiaquine for only one month caused significant reductions in electro-oculogram ratios.

In Case No. 13, early chloroquine retinopathy seemed to be developing and the patient was referred for testing at the time the drug was discontinued. An average ratio of 130 at that time correlated well with funduscopic findings. Two months later her average ratio had increased to 173. No progression of macular changes was seen, and visual acuity remained good. Analysis of the results obtained in the other cases suggests that only rough correlation exists between the amount of visible retinopathy and the degree of depression of the electro-oculogram ratio.

Discussion

Chloroquine retinopathy has been shown to be reversible in its early stages. Recent informal requests as to present frequency of development of new cases indicated a pronounced decline. Physicians are prescribing shorter courses of the antimalarial drugs at lower dosage levels (Scherbel and coworkers[39]) and are observing patients more closely for side effects.

In addition to funduscopy, electro-oculography has been shown to be of definite aid in predicting retinopathy. The clinical test as described here can be performed in non-academic institutions at relatively low expense with no sacrifice in utility.

Future developments in this general field are indicated by a recent report: McChesney, Banks and Wiland[28] found that ascorbic acid intake by guinea pigs significantly decreased tissue retention of chloroquine. Perez and coworkers[33] reported that although British anti-lewisite (BAL) and ammonium chloride administration increased excretion of chloroquine in rabbits, it did not remove the drug from choroid or iris. However, chlorpromazine pretreatment did interfere with the ocular deposition of the drug. These investigators also demonstrated sharply decreased chloroquine binding to melanin after pretreatment with ascorbic acid.

Carr, Gouras and Gunkel[13] recently developed what they believe to be a more sensitive test of retinal function for use in predicting chloroquine retinopathy. Perimetric thresholds to red light become elevated after chloroquine doses of one tablet daily in total intakes exceeding 100 gm. Slow reduction in thresholds followed discontinuance of the drug.

GENERIC AND TRADE NAMES OF DRUGS

Chloroquine—*Aralen.*
Hydroxychloroquine—*Plaquenil.*
Amodiaquine—*Camoquine.*

REFERENCES

1. Alving, A., Eichelberger, L. C., Craig, B. Jr., Jones, R. Jr., Whorton, C. M., and Pullman, T. N.: Studies on the chronic toxicity of chloroquine (SN-7618), J. Clin. Invest., 27:60-65, 1948.

2. Arden, Geoffrey B., Personal Communication.

3. Arden, G. B., and Barrada, A.: Analysis of the electro-oculograms of a series of normal subjects, Brit. J. Ophth., 46:468, 1962.

4. Arden, G. B., Barrada, A., and Kelsey, J. H.: New clinical test of retinal function based upon the standing potential of the eye, Brit. J. Ophth., 46:449, 1962.

5. Arden, G. B., and Fojas, M. R.: Electrophysiological abnormalities in pigmentary degenerations of the retina, A.M.A. Arch. Ophth., 68:369, September 1962.

6. Arden, G. B., Friedmann, A., and Kolb, H.: Anticipation of chloroquine retinopathy, Lancet, 1:1164-65, 2 June 1962.

7. Arden, G. B., and Kelsey, J. H.: Some observations on the relationship between the standing potential of the human eye and the bleaching and regeneration of visual purple, J. Physiol. (London), 161:205-26, 1962.

8. Arden, G. B., and Kelsey, J. H.: Changes produced by light in the standing potential of the human eye, J. Physiol. (London), 161:189-203, 1962.

9. Bernstein, H. N., and Ginsberg, J.: The pathology of chloroquine retinopathy, A.M.A. Ophth., 71:238-245, February 1964.

10. Bernstein, H. N., Zvaifler, N., Rubin, M., and Mansour, Sister, A. M.: The ocular deposition of chloroquine, Invest. Ophth., 2:384, August 1963.

11. Bregeat, P., Grupper, C., Juge, P., Coscas, G., and Dorlencourt, M.: Ocular complications due to synthetic antipaludians, Arch. Ophth. (Paris), 25:417-44, 1965 (in French).

12. Cambiaggi, A.: Unusual ocular lesions in a case of systemic lupus erythematosus, A.M.A. Arch. Ophth., 57: 451, March 1957.

13. Carr, R. E., Gouras, P., and Gunkel, R. D.: Chloroquine retinopathy . . . Early detection by retinal threshold test, A.M.A. Arch. Ophth., 75:171-78, 1966.

14. Cohen, S. N., and Yielding, K. L.: Further studies on the mechanism of action of chloroquine . . . Inhibition of DNA and RNA polymerase reactions (Paper presented at Am. Rheumatism Assn. Meeting, San Francisco, Calif., 1964).

15. Copeman, P. W. M., Cowell, T. K., and Dallas, N. L.: Screening test for chloroquine retinopathy, Lancet, 1:1369-70, 20 June 1964.

16. Crews, S. J.: Chloroquine retinopathy with recovery in early stages, Lancet, 2:436-38, 29 August 1964.

17. Dale, A. J. D., Parkhill, E. M., and Layton, D. D.: Studies on chloroquine retinopathy in rabbits, J.A.M.A., 193:141-43, 1965.

18. Elliott, J. H., and Mills, J. B.: Chloroquine retinopathy, J. Oklahoma M. A., 56:391, August 1963.

19. Ellsworth, R. J., and Zeller, R. W.: Chloroquine (Aralen)-induced retinal damage, A.M.A. Arch. Ophth., 66:145-48, 1961.

20. Fitzhugh, O. G., Nelson, A. A., and Holland, O. L.: The chronic oral toxicity of chloroquine, J. Pharmacol. and Exp. Therap., 93:147, 1948.

21. Gouras, P., and Gunkel, R. D.: The EOG in chloroquine and other retinopathies, A.M.A. Arch. Ophth., 70: 91-101, November 1963.

22. Henkind, P., Carr, R. E., and Siegel, I. M.: Early chloroquine retinopathy: Clinical and functional findings, A.M.A. Arch. Ophth., 71:157-65, February 1964.

23. Henkind, P., and Rothfield, N. F.: Ocular abnormalities in patients treated with synthetic antimalarial drugs, New Engl. J. Med., 269:433, 29 August 1963.

24. Hobbs, H. E., and Calnan, C. B.: Visual disturbances with antimalarial drugs with particular reference to chloroquine keratopathy, A.M.A. Arch. Derm., 80:557, 1959.

25. Kris, C.: Electro-oculography; From Medical Physics, Vol. 3, Edited by Otto Glasser, The Year Book Publishers, Chicago, pp. 692-700, 1960.

26. Maguire, A., and Kolb, H.: The effect of a synthetic antimalarial (Amodiaquine) on the retina, Brit. J. Derm., 76:471-74, November 1964.

27. Marg, E.: Development of electro-oculography, A.M.A. Arch. Ophth., 45:169-85, 1951.

28. McChesney, E. W., Banks, W. F., Jr., and Wiland, J.: Effect of ascorbic acid on tissue deposition of chloroquine in the guinea pig, Proc. Soc. Exp. Biol. & Med., 119:740-42, 1965.

29. Meier-Ruge, William: Experimental investigation of the morphogenesis of chloroquine retinopathy, A.M.A. Arch. Ophth., 73:540, 1965.

30. Nozik, R. A., Weinstock, F. J., and Vignos, P. J., Jr.: Ocular complications of chloroquine . . . A series and case presentation with a simple method for early detection of retinopathy, Am. J. Ophth., 58:774-78, November 1964.

31. Okun, B., Gouras, P., Bernstein, H., and Von Sallmann, L.: Chloroquine retinopathy, A.M.A. Arch. Ophth., 69:59, January 1963.

32. Penner, R., and Sommers, K.: Bilateral macular degeneration associated with chloroquine therapy, Am. J. Ophth., 54:381, September 1962.

33. Perez, R., Mansour, Sister, A. M., and Zvaifler, N.: Chloroquine binding to melanin—Characteristics and significance, (Paper presented at Am. Rheumatism Assn. meeting, San Francisco, Calif., 1964).

34. Price, Lennox: Symposium: Retinopathy due to chloroquine, retinopathy associated with chloroquine, Trans. Ophth. Soc. Aust., 21:35, 1961.

35. Rebello, D. J. A.: Ocular reactions to antimalarial drugs, A.M.A. Arch. Derm., 83:785, 1961.

36. Rees, R. B., and Maibach, H.: Chloroquine, A.M.A. Arch. Derm., 88:96-105, September 1963.

37. Rothermich, N.: Coming catastrophies with chloroquine? Editorial, Ann. Int. Med., 61:1203, 1964.

38. Sachs, D. D., Hogan, M. J., and Engleman, E. P.: Retinopathy induced by chronic administration of chloroquine phosphate, (Paper presented at Am. Rheumatism Assn. Meeting, Chicago, Ill., 21 & 22 June 1962).

39. Scherbel, A. L., Mackenzie, A. H., Nousek, J. E., and Atdjian, M.: Ocular lesions in rheumatoid arthritis and related disorders with particular reference to retinopathy, New Engl. J. Med., 273:360-366, 1965.

40. Smith, J. L.: Chloroquine macular degeneration, A.M.A. Arch. Ophth., 68:186-190, 1962.

41. Sternberg, T. H., and Laden, E.: Discoid lupus erythematosus: Bilateral macular degeneration due to chloroquine, A.M.A. Arch. Derm., 79:116, 1959.

42. Tamler, Edward: Ocular complications of chloroquine therapy, Calif. Med., 103:30, July 1965.

43. Wetterholm, D. H., and Winter, F. C.: Histopathology of chloroquine retinal toxicity, A.M.A. Arch. Ophth., 71:82-87, January 1964.

44. Zvaifler, N. J., Bernstein, H., and Rubin, M.: Chloroquine deposition in ocular tissues, Arth. & Rheumat., 5:667, 1962.

45. Zvaifler, N. J., and Rubin, M.: The metabolism of chloroquine, Arth. & Rheumat., 5:330, 1962.

Community Psychiatry and The Population Explosion

AMES FISCHER, M.D., *San Francisco*

■ *Although excessive population has long been a dilemma in certain areas of the world, current rates of growth present a global threat to all nations. Arguments based on economics, world peace, individual freedom, humanism and the preservation of life's amenities all point to the urgent need for programs to control birth rates. This applies to the United States also, although we have the advantage of a relatively high cultural and economic level from which to attack the problem.*

Modern psychiatry is increasingly committed to the Community Mental Health concept, which in part implies greater involvement with the element of the population that is socially and economically at a disadvantage. Effective treatment cannot ignore the effects on the patient of intolerable reality situations, often produced by successive unwanted pregnancies. In addition, a positive approach to family planning by psychiatrists and social workers will provide the structure needed for truly preventive psychiatry; as our knowledge of the determinants of psychosis, delinquency and mental retardation expands, selective counseling can do much to prevent these family and social tragedies.

Both as world citizens and as practitioners of a profession, psychiatrists cannot afford to delay pledging their resources to the solution of so compelling a problem.

TODAY'S PSYCHIATRIST, like the rest of humankind, faces a problem unique in the history of our species. Uncontrolled population growth, a subject hardly discussed a decade ago, has become a preoccupation of thoughtful men. What was once the occasional outcry of a prophet is now a clamor. No day passes that the subject is not apparent in the mass media, and only the most ignorant or indifferent among us can remain unstirred.*

Professor Revelle[32] of Harvard is one who presents the challenge:

"Bringing down rates of population growth to a manageably low level will require far more knowledge and experience than we now possess. Economic, sociological, medical and educational research on a large scale and a wide front are urgently required. The problem may well be the most difficult mankind has ever faced, for its solution lies in controlling one of the basic drives of all living things—to reproduce."

Part of the uniqueness of this new peril is that to appreciate it requires the reversal of an ancient attitude. What has always been a good must now

---

From the Department of Psychiatry University of California School of Medicine and The Langley Porter Neuropsychiatric Institute, San Francisco.

Submitted 1 August 1966.

Reprint requests to: The Langley Porter Neuropsychiatric Institute, 401 Parnassus Avenue, San Francisco 94122.

*Reference Nos. 15, 19, 20, 34, 35.

become an evil. For eons instinct and circumstance coincided in the struggle to replenish the earth. War, famine and pestilence have always been viewed as scourges whose remedy was increased fecundity, and until modern times this was perfectly true. Suddenly we are faced with evidence that this is wrong and that we now have to fight both our drives and our history. And we have very little time; the former leisurely accretion of people on a vast planet has given way to a geometric progression whose urgency brings future and present together and demands action now.

The Global Background

For many years Americans have been accustomed to hearing of India's burgeoning masses and the teeming congestion of Asian cities. To most of us these facts were deplorable, and although a world problem, not new and not close. Following World War II the concept of a shrinking and interdependent world gained reality. Our foreign aid programs seemed an answer—feed the starving and alleviate misery. The fallacy eventually became apparent in areas of gross overpopulation, high birth rate and sub-subsistence, such as Asia and Latin America. Here the extra infusion of food simply allowed a population increase in rough proportion to its sustaining power; the misery was the same for each but shared by more, and the overall situation was worse because there were even more people.[11] In contrast, Europe was an already industrialized region without overpopulation, so our aid promoted prosperity and self-sufficiency.

Accordingly, another solution suggested itself: Exert every effort to help underdeveloped nations advance themselves; modern agricultural methods and machinery are clearly capable of producing enormous increases in land yields, and irrigation and power can make wastelands bloom. Once their food problems have been controlled, these nations may industrialize and eventually produce the goods and services which enable people to live rather than to exist.

This sanguine prospect is, at best, nearly vitiated by a number of flaws, and contemporary conditions of population growth destroy it utterly. Consider that world population has risen from 1¼ billion in 1900 to 2½ billion in 1950, and that the year 2000 will see nearly 7 billion if nothing is done. The sheer acceleration defeats any program of production expansion.[10,14]

To illustrate, let us take the economic issue. For an area vastly to increase its food production requires that it acquire vast amounts of capital goods—machinery, dams, power plants, irrigation systems and the like. But capital is formed only by what is left over after the subsistence needs of the population have been met. Now, in 1967, population is growing faster than world food production. And the rate of growth is such that it outweighs the capital-donating potential of rich areas (for example, the U.S.A.). Even if it did not, the problem is far more complex: Where would the expert manpower come from? Is there time to build the projects needed? How fast can backward peoples be educated and ancient prejudices overcome?

The solution lies only in drastic curtailment of population growth. There are two conceivable ways to accomplish this—by a program of mass extermination (for example, atomic holocaust, mandatory death age) or by far-reaching birth control programs. The results of conception control would "buy time" in which to meet the severe tests of the next 50 years.[4]

Compelling as they are, the economic arguments by no means constitute the case for population control; there are moral issues which cannot be ignored. As conditions are, having a child that one cannot support amounts to an imposition on the rest of society and compounds the problem. There is also a moral imperative toward the unborn child—the right not to be born. When giving birth condemns the child to a life of want and misery, it is indeed "the supreme human selfishness."[36]

Moreover, population pressures are central to political unrest and war; but the intensity of these pressures in the 20th century far exceeds what has ever before been experienced. In an atomic world, these implications arrest the imagination.[21]

Excessive growth also threatens individual freedom by leading to over-centralization of power and over-organization. As Aldous Huxley[24] states it:

"Whenever the economic life of a nation becomes precarious, the central government is forced to assume additional responsibilities for the general welfare. It must work out elaborate plans for dealing with a critical situation; it must impose even greater restrictions upon the activities of its subjects; and if, as is very likely, worsening economic conditions result in political unrest, or open rebellion, the central government must intervene to preserve public order and its own authority."

Some have tried to present a more optimistic view of the future, basing their arguments on the general premise that "science will find a way." When they are examined, however, one finds that these predictions center on the problem of food production, and this is their shortcoming.[7] Even if huge strides should be made in algae-proteins, hydroponics, sea-farming or other techniques, so that all might be fed, the other problems would still exist, with a miserable, strife-torn, regimented world in prospect.

Science *has* found a way, and is working on better ones. The "pill" and the intrauterine devices have some applicability to huge, poor and technologically primitive populations. The idea of vaccination against pregnancy hints at what may be forthcoming. Every valid (non-theological) argument urges the establishment of widespread, emphatic programs of control. Our government finally stopped evading the issue in 1965, when President Johnson stated, "I will seek new ways to use our knowledge to help deal with the explosion in world population. . . ."

There is much to be done and little time to do it.

What the U.S. Faces

Many Americans who accept the need for population control in foreign nations do not agree that we must have a similar program. The U.S., it is argued, is the richest nation in history; our standard of living is the highest ever known, and no one starves. Furthermore, we have but 6 per cent of the world's people, and our yearly increment at most represents only a 6 per cent contribution to the total explosion. This line of reasoning does not bear scrutiny; the fact that our people do not face the same problems as the Asian peasant is not to say that we face no problems. Fortunately, we have different problems, but they are nonetheless real, and they must be approached on their own terms. In addition, our very opulence creates a moral issue on a global scale. Our use of the earth's nonrenewable resources is so high that what we consume makes up one-half of the annual consumption of all the peoples of the earth, and our population increase then really accounts for 50 per cent of the problem.[12] Many of these resources are brought from underdeveloped areas; in the long run, this means that these regions will not have them at a time when they might achieve their own industrialization. Nehru of India warned, "It is simply not possible for small oases of prosperity to continue to exist amidst vast deserts of poverty without engendering storms that might engulf those oases."

Although our population expands no faster than the world rate, we will be able to "afford" this in the foreseeable future only at a price. We do not accept the goals of merely accommodating and feeding too many people, nor are we prepared to sacrifice many of our present amenities. If we are to have 350 million United States citizens by 2000 A.D., we too are faced with an acceleration problem which is already hurting us and which will only become more severe.[13]

At this moment we are appalled by the pollution of our air and water; new classrooms cannot be built fast enough; our crime rate and our juvenile problems make us feel that new dimensions have been added to old dilemmas; freeways are overcrowded from the day they are opened; recreation areas and rural acres are swallowed by suburban slums; everything we want to do or to enjoy becomes more difficult and expensive.[8] City planners foresee the megalopolis of the near future, a city of huge area, super-slums and an exaggeration of all the urban problems that at present plague us.[16] Today, for example, many experts feel that Greater New York is in an impossible situation; the middle to upper income taxpayers flee to the suburbs, the welfare rolls mount, taxes go steeply up, crime is rampant and transportation and utility systems are strained. Birth rates are highest among those with the lowest incomes, and the cycle is perpetuated under increased momentum. Welfare clients, one-sixteenth of the City's population, account for about half of the yearly births. The massing of the disadvantaged extends a social culture of faceless, discouraged persons who are nonresponsible, resentful and neglected.[33]

The velocity of these changes engenders a pervasive hopelessness or cynicism among those who day on day must come to grips with their results. Social welfare workers, police, teachers and planners increasingly see their efforts washed away by the tide. By the time a new program or expansion can be brought into operation, it is already inadequate.

There is a brighter side: The United States has ample resources to meet the problem if it has the wit and determination to do so. But we do not have unlimited time to launch our programs. It was only a few months ago that the *first* public discussion of family planning ever held under Federal auspices took place (and the event unfortunately represents progress alloyed with shortsightedness).

Family Planning and Community Mental Health

Having been powerfully encouraged by the late President Kennedy, the community psychiatry movement has recently achieved a remarkable prominence in professional thinking. The March 1966 issue of the *American Journal of Psychiatry* contained a special section with 10 articles on a wide range of issues related to the objectives of Community Mental Health.[1] Since this reflects the official position of the profession, one may ask how family planning is handled. The answer is that it is not. In fact, the Medical Director of the American Psychiatric Association stated:

"The APA has no policy statement on birth control or planned parenthood. It is surprising to me that there has been so little expression by our members on problems of such far-reaching importance."[2]

Such a state of affairs implies curious paradoxes. For example, a number of recent papers in psychiatric journals have developed the theme of reaching the poor and culturally deprived. Suggestions have been made for modifying traditional therapy —making it briefer, more directive, more responsive to the expressed complaints, geared to the argot and comprehension of the client and offering more tangible help than "just talk."[3,37] Some authors seem to scold the psychiatrist for assuming that the root of the patient's problems lies mainly in his intrapsychic conflicts rather than in a harsh reality situation that should be corrected first.[27] Yet these authors avoid explicit mention of the wretchedness born of poverty, overcrowding and successive unwanted pregnancies. Nowhere is there a definition of the therapist's responsibilities with regard to planned parenthood. This situation raises a most fundamental paradox: The universal goal of all psychiatric therapy is to work with patients so as to free them to make more rational and effective choices for themselves. It is a fact that the poorer and less educated people are ignorant of family planning and unable to afford it; naturally, it is they who produce the unwanted children. For the therapist to remain uninvolved with this issue makes him a party to constricting the patient's freedom of choice.

There must be many reasons why psychiatry has not already dealt with these problems.[18] Most obvious are those arising from the recent history of psychiatric practice. Office therapy, particularly private practice, concerned itself with the better educated and more affluent persons, who were capable of handling family planning on their own. The sicker and more deprived patients tended to be concentrated in the protected subsociety of a state hospital, often for long periods. Added to this were the putative virtues and status of being a non-directive therapist, a bias eagerly adopted by many social workers as well. These influences fitted with the notion that, although recognized as a national or world problem, fertility control was not really a psychiatric problem; even if it should have been considered important to the mental health of some patients, it was easy to assume that "someone else" would and should deal with it.

The profession is preparing itself for a new and broadened psychiatric milieu, now loosely described by the term "social psychiatry." This coming professional world will be more complex and diverse than the one we know. Already much thought has been devoted to analyzing and developing it, and these efforts have been commendable for both quality and scope. In the following recommendations perhaps a further dimension can be introduced into comprehensive psychiatric care.

A Program for Psychiatry

Agreement on a Policy

Psychiatry should no longer remain officially silent on family planning, and what is developed should be much more than a statement of good intentions. A policy formulation should begin with taking a position on the world and United States problems, but should move from there to a much more explicit delineation of the contributions of a social psychiatric program. This involves envisioning models of the sorts of patients and their situations which would call for recommendations and services to regulate fertility. Surely psychiatrists, with their years of fascination by almost every nuance of sexuality, should have no difficulty in addressing themselves to its principal product.

Role of the Social Worker

In any community mental health effort, the social worker will be a prominent participant in promoting family planning. Since the traditional functions of social welfare have centered around people with poverty problems, it seems reasonable to ask how they have handled the associated question of uncontrolled fertility. The answer is astonishing: They have done virtually nothing. In 1962 the National Association of Social Workers adopted a policy statement which, in general terms, supports planned parenthood and the social worker's part in promoting it. However, detailed examination of welfare agency operations reveals that contraception has been treated as a loaded issue, better let alone, and this has been agency policy with few exceptions. Militant Catholic pressure groups have been a potent influence in suppressing public aid efforts, but there are other reasons. Underpaid, overworked, poorly trained workers are the rule in welfare departments. Often stifled by enormous caseloads and mountains of paper work, they have low morale and a high turnover rate. Their superiors could give no encouragement or funds to those who did wish to offer contraceptive help to their clients. And the organized profession did not provide the leadership to "fight City Hall" and change policy.[25]

Recent months have brought signs of reform. The groundwork was laid by decisions of the Supreme Court and by presidential recognition of the foreign population problem. The Great Society-War on Poverty programs have necessarily focused attention on unwanted children; the recent emphasis on urban affairs has culminated in the creation of a Cabinet-level post. It has become increasingly obvious that women of all socioeconomic levels urgently desire family planning and that the public at large (Catholics included) overwhelmingly approve of its being made available. It follows that the poor and uninformed are those being denied its benefits, and that this amounts to discrimination based on poverty and ignorance.

A variety of Federal bureaus are now appearing with funds to offer birth control services in their field agencies. The social work profession has certainly missed being in the vanguard, but it should join the main body without delay. The following might be taken as a statement of goal: "The major issue is to make information [and services] about all methods of family planning easily available to all Americans. Welfare workers must be as free to suggest that a welfare recipient seek this advice as they are to suggest that an employment agency might help with a job problem or that night school might resolve illiteracy."[26]

As would be expected, psychiatric social workers have been even less concerned with family planning than have their counterparts in general welfare work. The psychiatric social worker has tended to follow the psychiatrist's example, at times outdoing him in her devotion to non-directiveness.

Working with the Psychiatric Patient

Assuming that psychiatrists take a positive position on family planning, many questions will be sure to follow. It is hardly possible to anticipate them all, but some general observations are in order. For example, is it proper for the psychiatrist (or mental health facility) to influence his patients to limit their families? Of course it is, in the same sense that any other significant activity of the patient is "influenced"—the psychiatrist draws attention to a problem and attempts to help his patient see the motivations and the issues that pertain to a rational decision; from there it is the patient's choice.[22]

Psychiatrists must guard against their tendency to be so cowed by the incompleteness of their knowledge that they fail to apply what they do know. The nature-nurture problem in schizophrenia can continue unresolved, yet this in itself should not prevent one from advising certain couples not to have additional children. There are persons who have been capable of producing only maladapted children; whether this is a result of genetic factors or of their own irremediably faulty interaction is quite academic.

Psychiatrists do know that the incidence of serious mental illness and delinquency is inordinately high in severely deprived urban cultures.[17] Many families will be seen who would be able to raise one or two healthy children, but whose chances with endless successive births are probably nil. Counseling in such cases becomes truly preventive psychiatry.[9]

When coupled with the anticipation of future research developments, a positive program of family planning offers genuinely exciting possibilities. On the one hand, there could well be breakthroughs in the recognition of specific genetic-biochemical determinants of subgroups now non-specifically viewed as schizophrenia or manic-depressive illness. On the other, there has recently been extremely promising work in analyzing interaction in families of schizophrenic and delinquent patients. With further refinement, it might well be that a high degree of predictability could be reached for prospective parents; some might be classified as treatable *in preparation* for the arrival of children; others might be spared the tragedy of grossly disturbed offspring.[5] Whatever directions research takes, it cannot but be stimulated by the presence of facilities for applying whatever insights it may develop.

The psychiatrist's traditional orientation has not been that of social reformer or public health officer; his concern has been to promote the personal welfare of his patient. Here too, fertility control accommodates this goal. If the sexual act is attended by the fear of pregnancy, marital adjustment suffers. Work with frigidity problems is handicapped if the therapist cannot separate neurotic sexuality from realistic procreativity. The presence of disturbed children, or simply unwanted children, in a family may give rise to serious intrapsychic conflicts which are often expressed interpersonally. Conversely, a family planning program can help correct appropriate instances of infertility to facilitate personal and marital harmony.

Social workers and psychiatrists should join now in the effort to include family planning as an integral part of all mental health organizations. Each socio-psychiatric evaluation should include an appraisal of the knowledge, attitudes and capabilities of each patient in relation to control of his fertility. This information should then be matched with the agency's analysis and recommendations with regard to child-bearing for each patient. The result should be a program that is designed for the individual patient and aimed at enabling him to coordinate his reproductive potential with his total treatment effort. This should, without delay, become a routine part of psychiatric practice.

Mental Subnormality

The "Cinderella of psychiatry," mental retardation, has recently emerged from the scullery, resplendent in new raiment bestowed by a rich Federal godmother. In her position as heiress, she is likely to have a secure place in the coming of social psychiatry.

Most experts on retardation dwell on the themes of retraining and rehabilitation; these are often valuable contributions, and it would be shortsighted to neglect the trainable retardates already among us. But should we not put the future emphasis on prevention? We already have a solid basis for genetic counseling in disorders such as Huntington's chorea and amaurotic idiocy.[6]

The economically deprived, by limiting their families, will be in a better position to give proper nutrition and care to the pregnant mothers and to their babies. In such a way, retardation resulting from deficiency diseases or other early insults can be reduced. As research continues to clarify the causes of retardation, there will be still more reason to see that the findings are applied.[30]

Many adult retardates, particularly females, have been retained in institutions solely because of their reproductive capacity. This is a cruel situation which can be remedied by appropriate contraceptive measures, including sterilization in selected cases. Retarded couples might then live in the community without having their marginal adjustment upset by the birth of children they cannot care for. The children, and society, would likewise be benefited.[31]

The Time for Action

"The time is now" is a phrase one encounters often in papers on population control. It is no less true for psychiatry. The United States has a priceless advantage in its struggle against unwanted millions: We are still in a position to exercise rational selectivity over who will be born and who will not. Nations such as India are forced to deal with sheer numbers, as if all humans were alike. For India, refinement of its program will be possible only after decades of strenuous effort.

The psychiatric profession is increasing its leverage and influence by virtue of the expanding community psychiatry movement. More than ever, "the time is now" for it to add its special skills and knowledge to our country's imperative effort.

REFERENCES

1. American Journal of Psychiatry—Special Section on Community Mental Health, Am. J. Psychiat., 122:977-1020, March 1966.

2. Barton, Walter E.: Personal communication, 25 February 1966.

3. Bernard, V. W.: Some principles of dynamic psychiatry in relation to poverty, Am. J. Psychiat., 122: 254-267, September 1965.

4. Black, E. R.: Population increase and economic development, in Osborn, F.,[29] pp. 83-91.

5. Blau, A.: The unwanted child, in Mudd, S.,[28] pp. 293-305.

6. Carter, C. H. (Ed.): Medical Aspects of Mental Retardation, Charles C Thomas, Springfield, Ill., 1965.

7. Cepede, M., Houtart, F., and Grond, L.: Population and Food, Sheed and Ward, New York, 1964.

8. Charter, S. P. R.: Man On Earth, Contact Editions, Sausalito, Calif., 1962.

9. Clausen, J. A.: Sociology of mental disease, in Freeman, H. E., Levine, S., and Reeder, L. G. (Eds.): Handbook of Medical Sociology, Prentice-Hall, Inc., Englewood Cliffs, N.J., 1963, pp. 145-165.

10. Cook, R. C.: Population and food supply, in Mudd, S.,[28] pp. 451-477.

11. Darwin, C. G.: The law of population increase, in Osborn, F.,[29] pp. 29-35.

12. Day, L. H.: The American fertility cult, in Mudd, S.,[28] pp. 231-238.

13. Day, L. H., and Day, A. T.: Too Many Americans, Houghton Mifflin Co., Boston, 1964.

14. Dorn, H. F.: World population growth: An international dilemma, in Mudd, S.,[28] pp. 47-59.

15. Draper, E.: Birth Control In The Modern World, Penguin Books, Baltimore, 1965.

16. Duhl, L. J. (Ed.): The Urban Condition, Basic Books, New York, 1963.

17. Dunham, H. W.: Epidemiology of psychiatric disorders as a contribution to medical ecology, Arch. Gen. Psychiat., 14:1-19, January 1966.

18. Flugel, J. C.: Some unconscious motives of birth-control opponents, in Maury, M.,[23] pp. 137-143.

19. Freedman, R. (Ed.): Population: The Vital Revolution, Anchor Books, Garden City, N.Y., 1964.

20. Hauser, P. M. (Ed.): The Population Dilemma, Prentice-Hall, Inc., Englewood Cliffs, N.J., 1963.

21. Hauser, P. M.: Demographic dimensions of world politics, in Mudd, S.,[28] pp. 109-121.

22. Lieberman, E. J.: Preventive psychiatry and family planning, J. of Marriage and the Family, 26:471-477, November 1964.

23. Maury, M. (Ed.): Birth Rate and Birth Right, Macfadden Books, New York, 1963.

24. Maury, ibid., p. 62.

25. May, E.: The Wasted Americans, Harper & Row, New York, 1964.

26. May, ibid., p. 167.

27. McPartland, T. S., and Richart, R. H.: Social and clinical outcomes of psychiatric treatment, Arch. Gen. Psychiat., 14:179-184, February 1966.

28. Mudd, S. (Ed.): The Population Crisis and the Use of World Resources, Dr. W. Junk Publishers, The Hague, 1964.

29. Osborn, F. (Ed.): Our Crowded Planet, Doubleday & Co., Garden City, N.Y., 1962.

30. Osborn, F.: The protection and improvement of man's genetic inheritance, in Mudd, S.,[28] pp. 306-313.

31. Reed, E. W., and Reed, S. C.: Mental Retardation: A Family Study, W. B. Saunders Co., Philadelphia, 1965, pp. 71-79.

32. Revelle, R.: The problem of people, Harvard Today, Autumn, 1965.

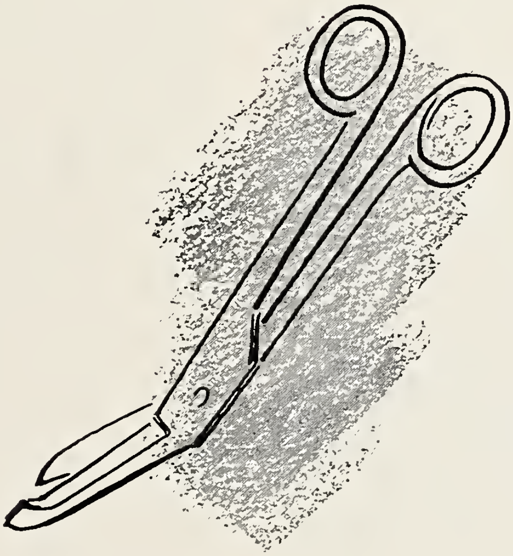
33. Rosenberg, B., Gerver, I., and Howton, F. W.: Mass Society In Crisis, Macmillan Co., New York, 1964.

34. Spencer, S. M.: The birth control revolution, The Saturday Evening Post, 15 January 1966.

35. Vogt, W.: People!, William Morrow, New York, 1960.

36. Vogt, W.: The ethics of parenthood, in Maury, M.,[23] pp. 23-36.

37. Yamamoto, J., and Goin, M. K.: On the treatment of the poor, Am. J. Psychiat., 122:267-271, September 1965.



RESIDENTS' CLINICOPATHOLOGIC CONFERENCE

Pineal Germinoma with Endocrine Manifestations

Residents' Clinicopathologic Conference from the Cedars of Lebanon Hospital Division, Cedars Sinai Medical Center, Los Angeles

HISTORY by SARAH TRIVEDI, M.D., Resident in Medicine

A 17-YEAR-OLD MEXICAN BOY was admitted to hospital 2 April 1964 with complaint of thirst, fatigue, double vision and seizures. He was an athletic, better than average student until December 1962, when polydipsia and polyuria developed, with nocturia (eight times a night), anorexia, fatigue and a 25-pound loss in weight. He stopped all outside activities, began to fail in school, slept 18 hours a day and cried for no reason at all. In June 1963, because of frequent vomiting unrelated to meals, he was admitted to another hospital. Results of physical examination, laboratory tests and x-ray studies were within normal limits and a diagnosis of enteritis was made. Symptoms were ascribed to emotional disturbance and the family was referred to our psychiatric clinic in January 1964. No sensorial defect was observed. After psychological testing the patient began psychotherapy.

In the next three months he had recurring episodes of occipital headache and projectile vomiting, accompanied once by a petit mal seizure. He suddenly noted horizontal diplopia, followed by a grand mal seizure with localization to the left hand. He apparently lost consciousness but did not bite his tongue or become incontinent. At another hospital a diagnosis of hyperventilation and loss of vision was made. An atypical seizure in the psychiatrist's office led to his admission to Cedars of Lebanon Hospital.

On questioning, the patient said he had no difficulty with sphincters and he had normal erections. He had shaved only about four times, the last time three months previously. Injuries included lacerations of the scalp after falling in the bathtub and after being thrown from his bike, in the spring and fall of 1963. There was a familial history of diabetes mellitus.

The patient was tall, well-developed, slightly obese and physically immature. He had little facial or body hair, a female escutcheon, normal genitalia and no gynecomastia. The voice was of medium depth. The thyroid gland was not enlarged. Skin, hair, pigment and nails were normal.

The left upper extremity was weak but there was no atrophy and no fasciculation. Gait was wide-based and slightly ataxic, with most difficulty on the left. The left arm drifted down. Finger-nose and heel-shin tests were performed poorly bilaterally. Dysdiadochokinesia and rebound were present on the left. There was slight hyper-reflexia on the left, a positive left Chaddock sign and questionably positive left Babinsky sign but no other pathological reflexes.

The patient was unable to do any calculations, consistently missed *W* in the alphabet and was unable to complete his own address but knew government structure.

Vision was slightly decreased on the right. The right pupil was dilated, reacted sluggishly to light and had no consensual reaction. The left pupil reacted normally. Slight left internal strabismus was present. On funduscopic examination a "watery" retina and venous pulsations were noted.

Submitted 8 June 1966.

Reprint requests to: Division of Laboratories, Cedars-Sinai Medical Center, 4833 Fountain Avenue, Los Angeles 90029 (Dr. Nathan B. Friedman).

Visual fields were normal to confrontation and perimetric examination.

On lumbar puncture the opening pressure was 250 millimeters of water and the final pressure was 160 mm after 20 ml of clear colorless fluid were removed. The fluid contained four red cells per cu mm and no white cells. The protein content was 17 mg, sugar 66 mg and chloride 708 mg per 100 ml. Lactic dehydrogenase was 17 units (normal less than 42). Routine cultures for acid-fast bacilli and fungus were negative.

Specific gravity of the urine was 1.005 and the osmolality was 180 milliosmols per liter, with 295 milliosmols per liter in the serum. Another specimen had 140 milliosmols per liter with 300 milliosmols per liter in the serum. Blood sugar, fasting, was 88 mg, and two hours post-prandial was 72 mg per 100 ml. Uric acid was 5.9 mg and urea nitrogen was 14 mg per 100 ml of serum. Serum lactate dehydrogenase was 140 units. Serum sodium was 147 mEq and potassium 4.0 mEq per liter, the pH, pO_2 and the calcium and phosphorus contents were within normal limits. Protein-bound iodine was 5.1 gamma per 100 ml and cholesterol was 270 mg per 100 ml of serum. Serum protein electrophoresis was normal. A rat hyperemia test for gonadotrophins was negative. Urinary excretion of potassium was 79 mEq and sodium 92 mEq per 24 hours. On admission urinary 17-ketosteroids in a 24-hour specimen were 7.2 mg and 17-ketogenic steroids were 11.0 mg. After angiography, urinary 17-ketosteroids and 17-ketogenic steroids each rose to 18.9 mg per 24 hours, presumably due to the stress of the procedure. A mercury brain scan was negative. Electroencephalograms were considered normal.

During the stay in hospital the patient slept most of the time but was easily aroused. Neurological symptoms waxed and waned. Intermittent sustained bilateral ankle clonus developed. The pupils were dilated and non-reactive at times, with no change in the fundi. Fluid intake and output was nine to 10 liters per day.

After radiologic studies (see following), the posterior fossa was explored. The cerebellar hemispheres revealed no evidence of tumor, but probing the fourth ventricle showed a block at the upper end. Dye did not pass from the right lateral into the fourth ventricle. A Torkildsen shunt was placed betwen the posterior horn of the right ventricle and the cisterna magna.

After the operation, diabetes insipidus was worse, the urine output reaching a maximum of 14 liters a day, but could be decreased with pitressin. However, signs of increasing intracranial pressure and midbrain involvement developed. On the eighth post-operative day the patient had a grand mal seizure, aspirated vomitus and died.

Roentgenologic Findings by Richard A. Friedman, M.D., Resident in Radiology

Examination of the skull revealed an increase and prominence of the convolutional markings, but the remainder of the bony structures, including the sella turcica, were normal. An amorphous collection of calcium 1 cm in diameter was present in the temporo-parietal region on the lateral view and approximately in the midline on the posterior-anterior projection. This calcification was interpreted variously as eccentric calcification in a large pineal body, calcification within a mass in the region of the pineal or calcification in a pineal which had been displaced.[3] Angiograms showed: (a) Stretching and elevation of branches of the left anterior cerebral artery (Figure 1); (b) elevation of the left middle cerebral group of arteries; (c) lateral and caudad displacement of portions of the striothalamic veins and (d) a block of the distal two-thirds of the right posterior cerebral artery

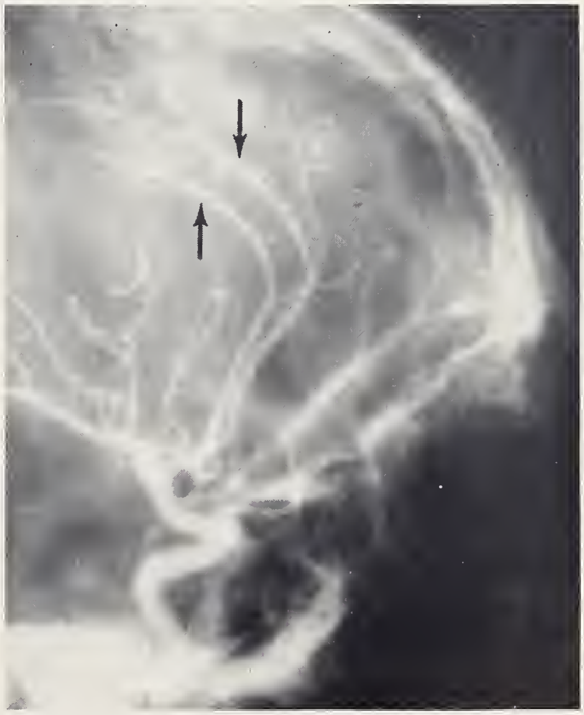


Figure 1.—Following injection of contrast material into left common carotid artery, stretching and elevation of the pericallosal and callosal marginal arteries is seen. Also note elevation of cerebral arterial axis.

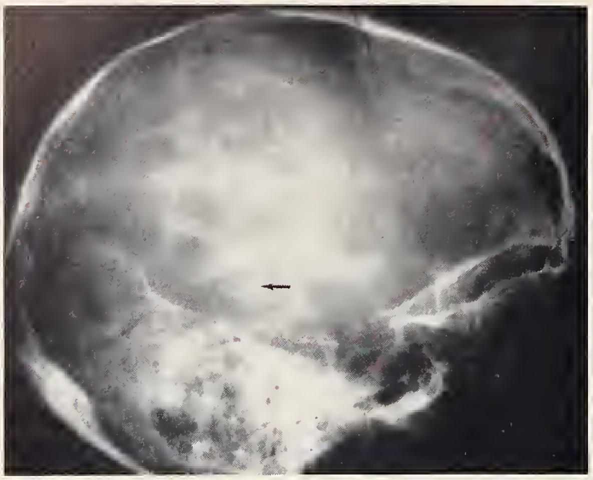


Figure 2.—Lateral projection shows tortuous vessels in region of pineal calcification.

adjacent to a small collection of tortuous vessels (Figure 2) in the region of the previously described amorphous calcific deposits.[12,16] Ventriculography demonstrated filling of both lateral ventricles and the third ventricle; all were dilated. No air was seen in the aqueduct or in the fourth ventricle. Therefore, the lesion postulated in the arterial studies proved to obstruct both inflow and outflow of the fourth ventricle.[17]

These findings indicated a partially calcified mass containing tumor vessels in the region of the pineal, which displaced local vessels and, by causing a hydrocephalus, also displaced distant vessels. While the most likely diagnosis was pinealoma or teratoma of the pineal body, other sites such as tumor of the quadrigeminal plate and posterior portion of the third ventricle were considered, since astrocytomas and glioblastomas in this area have similar radiographic signs.

Finally, it is interesting that whereas pineal body calcification is present in only one-sixth of persons of the same age as the patient in the present case, the condition exists in half of patients who have a tumor in the pineal region.

Clinical Discussion by Marvin Kanter, M.D., Resident in Pediatrics

In the case here presented we have the puzzling problem of a progressive illness which lasted approximately two years and was manifested in, among other things, excessive thirst, polyuria and weight loss. As often happens, these symptoms, together with the extreme change of personality noted in this patient, were attributed first to an emotional disturbance; but this diagnosis was discarded subsequently when signs of organic involvement supervened. In passing, it is worth mentioning that compulsive water-drinking can lead to impaired ability to secrete anti-diuretic hormone and to concentrate urine; but when fluids are withheld, such patients are able to produce concentrated urine.

Diabetes mellitus as a cause for the polydipsia, polyuria and weight loss was easily excluded, as were other causes of polyuria, such as chronic renal disease, hypocalcemia and hypokalemia.

We are left, then, to explain diabetes insipidus, emotional outbursts and somnolence. This constellation points to the hypothalamus as the seat of the problem. As to diabetes insipidus, it may occur in three forms: (1) Idiopathic type, with onset in infancy, little effect on general health, and a familial tendency; (2) nephrogenic type, in which the kidney is unresponsive to vasopressin, also familial, with sex-linked inheritance; and (3) organic type, resulting from destruction, by any of a variety of lesions, of the posterior lobe of the pituitary gland, the infundibulum or the hypothalamic region. It is difficult to assess the absence of sexual infantilism and typical obesity of Froehlich's syndrome and the presence of secondary sexual characteristics in the present case. Nevertheless, more than half the cases of diabetes insipidus are due to destructive neurophypophyseal lesions, and I believe that the patient belongs in this group.

To recapitulate, the patient first had symptoms of increased intracranial pressure without physical findings, and then (consecutively) had a watery retina, noted on admission and taken to be an early sign of papilledema. Subsequently waxing and waning neurological signs pointed to involvement of the cerebellum and cranial nerves, chiefly oculomotor, evidence of pyramidal tract involvement and aphasia, indicating cortical involvement.

The differential diagnosis is long. Hand-Schuller Christian disease can be excluded for lack of typical skull findings as well as of other features. Leukemia and syphilis are eliminated by the protocol. A tuberculin test was not done. In the absence of antecedent trauma, basilar skull fracture is excluded. An angiogram showed neither arteriovenous malformations or aneurysm of the internal carotid artery. In sarcoidosis, usually there is elevation of protein, and the presence of many lymphocytes in the cerebrospinal fluid and elevation of the alpha-2 globulin on serum electrophoresis. X-ray films of the chest frequently show hilar adenopathy. Uveo-parotid fever and facial nerve palsies are frequent concomitants. Encephalitis might cause the manifestations noted in the present case, but it is usually associated with a previous febrile illness. Stenosis of the aqueduct of Sylvius secondary to either gliosis or an obstructive membrane may engender many of the characteristics observed in this patient, but I don't believe that the sensory findings would be so great, and moreover the findings of the pneumoencephalogram are incompatible with such a diagnosis. Multiple sclerosis may give recurrent episodes of acute hydrocephalus, but with the degree of deterioration in this patient I would expect the characteristic tabetic curve in the colloidal gold test.

Pseudotumor cerebri belongs with Wilson's disease at the bottom of the list, because of the findings of the ventriculogram. A "far out" consideration is parasitic cyst of the brain, an extremely rare condition in this country but one to be taken into account because the patient was Mexican. Symptoms may fluctuate due to edema around the cysts, and also floating cysts in the ventricle may cause intermittent obstruction, with sudden attacks of headache and vomiting. Usually the patient has eosinophilia.[7]

We are now coming to the various tumors that may cause these syndromes and it is here, I believe, that our answer lies. With diabetes insipidus the first tumor that comes to mind is craniopharyngioma, which commonly occurs at this age. Various endocrinopathic abnormalities may accompany this tumor and indicate hypothalamic involvement. However, craniopharyngioma usually is associated with calcification and most often causes a flattening of the sella with destruction of the clinoid processes. Frequently, visual field defects are present and the third ventricle is obliterated or distorted—findings not present in this patient. Pituitary adenoma is unlikely because the tumor is uncommon at this age and it causes disturbed visual fields and ballooning of the sella. A colloid cyst of the third ventricle may cause fluctuating neurologic symptoms by intermittently obstructing outflow of cerebrospinal fluid from the ventricles, causing elevated pressure which ultimately displaces tumor and discharges the fluid, with temporary remission of symptoms. Also, change of position exaggerates symptoms, a finding not described in this case.[1]

A solid tumor of the third ventricle can produce the findings noted in this case. It is interesting that, due to pressure, hydrocephalus alone may cause hypothalamic symptoms. Against tumor of the third ventricle is the non-filling of the fourth ventricle. Also, tumors of the third ventricle may extend into the hypothalamus, a significant factor in the differential diagnosis.[1,7] The irregular calcification in the area of the pineal body, and also abnormal vessels in this area, arouse strong suspicion of a tumor in this region.

Pinealoma now comes under consideration. The literature is conflicting about the nature of pinealoma and the tumor has gone under various names.[2,15,19] The greatest incidence is in males under 25 years of age. A common finding in this condition (50 per cent of cases) is Parinaud's ophthalmoplegia, but this is usually unremitting and I am told that while our patient may have exhibited this finding once, it abated and did not recur. Ataxia and other cerebellar signs are common; spasticity, exaggerated deep tendon reflexes and Babinski's sign also are frequent. Most of the patients have involvement of the third cranial nerve, and hypothalamic involvement is frequent. Precocious puberty with pineal tumor is referred to as the pineal syndrome. Signs of increased intracranial pressure develop early due to obstruction of the aqueduct, and the fourth ventricle may be compressed. Seizures and defects of mentation are quite common. The spinal fluid is normal in 50 per cent of cases.

Tumors found in this region are pinealomas, atypical teratomas, astrocytomas, gliomas, ependymomas and cysts. As a sidelight, pinealomas have been found in the suprasellar region with an apparently normal pineal body in the usual location.[7]

The atypical teratomas, most common tumor of the region, may be highly invasive and give multiple symptoms such as were present in this case. The course is usually one to two years. I cannot rule out a cerebellar tumor, but because of the calcification of the pineal body and the abnormal vessels in that region, I will say that on postmortem examination an atypical teratoma with extensive involvement of the brain substance will be found to explain all the above findings.

PATHOLOGY by C. JACK BARK, M.D., Resident in Pathology

THE MOST INTERESTING postmortem findings were in the central nervous system. The occipital portion of the skull was absent and there was an overlying sutured semilunar skin flap. There was 25 cc

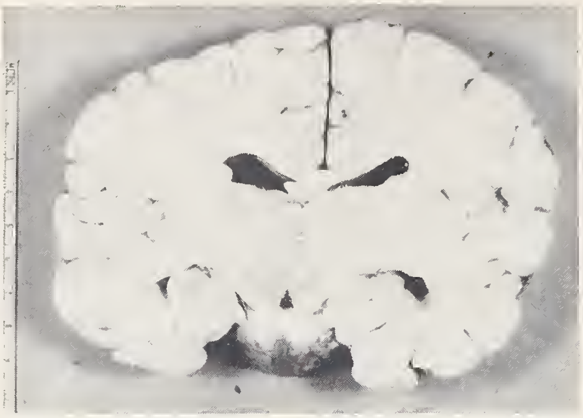


Figure 3.—Tumor filling the third ventricle and invading adjacent thalamic tissue.

of dark blood in the posterior fossa. A rubber catheter extended from the posterior portion of the right lateral ventricle out through the dura, running subcutaneously to reach the cisterna magna. The brain weighed 1,140 gm and was uniformly edematous. The lateral and third ventricles did not appear dilated, suggesting functioning of the shunt. At the level of the mamillary bodies a 0.8×0.8×0.5 cm area of gray, friable tumor was found in the floor of the third ventricle. On viewing the third ventricle from the front, more tumor was seen, beginning at the massa intermedia and filling the posterior portion of the ventricle (Figure 3). No connection between the small focus in the floor of the third ventricle and the bulk of the tumor in the posterior portion of the ventricle was demonstrated. This larger tumor measured approximately 2×2×1 cm and extended into the cystic pineal posteriorly (Figure 4). Laterally both thalamic nuclei were invaded for a short distance. Superiorly the tumor extended into the corpus callosum. Inferiorly it extended into the third ventricle and the anterior portion of the aqueduct of Sylvius.

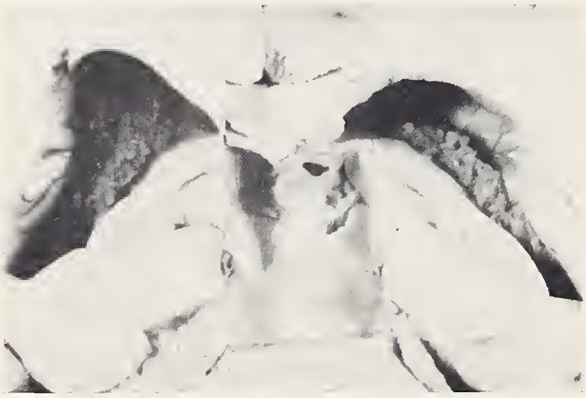


Figure 4.—Cystic pineal body continuous with tumor.

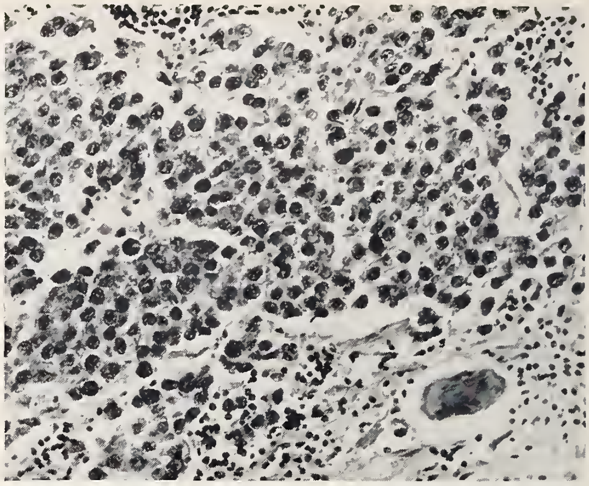


Figure 5.—High power view of pineal tumor, showing classic germinoma with both spheroidal and small cell types. Also note giant cell (×325).

Histologically the tumor was a germinoma of the pineal body. The two classic cell components were present, the large spheroidal cell and the small cell which is indistinguishable from the lymphocyte. In addition, there were scattered giant cells with pseudoturbercles (Figure 5). (Such cells are also present in germinomas of the testis, ovary and thymus.) The tumor in the floor of the third ventricle was histologically identical and presumably was metastatic via the ventricular fluid, a finding not uncommon in pineal germinomas (Figure 6). The pineal gland was almost completely replaced by tumor. Not observed grossly, but nicely demonstrated histologically, was invasion of the posterior lobe of the pituitary gland, presumably a direct extension from the floor of the third ventricle down the pituitary stalk. (It is the frequent involvement of the hypothalamic-hypophyseal area which leads to diabetes insipidus as a presenting complaint.) The tissue surrounding the catheter showed reactive changes. Small bilateral cerebellar hemorrhages outlined the path of the cerebellar aspiration carried out during operation. The terminal event appears to have been aspiration pneumonia. The lungs weighed 1,140 grams and showed focal atelectasis, emphysema and hemorrhages. Histologically they showed multiple foci of aspirated fine debris with surrounding neutrophils and fibrin.

The endocrine organs were an interesting facet of this case. Those examined were smaller than usual. The testes weighed 15 grams each and histologically showed atrophy. Both were studied by multiple blocks of the entire organ in searching

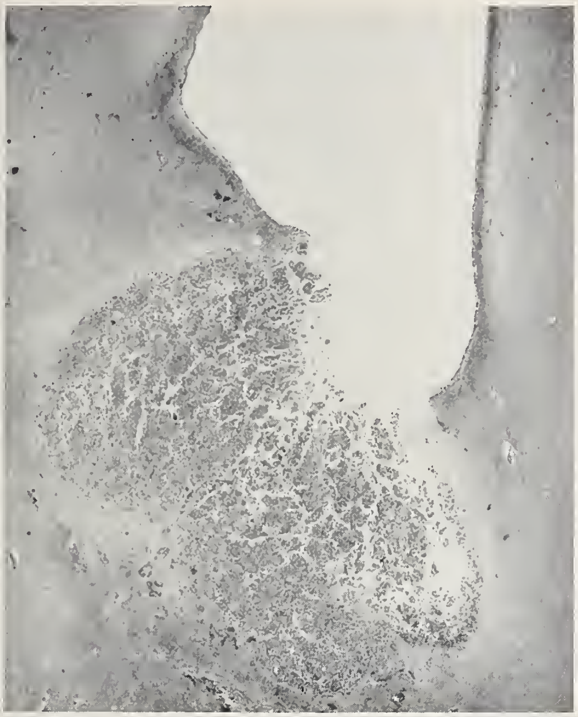


Figure 6.—Tumor in the floor of the third ventricle. (×20).

for a theoretically possible primary germinoma, but neither tumor nor scar was found. The thyroid gland weighed 6 gm and was small. Histologically it appeared inactive, although this can be misleading. The adrenal glands each weighed approximately 6 gm and had a thin zona fasciculata.

More than 20 years ago, Collins[4] described panendocrine atrophy in a woman with a germinoma of the hypothalamus, although the neoplasm was not so identified at the time. The anterior lobe of the pituitary was histologically intact, but the hypothalamus was destroyed by tumor. Collins postulated some sort of hypothalamic control over anterior lobe function, in addition to the well-known posterior lobe control. In the present case also the changes may be manifestations of early panendocrine atrophy due to loss of hypothalamic control.

Germinoma of the pineal is a rare lesion, almost a curiosity. Pineal tumors constitute less than 1 per cent of all intracranial tumors but germinomas are the most common. The clinical discussion brought out the fact that the tumors do not necessarily occur in the pineal body itself. Ectopic "pinealomas" with a predilection for midline structures such as the optic chiasm and the floor of the third ventricle are well known. The frequent obstruction of the aqueduct of Sylvius and involvement of the hypothalamus with the resulting internal hydrocephalus and diabetes insipidus prompt the patient to seek medical aid. Weight gain, emotional disturbances and temperature elevation also can occur with hypothalamic involvement. Metastasis occurs most commonly by the spinal fluid route, involving the ventricular system and the spinal cord; and spread to the lungs has been described. Usually the tumor extends into a large vascular channel within the brain and then finds its way to the lungs.[20]

Perhaps the most intriguing aspect of germinomas of the pineal body is their relationship to germinomas and teratomas elsewhere. Russell[18] in 1944 described the similarity between these tumors of the pineal body and the seminomas of the testis. It was her feeling that they represented a one-sided development of a teratoid tumor. She believed that if the tumor was sufficiently sampled, areas of further differentiation would be found. A postulation that lends support to the name "atypical teratoma." Friedman[8] two years later reaffirmed certain of Russell's findings. In a later work on teratoid tumors, he further pointed out that germinomas, embryonal carcinoma and teratocarcinomas of pineal origin are indistinguishable from those occurring in the gonads.[9] The similarity in clinical behavior and identical histologic pattern led him to conclude that these tumors had a common cell of origin, the primordial germ cell.[10] It is not hard to see how the gonads become the site of these tumors. Germinomatous tumor cells closely resemble the primordial germ cells in the embryo. The fact that primordial germ cells wander through various tissues before coming to rest in the gonadal region might account for the occurrence of extragonadal teratoid tumors.

THERAPY by G. STEPHEN BROWN, M.D., Resident in Radiation Therapy

THESE INFREQUENT tumors have their greatest incidence in the mid-teens. In a series of some four thousand cases of brain tumors reported by the Armed Forces Institute of Pathology, the incidence of pinealoma was a little more than 1 per cent.[13] Early diagnosis of the lesion is often difficult, since all symptoms arise from compression or local destruction of tissue.

Since the first series reported by Dandy in 1936, the operative mortality associated with surgical decompression of these tumors has been between

20 and 70 per cent.[5,6] In the late 1940's reports of series in which radiation therapy was used began to appear and the survival was improved. As these tumors are frequently radiosensitive, and surgical extirpation can be extremely dangerous, radiation therapy has been suggested as the first mode of therapy unless immediate surgical decompression is necessary. In cases in which operation is imperative due to acutely increased pressure, the mortality is high. Biopsy of the region involves considerable risk and because of the rather typical clinical and roentgenologic manifestations associated with pineal tumors, definitive histologic diagnosis before treatment may sometimes not be possible. The course of treatment which has had the greatest success to date is simple decompression to relieve the pressure and to anticipate the swelling which occurs early in the therapy. Ventricular shunt, which has a low operative mortality, can be performed to prevent obstructive phenomena from either growth of the tumor or edema secondary to treatment. Radiation therapy is undertaken through opposed lateral ports to deliver 4,000 roentgens (in air); the tumor dose is approximately 50 per cent of this value.[11] For children aged one year the dose is 50 per cent of the adult dose; at five years the dose is 75 per cent, and in children eight years of age and older the full adult dose is given.[5] The therapy is fractionated over four to five weeks. Following the treatment, the patient is seen at least at one-month intervals for check-up. If the decompression site continues to bulge, a second course of therapy is begun. It has been noted, however, that if the tumor is sensitive it will usually respond during the first course of therapy. Persistence of symptoms following radiation therapy is taken as an indication for surgical treatment.

According to reports scanned in a review of the literature, the operative mortality from tumor removal or biopsy averages 30 to 35 per cent.[14] Ventricular shunts have an operative mortality of approximately 5 per cent.[13] The five-year survival rate in patients treated by radiation therapy is 61 per cent whether previously treated by partial removal, biopsy or shunt or not operated upon at all.[5] Therefore, radiation therapy is the preferred mode of primary treatment.

REFERENCES

1. Baker, A. B.: Clinical Neurology. Hoeber-Harper, New York, Vol. I, pp. 460-563, Vol. III, pp. 1338-1395, 1962.
2. Booth, C. B., Swartz, E., Janis, R., and Kruger, E. G.: Atypical teratoma, Neurology, 13:999, 1963.
3. Camp, J. D.: Significance of intra-cranial calcification in the roentgenological diagnosis of intra-cranial neoplasms, Radiology, 55:659, 1950.
4. Collins, Vincent P.: Effects of destruction of hypothalamus by tumors, Arch. Neurol., 48:774, 1942.
5. Cummins, F. M., Taveras, J. M., and Schlessinger, E. B.: Treatment of gliomas of the third ventricle and pinealomas, Neurology, 10:1031, 1960.
6. Dandy, W. E.: Operative experience in cases of pineal tumors, Arch. Surg., 33:19, 1936.
7. Ford, Frank R.: Diseases of nervous system in infancy, childhood and adolescence, Charles C Thomas, Springfield, Chapter VII, Neoplasms and Related Conditions Involving the Nervous System, pp. 952-1073, 4th ed., 1960.
8. Friedman, Nathan B.: Germinoma of the pineal—its identity with germinoma ("seminoma") of the testis, Cancer Res., 7:363, 1947.
9. Friedman, Nathan B.: The comparative morphogenesis of genital and gonadal teratoid tumors, Cancer, 4:265, 1951.
10. Friedman, Nathan B.: Choriocarcinoma of the testis and extragenital choriocarcinoma in man, Ann. N.Y. Acad. Sci., 80:161, 1959.
11. Horrax, G.: Diagnosis and treatment of pineal tumors, Radiology, 52:186, 1949.
12. Johannson, C.: The central veins and deep dural sinuses of the brain, Acta Radiol. Supp., 107, 1954.
13. Kernohan, J. W., and Sayre, G. P.: Atlas of tumor pathology section X fascicles 35 and 37—Tumors of the central nervous system, Armed Forces Inst. of Path., 124-129, 1952.
14. Kunicki, A.: Operative experiences in eight cases of pineal tumor, J. Neurosurg., 17:815-823, 1960.
15. Lewis, J., Baxter, D. W., and Statford, J. G.: Atypical teratomas of the pineal, Canad. Med. Assn. J., 89:103, 1963.
16. Löfgren, F. O.: Vertebral angiography in the diagnosis of tumors, Acta Radiol., 50:108, 1958.
17. Rand, R. W.: Tumors of the posterior portion of the third ventricle, J. Neurosurg., 10:1, 1953.
18. Russell, Dorothy S.: The pinealoma; its relationship to teratoma, J. Path. Bact., 56:145, 1944.
19. Suzuki, J., Tokuo, W., and Kowada, M.: Clinical observations on tumors of the pineal region, J. of Neurosurg., 19:441, 1962.
20. Tomkins, V. N., Haymaker, W., and Campbell, E. H.: Metastatic pineal tumors, J. Neurosurg., 7:159-168, 1950.

Colloidal gold, protein, sugar and cells were all found to be within normal limits. Blood creatinine and fasting blood sugar were normal, as were complete hemogram and urinalysis.

Diphenylhydantoin sodium (Dilantin®), 100 mg three times a day was prescribed and in a one month follow-up there were no seizures. The patient then stopped taking Dilantin and at last report no further seizures had occurred. However, it should be noted that the patient had not taken LSD since the occurrence of the seizure, several months before, that had caused him to seek medical consultation.

Discussion

Although this appears to be the first reported case of grand mal seizures associated with ingestion of LSD, it should be noted that many persons with complications of LSD are not seen by physicians or do not go to hospitals.[4] The mechanism by which LSD produced the seizure activity is not known. The drug may have reduced the seizure threshold.

With the continued widespread use of LSD, physicians should be alert to the possibility of yet another side effect from this very controversial drug.

Summary

A man who had no past history of such occurrences, had two grand mal seizures after oral ingestion of LSD. Subsequent neurological and electroencephalographic evaluation showed no abnormalities. Dilantin was prescribed and when at the end of a month no further seizures had occurred, the patient stopped taking the drug. He had not taken LSD since he had the seizure that caused him to seek medical advice.

GENERIC AND TRADE NAME OF DRUG

Diphenylhydantoin sodium—*Dilantin.*

REFERENCES

1. Cohen, Sidney: Personal communication.
2. Cohen, Sidney, and Alpert, Richard: LSD, New American Library, New York, 1966, page 84.
3. Cohen, Sidney: A classification of LSD complications, Psychosomatics, VII:185, May to June 1966.
4. Frosch, W. A., Robbins, E. S., and Stern, M.: Untoward reactions to LSD resulting in hospitalization, New Engl. J. Med., 273:1235-1239, 2 December 1965.
5. Rosenfeld, Albert: The vital facts about the drug (LSD) and its effects, Life, 60:30-31, March 1966.
6. Rosenfeld, Albert: Personal communication.
7. Ungerleider, J. T., and Fisher, Duke D.: LSD: Research and joy ride, The Nation, 16 May 1966.
8. Ungerleider, J. T., Fisher, Duke D., and Fuller, M.: The dangers of LSD, J.A.M.A., 197:389-392, 8 August 1966.

Autoimmune Progesterone Urticaria

THEODORE A. TROMOVITCH, M.D., AND
WILLIAM F. HEGGLI, M.D., *San Francisco*

URTICARIA CAN BE caused by a wide variety of factors. The following case report of autoimmune progesterone urticaria stresses that endogenous hormones should be considered in cases of chronic urticaria.

Report of a Case

A 29-year-old white married woman, gravida 4 para 4, had recurrent dermatitis for eight years. The lesions were typical hive-like wheals 0.5 to 3.0 cm in diameter which occurred anywhere on the skin. They regularly appeared seven to 10 days before the menstrual period and spontaneously disappeared approximately the third day past the period. Between times the patient was completely free of dermatitis of any type. Except for this recurrent disease she was in excellent health.

The history did not elicit any relationship of the disease to drugs, and elimination of such common offenders as aspirin and vitamins brought about no remission, nor did elimination diets provide a clue.

A month before she was observed, the temporal pattern of urticaria changed: the lesions continued for a month instead of abating with the cessation of menses. Treatment with antihistamines had given partial relief for a time, but the severity of the eruption had also increased to the point that the use of oral steroids was necessary.

On physical examination the only abnormalities noted were florid bilateral and symmetrical urticarial lesions of the trunk and extremities.

Results of routine examination of blood and urine were within normal limits.

When the lesions were present in a relatively mild state, a single intramuscular injection of 20 mg of progesterone was given. Within an hour the hives already present became larger and new lesions developed over the entire cutaneous surface. The size and number of lesions increased for another hour and it became necessary to stop further progress of the urticaria.

From the Department of Medicine (Dermatology), University of California Medical Center, San Francisco.

Submitted 6 September 1966.

Reprint requests to: 909 Hyde Street, San Francisco 94109 (Dr. Tromovitch).

Premarin® was given on a cyclic schedule, 0.625 mg a day for 21 days each month, to suppress endogenous progesterone production. In addition 15 mg of prednisolone and 32 mg of chlorpheniramine were given daily. Hives continued. At approximately the sixteenth day of the menstrual cycle the dose of Premarin® (conjugated estrogens, equine) was increased to 1.25 mg daily. Within five days urticaria had almost completely disappeared and over the next few weeks the patient was able to discontinue the prednisolone and chlorpheniramine. For three months while taking Premarin 1.25 mg daily she was free of hives. Then a few lesions developed, and at last report the disease was controlled with 1.875 mg of Premarin a day.

Discussion

Shelley and coworkers[2] recently described autoimmune progesterone dermatitis. Their patient was a 27-year-old woman who, at the time of menses, had an extensive vesicular and bullous eruption resembling dermatitis herpetiformis clinically and erythema multiforme histologically. Flares of the dermatitis could be regularly produced by injection of progesterone and progestational compounds. Several hours after a single dose of progesterone, the characteristic lesions would develop and show degranulation with disappearance of circulating basophil leukocytes. Clinical remission could be induced by cyclic oral administration of ethinyl estradiol, 0.05 mg daily. Following oophorectomy the patient had complete involution of all clinical signs and symptoms.

Zondek and Bomberg[3] in reviewing endocrine allergy also described a case of progesterone urticaria. Both they and Kupperman[1] discussed skin testing with hormones. However, skin testing gives variable results. Direct production of urticaria by intramuscular injection of progesterone proved to be a definitive test in the case herein reported and in one reported by Shelley and coworkers.[2] The patients in both cases may well present part of a spectrum. In the present case, the appearance of urticaria within one hour after injection of 20 mg of progesterone suggests an immediate hypersensitivity reaction. Shelley's case, in which there was reaction of erythema multiforme type and a latent period of approximately seven hours after injection of the progesterone, suggests an intermediate hypersensitivity.

Summary

In a case of autoimmune progesterone urticaria, the patient had typical urticaria for the week before menstruation and through the menstrual period, with complete freedom of eruption in the intervening time. This persisted for eight years. In tests, the lesions were found to occur within an hour after administration of 20 mg of exogenous progesterone. Oral administration of Premarin, 1.25 to 1.875 mg a day, in cyclic fashion, controlled the condition.

GENERIC AND TRADE NAME OF DRUG

Conjugated estrogens (equine)—*Premarin.*

REFERENCES

1. Kupperman, H. S.: Human endocrinology, F. A. Davis Co., Philadelphia, 1963.

2. Shelley, W. B., Preucel, R. W., and Spoont, S. S.: Autoimmune progesterone dermatitis, J.A.M.A., 190:35-38, 5 October 1964.

3. Zondek, B., and Bomberg, Y. M.: Endocrine allergy: Clinical reaction of allergy to endogenous hormones and their treatment, J. Obs. & Gyn. Brit. Comm., 54:1-19, 1947.

Amebic Granuloma Of the Cecum

WALTER M. SHAW, M.D., *Chula Vista,* AND
BENEDICT R. WALSKE, M.D., *Tucson, Arizona*

INFREQUENTLY, INTESTINAL amebiasis may be manifested by a granuloma which is an end result of destruction and repair with an overgrowth of granulation tissue. Microscopically, the granuloma shows fibroblasts, collagen, chronic inflammatory debris and necrosis and is located chiefly in the submucosal and the subserosal layer.[7] The rectum and cecum are the most common sites for these granulomas.[5]

This lesion may mimic carcinoma and it is difficult and sometimes not possible to differentiate between an amebic granuloma and a carcinoma of the colon. The deformity of the colon presented by most neoplasms is more likely to be asymmetrical as compared with the more characteristic symmetrical distribution of defects in the intestinal wall caused by amebiasis.[2] It may, of course, not be possible to distinguish the difference. The differential diagnosis will also include consideration of

---

From the Surgical Service, Veterans Administration Hospital, Tucson, Arizona.

Submitted 10 October 1966.

Reprint requests to: McCausland-Robinson Clinic, 275 F Street, Chula Vista 92010 (Dr. Shaw).

tuberculoma and other types of granulomata, particularly those of regional enteritis.[1]

The prevalence of infestation with endoameba histolytica is greater in the tropics than in temperate or arctic zones but the incidence of infestation by endoameba histolytica is by no means analogous to the incidence of the disease known as amebiasis.[3]

Although the morbidity and mortality rates following intestinal operations for these lesions have been reported to be very high,[5,7-10] French[4] advocated operation in such cases when the nature of the lesion is in doubt and stools are negative for ameba. In a recent report,[6] a granuloma in the colon disappeared after antiamebic therapy, and surgical intervention was avoided.

Report of a Case

A 45-year-old man of Mexican extraction was admitted to the Tucson Veterans Administration Hospital 21 April 1965 because of anorexia, nausea, weakness and a weight loss of 20 pounds since the beginning of the illness approximately three weeks previously. He denied having had diarrhea. Four months before admission, the patient had returned to this country from Mexico.

Initially, results of multiple stool examinations for endoameba histolytica were negative. A barium enema study showed a constant filling defect of the lateral aspect of the cecum and the radiologist reported some degree of irritability and change in caliber of the defect (Figure 1). Right hemicolectomy was planned, but when a preoperative stool examination revealed numerous motile endoameba histolytica, the operation was cancelled and an antiamebic regimen was begun. An x-ray study three months after treatment demonstrated no radiographic evidence of residual granuloma (Figure 2).

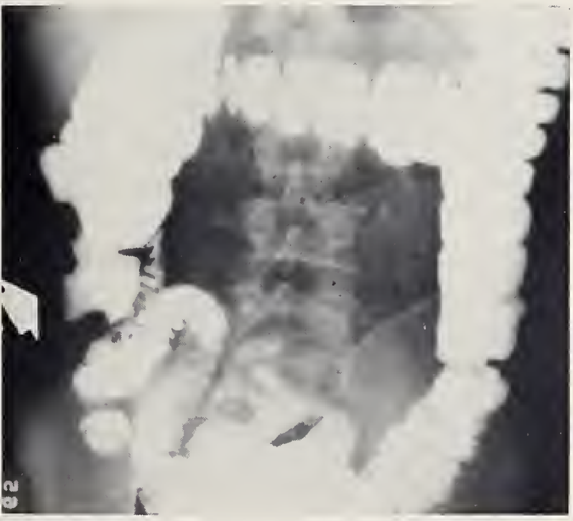


Figure 1.—Barium enema study showing filling defect in lateral aspect of cecum.

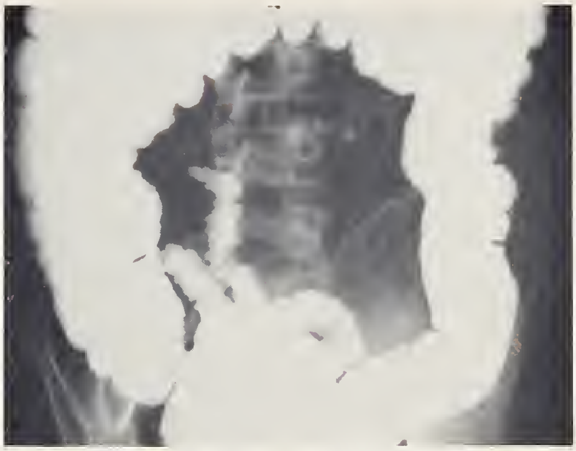


Figure 2.—X-ray film, three months after treatment, showing no evidence of residual granuloma.

Discussion

We were prepared to follow French's advice that operation be carried out in cases of demonstrable intestinal tumor when stools are negative for ameba. However, it must be borne in mind that endoameba histolytica can be difficult to demonstrate in the stool and therefore there is possibility of operating in an active case of intestinal amebiasis, with disastrous outcome. In the present case the organism was demonstrated at the last minute, after earlier stool examinations had been negative. Periodic barium enema studies are being carried out as a precautionary measure. We believe that in similar cases if there should be a residual defect after a full course of antiamebic treatment and multiple stool examinations were negative for ameba, exploratory celiotomy would be mandatory.

Summary

In a case of intestinal amebiasis with a granuloma of the cecum, diagnosis was made on stool examination, and exploratory operation was avoided. However, despite multiple studies, amebae were not demonstrated until late, barely in time to forestall operation which otherwise was indicated because carcinoma is difficult to distinguish from amebic granuloma in the absence of demonstrable organisms.

REFERENCES

1. Bockus, H.: Amebic Granuloma, Gastroenterology, Vol. II, 2nd ed., W. B. Saunders Co., Philadelphia, 1964, p. 904.

2. DeLorimier, A., Moehring, H. G., and Hannon, J. R.: Clinical Roentgenology, Vol. IV, Chas. C Thomas, Springfield, 1956.

3. Elsdon-Dew, R.: Factors Influencing the Pathogenicity of Endoameba Histolytica, Proc. World Congress of Gastroenterology, Williams and Wilkens Co., Baltimore, 1959, p. 770.

4. French, W. E.: Multiple amebic granulomas of the large intestine: Report of a Case, American Surg., 26: 457, 1960.

5. Gunn, H., and Howard, W. J.: Amebic granulomas of the large bowel, J.A.M.A., 97:166, 1931.

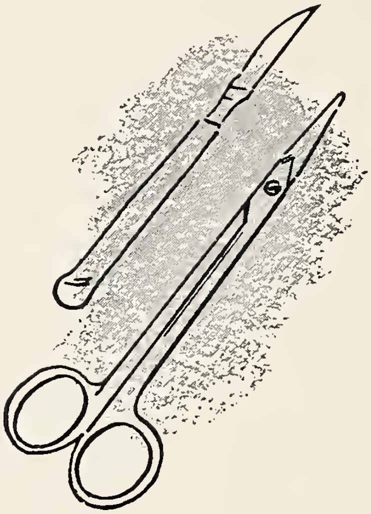
6. Kehoe, E. L.: Granuloma of the rectosigmoid producing bowel obstruction, Med. Ann. of the Dist. of Columbia, 32:405, 1963.

7. Radle, R. A.: Ameboma of the intestine, Ann. Int. Med., 43:1048, 1955.

8. Sodeman, W. A., and Engle, P. R.: Amebic granuloma of the colon, Am. J. Digest. Dis., 19:363, 1952.

9. Spicknell, C. G., and Pierce, E. E.: Amebic granuloma, New Engl. J. Med., 250:1055, 1954.

10. Yeomans, F. C.: Amebic granuloma simulating caricnoma of colon and rectum, Am. J. Surg., 31:365, 1936.



Hematologic Manifestations Of Tuberculosis

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California Medical Center, San Francisco. Taken from transcriptions, they are prepared by Drs. Martin J. Cline and Hibbard E. Williams, Assistant Professors of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine.

DR. SMITH*: The Medical Grand Rounds this morning are, as you notice, in the general area of infectious disease. The first case, that of Mr. A, will be presented by Dr. Terry Andrew.

DR. ANDREW*[1]: The patient, Mr. A, is a 50-year-old white married building contractor who came to the University Medical Center for evaluation of a fever of about seven months' duration. He was in good health until November 1965, when he had the first of a series of episodes of severe pain in the left upper quadrant of the abdomen, and nausea. These symptoms were followed in a few days by fever and generalized malaise. The patient described the pattern of the fever as follows: The episodes would last for approximately one week, and recurred at approximately four-week intervals with temperatures in the range of 38.3° to 40.6°C (101° to 105°F). He went to a physician in January 1966 because of persistence of these symptoms. At that time axillary and inguinal adenopathy were noted. Laboratory data included hemoglobin of 12.2 gm per 100 ml, accelerated sedimentation rate, increased concentration of serum gamma globulin, a positive tuberculin and negative coccidioidin skin test. In March 1966 the patient was operated upon for an incarcerated incisional hernia, and an exploratory laparotomy was performed with observations described as "within normal limits."

Following operation the patient felt well until May 1966, when fever recurred and was associated with night sweats. In June he was admitted to a hospital in Stockton for further evaluation. It was found that he had lost approximately 13 pounds since the onset of symptoms. Body temperature at the time of admission was 38.3°C (101°F). On physical examination hepatomegaly and lymphadenopathy were noted and there was some question of a left retroperitoneal mass. Laboratory data at that time included hemoglobin of 9.3 gm per 100 ml of blood and leukocytes numbering 1,450 per cu mm with 30 per cent polymorphonuclear cells and 68 per cent lymphocytes. Examination of aspirated bone marrow showed myeloid hyperplasia and erythroid hypoplasia. No cause could be found for the fever and the patient was referred to the University of California Hospital, San Francisco. At that time no history could be obtained of cough, pleurisy, hemoptysis or production of sputum. The family history was negative. The patient said he smoked approximately one pack of cigarettes a day.

On physical examination the only abnormalities noted were slight adenopathy in the inguinal and axillary regions, hepatomegaly and questionable splenomegaly. Hemoglobin content was 8.7 gm per

---

*Lloyd H. Smith, Jr., M.D., Professor of Medicine, Chairman, Department of Medicine.

*[1]Oliver T. Andrew, M.D., Resident in Medicine.

100 ml of blood, and leukocytes numbered 3,400 per cu mm—30 per cent polymorphonuclear cells, 65 per cent lymphocytes, 3 per cent monocytes and 2 per cent basophils. Platelet count was 301,000 per cu mm, with reticulocytes 2.1 per cent of the total. Results of urinalysis were within normal limits. Additional hematologic data included haptoglobin of 325 mg per 100 ml (normal), leukocyte alkaline phosphatase of 62 units (normal), prothrombin time of 40 per cent with a normal partial thromboplastin time, negative Sia water test and serum iron of 31 mcg per 100 ml, with a total iron-binding capacity of 230 mcg per 100 ml. The bone marrow at this time showed generalized hyperplasia, predominantly of the erythroid series.

Numerous bacteriologic cultures were taken of all available secretions and excretions from every orifice. These were all negative for acid-fast bacilli, fungi and ordinary pathogens. At the time of this admission, skin tests were positive with purified protein derivative, questionably positive for histoplasmosis, and negative for coccidiomycosis and blastomycosis. An x-ray film of the chest showed fibrocalcific densities. Additional x-ray studies showed degenerative disc disease in the cervical spine. An intravenous pyelogram, barium enema studies, an upper gastrointestinal tract series and small bowel follow through and abdominal lymphangiograms were all within normal limits.

Additional data obtained included a liver scan and liver biopsy, neither showing abnormality. A scalene lymph node biopsy showed multiple confluent and epithelioid caseous granulomas, whereupon administration of isoniazid and paraaminosalicylic acid was begun. The patient improved on this therapeutic regimen. Cultures of the scalene nerve specimen grew a few acid-fast organisms.

Dr. Smith: Dr. Youker will discuss the x-ray findings.

Dr. Youker*[2]: We saw no active pulmonary disease but there was evidence of old granulomatous disease. There are granulomas in the right upper lung field and some scattered granulomas in the right mid-lung field (Figure 1). The lateral film of the chest also showed these same granulomas.

Dr. Smith: This patient was referred to the hospital by Dr. Robert Talley of Stockton. I do not believe that he was able to be here today. We would like to present a second case which illustrates other aspects of the hematologic manifestations of tuberculosis. The summation will be given by Dr. Stuart Aaronson.

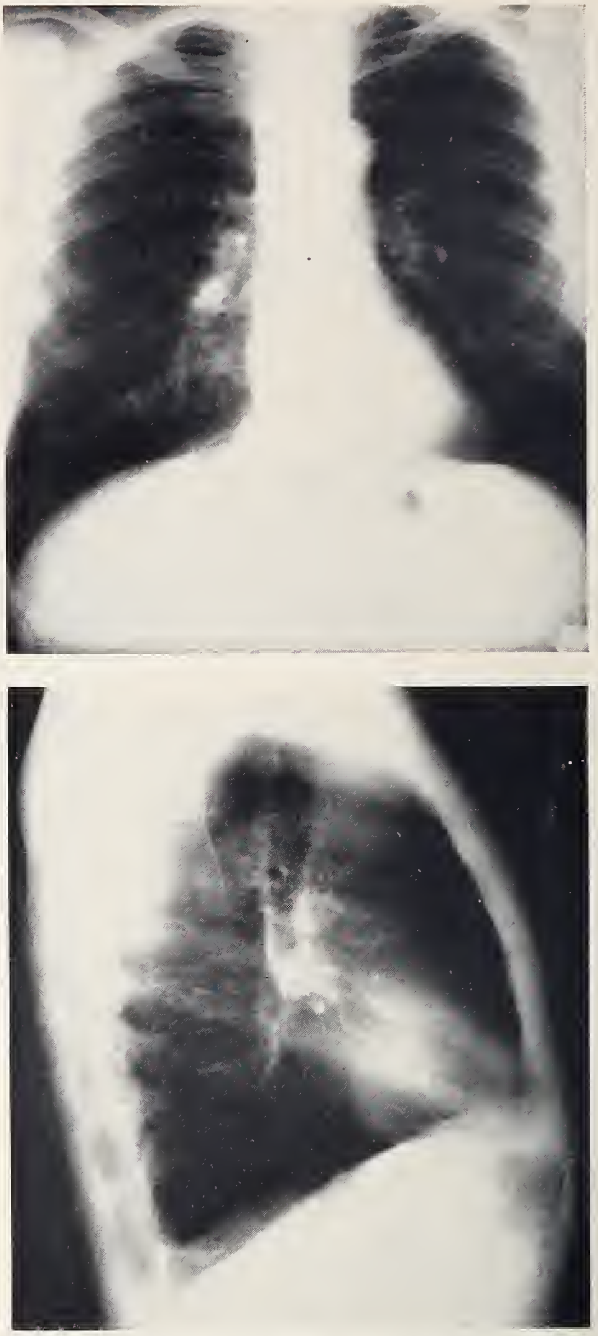


Figure 1.—*Upper*, x-ray film of lungs showing granulomas in upper right field and right mid-lung field. *Lower*, Lateral film of chest showing granulomas.

Dr. Aaronson*[3]: The patient, Mr. B, is a 62-year-old painter with chief complaint of tenderness in the left upper quadrant of the abdomen, with weight loss and weakness of five months' duration. A mass in the left upper quadrant had been noted on routine physical examination in November 1965. In March 1966, the patient first noticed anorexia, weight loss and weakness. In June 1966, anemia was noted by a physician, and in August 1966 he was admitted to the University of California Medical Center. The patient is an employee of the Medical Center and for the past two years has had x-ray films of the chest taken every six months. These films have shown stable apical infiltrates. He was also known to have a positive purified protein derivative skin test. The patient has smoked about two and a half packages of cigarettes a day for the past 45 years; he has had a chronic non-productive cough, and in 1964 had an acute bleeding ulcer which responded to medical management.

---

*[2] James E. Youker, M.D., Assistant Professor of Radiology.

*[3] Stuart Aaronson, M.D., Intern in Medicine.

On physical examination the patient was observed to be pale and cachectic. Body temperature was 38°C (100.4°F). Shotty lymphadenopathy was noted. The conjunctivae and mucous membranes were pale. Lung expansion was decreased bilaterally. The anteroposterior diameter of the chest was increased. There was dullness and increased fremitus on the right. Peristaltic movement was visible beneath a thin abdominal wall. The spleen, which was large and rock-hard, extended 8 cm below the costal margin, and there was a tender 2×3 cm nodule at the anterior margin. The liver edge was felt 4 cm below the right costal margin. It was nontender and firm. On examination of the extremities, early clubbing of the fingers and severe muscular wasting were noted.

Leukocytes numbered 16,000 per cu mm at the time of admission, with 60 per cent neutrophils and many immature cells, 10 per cent basophils and 10 per cent eosinophils. Packed cell volume was 19 per cent, the reticulocyte count 2.5 per cent of the total, and platelet count 80,000 per cu mm. Normoblasts were present in the peripheral blood.

X-ray examination revealed dense bones. Patchy apical infiltrates, which had increased since the previous examination, were noted on x-ray films of the chest. There was a large right pleural effusion. An intravenous pyelogram was within normal limits. A skin test with purified protein derivative was positive.

Thoracocentesis and pleural biopsy were performed. The latter revealed caseating granulomas. No intrabronchial lesions were seen at bronchoscopy. A bone biopsy was performed at the time of bronchoscopy when three previous marrow taps had been dry. The bone biopsy revealed sclerotic bone and myelofibrosis. Triple antituberculous therapy was begun and the patient was transferred to San Francisco County Hospital for continued treatment. Subsequently cultures for acid-fast bacilli of both sputum and pleural fluid were positive. The patient's condition has been stable at the county hospital.

Dr. Smith: Thank you very much, Dr. Aaronson. I have just been informed by the Radiology Department that with his transfer, the patient's films were also transferred, so we will not be seeing them this morning. With the two cases that have been summarized here as a starting point, there are many aspects of tuberculosis that could be presented. We have decided to concentrate on the hematological manifestations. I have asked Dr. Martin Cline to open the discussion.

Dr. Cline*[4]: Before embarking upon a general discussion of this subject, I would like to call attention to certain features of the clinical histories of these patients—in particular, the striking difference in the hematological manifestations. The first patient, A, had fever, anemia and severe leukopenia but with a relative and absolute increase in the lymphocyte count, when first observed. There were 60 to 70 per cent lymphocytes in the peripheral blood at a time when the number of leukocytes in the blood was in the range of 1,500 to 3,000 per cu mm. This observation is pertinent, in that Hodgkin's disease was considered in the differential diagnosis because it frequently produces adenopathy and a fever pattern of the type observed in this patient. A point against this diagnosis was the differential leukocyte count which would be extremely unusual in Hodgkin's disease where granulocytosis and lymphopenia are the rule. The clinical features in the second case were quite different, although this patient also had tuberculosis. He appeared to be a typical example of persons with myelofibrosis and myeloid metaplasia. He was cachectic, anemic, had a greatly enlarged liver and spleen and had dense bones. The initial radiologic reports suggested myelofibrosis. A peripheral blood smear (Figure 2) was characteristic of myeloid metaplasia, showing decided variation in the size and shape of the erythrocytes, the typical "tear drop" red cells and the

---

*[4] Martin J. Cline, M.D., Assistant Professor of Medicine.

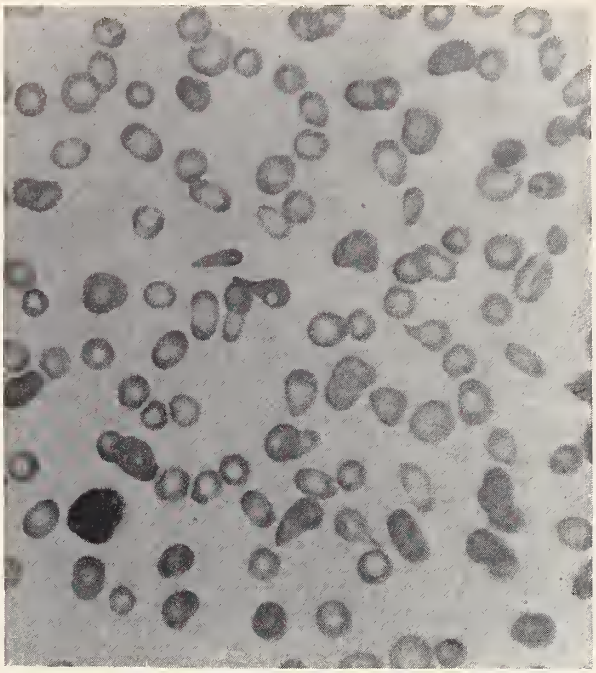


Figure 2.—Peripheral blood smear showing the teardrop forms and pronounced variation in size and shape of erythrocytes.

occasional fragmented or "helmet" cells also seen in this condition. Occasional giant platelets and nucleated red cells were also seen on the blood smear.

A pertinent feature of the history in this case is that two years before the patient was admitted to hospital he was seen by a reliable physician who did not observe either pallor or splenomegaly but seriously considered the diagnosis of active pulmonary tuberculosis. There is, then, indirect evidence that the tuberculous process preceded the hematologic manifestations in this patient.

Let us turn now to the hematologic manifestations of tuberculosis in those cases in which the hematologic changes are *not* the most prominent clinical feature (Table 1). I would like to extract two clinical pearls from that accumulation of statistics: (1) The incidence of various hematologic abnormalities is, with few exceptions, fairly low; (2) if the physician observes hemoglobin content of less than 10 gm per 100 ml of blood, he should look for extrapulmonary tuberculosis.

TABLE 1.—*Incidence of Various Hematologic Manifestations of Tuberculosis*

Abnormality	*Incidence (Per 100 Cases) in* *Pulmonary*	*Miliary*
Anemia	17	60-80
Leukocytosis	10	....
Leukopenia	10	....
Monocytosis	35	....
Lymphocytosis	4	....
Eosinophilia	15	....
Thrombocytopenia	rare	....

And now, on to a consideration of each of the formed elements of the blood. Anemia, as I have observed, is quite rare in large series of cases of pulmonary tuberculosis, occurring in less than 20 per cent of patients with this disease. It is, however, quite common in disseminated and miliary tuberculosis, and occurs in between 60 and 80 per cent of cases. Leukocytosis and leukopenia are equally uncommon in tuberculosis and lymphocytosis is quite rare. Eosinophilia occurs in about 15 per cent of cases and is more common in pulmonary tuberculosis than in extra-pulmonary tuberculosis. The second patient presented this morning did have eosinophilia. Thrombocytopenia has been reported but its frequency is unknown. A very interesting observation is that monocytosis occurs not infrequently in tuberculosis. The older phthisiologists used the differential leukocyte count in predicting the clinical course of tuberculosis. They considered a rising lymphocyte count and a falling monocyte count to be a sign of poor prognosis, and a rising monocyte count a sign of good prognosis. While such observations have not stood the test of time, I was fortunate enough to come upon an interesting reference in the American Review of Tuberculosis of 1929 which reinforces my impression that the lung serves merely as a bellows to aerate the blood and that the hematology service should be expanded to encompass pulmonary diseases as well.

> In connection with the above studies we have formed the impression that an extension of the pathological process in a tuberculous lung usually manifests itself first in the blood picture, later by physical signs and x-ray, and often last of all by symptoms. (Flinn, J. W., and Finn, R. S., Ann Rev. Tuberc., 20:347, 1929.)

I would draw this quotation to the attention of Dr. Charles Carman and the members of the Pulmonary Disease Service.

If we consider situations in which the hematologic manifestations of tuberculosis dominate the clinical picture, the disease syndromes are three in number: (1) Leukemoid reactions, (2) myelofibrosis and myeloid metaplasia, and (3) pancytopenia which resembles that of aplastic anemia.

I would like to consider each of these. First, the leukemoid reactions. They are extremely rare, occurring in less than 3 per cent of cases of disseminated tuberculosis. The peripheral blood findings and the clinical features may be exactly the same as those of chronic myelocytic leukemia or, rarely, acute myeloblastic leukemia. Auer rods have been reported in some of these cases. Tuberculous leukemoid reactions and leukemia may be differentiated by a marrow chromosome preparation, which in the case of leukemia, will reveal an abnormal karyotype pattern. I would stress that in cases in which tuberculosis is associated with the hematologic picture of acute leukemia, it is likely that both diseases are present and that tuberculosis is complicating acute leukemia. The documented instances of tuberculosis producing the entire disease picture of acute leukemia are extremely rare.

Tuberculosis may be associated with pancytopenia and aplastic anemia. I have given the reference to an excellent article by Medd and Hayhoe.[1] They described a disease entity, tuberculous miliary necrosis with pancytopenia. In this disorder there are rather distinctive pathologic features with widespread miliary necrosis and very little tissue reaction. An outstanding clinical symptom is a high, spiking fever which the older phthisiologists called the "typhoidal variant" of tuberculosis. Leukopenia and splenomegaly are present. Tuberculous miliary necrosis with pancytopenia is a quite rare disease but it is important to be aware of its existence and to differentiate it from aplastic anemia of other causes. The clue to the diagnosis may be splenic enlargement, which is, of course, quite rare in typical aplastic anemia.

Tuberculosis may occur in all the myeloproliferative disorders. In the very first reported case of polycythemia vera, described in 1892 by Rendu and Widal, the patient had pulmonary tuberculosis, as did the patient in the second case reported some seven years later. In all, more than a hundred cases of pulmonary tuberculosis and polycythemia vera have been reported. The association was sufficiently frequent that older clinicians considered that the lipids of the tubercle bacilli might have erythropoietic activity. This, of course, is unlikely. Of all the myeloproliferative syndromes, it is myelofibrosis and myeloid metaplasia that has the most intriguing association with tuberculosis. There are a number of reasonably documented cases in which the myloid metaplasia followed the onset of tuberculosis as in the case of Mr. A, discussed today. The following is the "traditional" list of the causes of myelosclerosis and myeloid metaplasia found in most of the major textbooks of medicine and hematology:

MYELOSCLEROSIS AND MYELOID METAPLASIA

1. Agnogenic
2. Metastatic malignant disease
3. Chemical or radiation-induced injury
4. Marble bone disease
5. ? associated with tuberculosis

The majority of the cases are of unknown cause. There are a few due to marrow replacement by metastatic malignant lesions, a few from chemical or drug-induced marrow injury (for example, chloramphenicol and benzene) or radiation injury. A very few cases are due to a genetic abnormality in which the bones are dense—marble bone disease. In each of these lists of myelofibrosis the association with tuberculosis is questioned. I have listed a reference to the first description of myelofibrosis and myeloid metaplasia in the American literature.[2] The patient in the case described had tuberculosis of the mesenteric lymph nodes. It is quite interesting to read a description of this case, since it parallels closely Dr. Aaronson's description of the physical examination in the second case reported here today, that of Mr. B. In both cases the liver was extremely large and quite tender to palpation; and in both there were visible peristaltic waves in the left upper quadrant of the abdomen.

From my review I would make one point: In patients with associated myelofibrosis and tuberculosis, treatment of the acid-fast disease rarely restores the blood to normal. I think this is important in the prognosis of these diseases.

What are the possible causes of the association of tuberculosis and blood diseases. I think there are four possibilities. The first is that the association of a blood disorder and tuberculosis may be purely coincidental. The second is that the blood disorder favors the development of tuberculosis. There is little evidence to support such a thesis. In *untreated* hematologic diseases, infectious complications are usually caused by pyogenic organisms. The exception to this general rule is Hodgkin's disease. I think it was Ewing who said that "tuberculosis follows Hodgkin's disease like a shadow." The point I wish to stress, however, is that in the untreated patient with hematologic disease, tuberculosis is relatively rare.

A third possible explanation of the association of acid-fast organisms and blood disease is that treatment results in increased susceptibility to tuberculosis. This may be a real possibility. Patients are treated with corticosteroids and with alkylating agents which may suppress the normal immunologic responses and impair host resistance.

The fourth and last possibility is that tuberculosis causes the blood disease. If I may be permitted to speculate, I would say there are four possible ways in which tuberculosis could cause a reduction in circulating blood cells. First, as with any chronic infection, there may be suppression of erythropoiesis. Chronic infections produce "chronic simple anemias." These anemias are certainly chronic, but I do not think they are quite so simple. We do not understand their pathogenesis although we can document certain obvious abnormalities of erythropoiesis. In association with the anemia of chronic infection there is usually low serum iron and low iron-binding capacity. The plasma iron turnover rate is rapid but there is poor utilization of iron for erythropoiesis. Suppression of erythropoiesis by chronic infection would therefore be one type of blood disorder caused by acid-fast organisms. Tuberculosis can produce hypersplenism, and this might be a second cause of cytopenia. In essence, work hypertrophy of the spleen produces a large spleen which can sequester and destroy blood elements. A third possibility is that there is actual replacement of the marrow with tubercles and the associated fibrotic reaction. I think there is little evidence to support such an idea. The fourth possibility has to do with the fact that the drugs used in the treatment of tuberculosis may produce hematologic manifestations. Of this there is no doubt. The three major drugs used in the treatment of tuberculosis, isoniazid, para-aminosalicylic acid and streptomycin, may all individually or collectively produce pancytopenia or decreases of individual blood elements.

With this background we use the following guidelines in the approach to the association of tuberculosis and hematological disorders. First, that the possibility of tuberculosis exists in any case of myelofibrosis and myeloid metaplasia, aplastic anemia or atypical or poorly characterized blood disorder. In such a setting, one must make a painstaking search for acid-fast organisms by cultures of gastric aspirates, sputum, bone marrow and urine. In addition, the physician may be forced to consider biopsy of an enlarged lymph node, or, failing this, a "blind" biopsy of the scalenus anticus node. This last procedure was done successfully in the first case presented this morning.

I would like to turn from these clinical considerations for a moment to consider an equally important and perhaps more interesting aspect of the association between tuberculosis and anemia—the historical significance. This historical significance is based on the fact that it was the tuberculous rather thin, pale and languorous female who provided much of the visual imagery for the literature of the early and mid-19th century. I have already mentioned that significant anemia is uncommon in pulmonary tuberculosis and common in the extrapulmonary form of the disease.

Armed with this fact, I decided to do some research into the literature of musical opera and made, I think, some interesting observations. I was surprised to find that Mimi in *La Boheme* and Violetta in *La Traviata* are both described by the heroes as being "pale and wan." I would submit that this is *prima facie* evidence that these unconventional women had disseminated tuberculosis rather than pulmonary tuberculosis. Unfortunately the heroes of both these operas were rather frivolous characters and did not follow up this important observation.

To support my arguments for this historical significance of tuberculosis and anemia, I would like to read a few excerpts from a very interesting book by the brothers Dubos.[3] They are writing of a party in the home of Edgar Allen Poe in 1842, and in describing his wife they make use of a borrowed description and comment upon it:

" 'Dressed in white, delicately, morbidly angelic, Virginia was singing and playing the harp in the glow of the lamplight. Suddenly she stopped, clutched her throat, and a wave of crimson blood ran down her breast.' Although accidents such as this caused much anguish to Poe, he looked on his wife's illness as a source of a strange additional charm, which rendered more ethereal her chalky pallor and her haunted liquid eyes."

To William Cullen Bryant as to many others, the death of a beautiful girl was the most poetic of all themes, especially "if she died in the fall of the year of some obscure, slow and wasting malady." However, it was not alone the langourous female in the early Nineteenth Century who "profited" by the association of tuberculosis and anemia; some of the male artists and poets of that time also felt that it added to their glamour. In conclusion, one more excerpt from Dubos frères to support this last argument: "Even the robust and sensuous Alexandre Dumas made occasional attempts to look frail and consumptive." [In 1823 and 1824] he writes in his memoirs, 'It was the fashion to suffer from the lungs. Everybody was consumptive, poets especially. It was good form to spit blood after each emotion, and to die before reaching the age of 30.' " Thank you.

DR. SMITH: Thank you Dr. Cline for spitting clinical pearls instead of blood before us this morning. We have time for some questions or comments. Dr. Carman, would you like equal time?

DR. CARMAN*[5]: Not now. Now that the incidence of the disease has fallen fairly sharply, I would emphasize the importance of recognizing the extrapulmonary manifestations of tuberculosis. This year we expect to have fewer than 300 new cases of tuberculosis found in San Francisco, a record low. Cases of the kind presented today are not an unusual occurrence in this hospital.

Perhaps the most important point to be made about both extrapulmonary and pulmonary tuberculosis is the importance of chemoprophylaxis in the patients over 50 years of age who have had previously untreated active or inactive tuberculosis. This is the source or reservoir from which more than 60 per cent of cases of active tuberculosis derive. Shortly the Public Health Service will officially recommend that all such patients be given chemoprophylaxis with isoniazid for a minimum period of one year. In answer to Dr. Cline's comment about the importance of the blood as compared with the pulmonary system in this disease, I would persist in looking at the lungs as the window of the soul.

REFERENCES

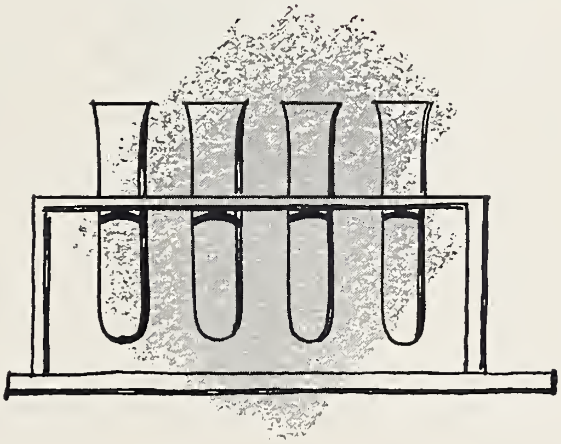
1. Medd, W. E., and Hayhoe, F. G. J.: Tuberculous miliary necrosis with pancytopenia, Quart. J. Med., 24: 351, 1955.

2. Donhauser, J. L.: The human spleen as haematoplastic organ, as exemplified in a case of splenomegaly with sclerosis of the bone-marrow, J. Exp. Med., 10:559, 1908.

3. Dubos, R., and J.: The White Plague, Little, Brown and Co., Boston, 1952.

---

*[5]Charles T. Carman, M.D., Associate Professor of Medicine.



California
Medicine

Editor
DWIGHT L. WILBUR, M.D.
•
Assistant to the Editor
ROBERT F. EDWARDS
For information on preparation of manuscript, see advertising page 2

Policy Committee—Editorial Board
JAMES C. MacLAGGAN, M.D., San Diego
JOHN G. MORRISON, M.D., San Leandro
WILLIAM F. QUINN, M.D., Los Angeles
JOSEPH W. TELFORD, M.D., San Diego
CARL E. ANDERSON, M.D., Santa Rosa
HELEN B. WEYRAUCH, M.D.
San Francisco
DWIGHT L. WILBUR, M.D., San Francisco

EDITORIAL

Health Care of the People

SINCE THEY ARE important in promoting the health of the people of this state, all factors bearing on the distribution of medical care and the conditions of medical practice are quite proper concerns of the California Medical Association and its component societies.

Those concerns bring into one focus a speech made by Dr. Charles L. Hudson, president of the American Medical Association, a report by the California Medical Association's Committee on Welfare Medical Programs which was accepted by the Council, and recent actions in three county medical associations having to do with the health care of segments of society needing medical attention and not knowing how to obtain it.

- Dr. Hudson, speaking before the AMA House of Delegates in November, said: "Government involvement . . . is less important in the form of Medicare as it is now than in the form it may take in the future. Our best efforts for containment will be to eliminate the areas where need for health care is apparent." The AMA President said further: "In directing our attention to the health needs of people, an occupation in which we are expert, we deal with primary factors first: How do we organize to provide service? Payment will follow."

- The CMA Committee on Welfare Medical Programs made a study of ways to delineate a relationship between the CMA and the Office of Economic Opportunity—particularly the medical aspects of OEO community action programs. In its report, the Committee said that whether the manner in which medical care is supplied under these programs is "one which the private practice of medicine can accept and relate to, depends to a large extent on the amount of guidance and leadership that county medical societies are willing to offer."

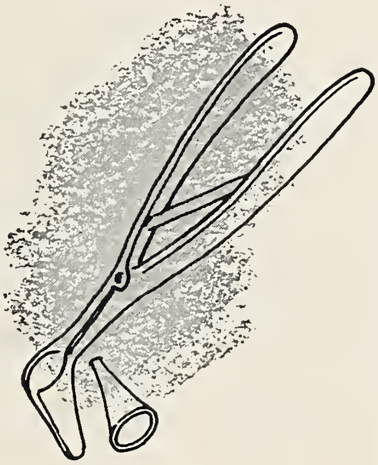
- In the Watts area of Los Angeles County, a comprehensive medical care program within the general framework of private practice has been worked out between the USC School of Medicine and the Office of Economic Opportunity.

- In southern Monterey County, the county medical society has supported an application by a private practice medical group to provide medical services for persons who are encompassed in an OEO program.

- In San Francisco the medical society and the dental society have entered into an agreement with the Economic Opportunity Council to set up a full scale neighborhood center which will supply health care and health education in poverty areas for a limited time until the persons using the center can be directed into the more conventional channels of private practice.

We believe, with Dr. Hudson and the CMA Committee on Welfare Medical Programs, that one of the purposes of a medical society is to find out where medical care is needed, what kind is needed and how to supply it. As to the people who need care and cannot pay for it, we believe that they and the continued advancement of medicine will be best served by finding ways to supply their needs within the structure of private practice. Of course, that very thing has been done informally for many years in many places, but where the needs of this kind have grown to such proportions that they cannot be met adequately without formal organization, it is a duty of county medical associations to take part in the formation and the shaping of whatever facility is needed. Should a county society come upon a situation in which a non-medical organization—a "community action committee"—is already at work on a legitimate problem concerned with the supplying of medical care, the society can do much for the community and the profession it represents, simply by joining in the effort, giving sound advice about subjects on which it is best informed and advocating the present and future advantages of providing care within the framework of private practice. Should a medical society itself find circumstances in which medical care is needed and those who need it cannot pay, it might well originate efforts to form whatever organization is needed to do the job and find ways to finance it.

To identify medical care needs of this kind, and to meet those needs within the structure of private practice, can be at once a boon to the health and dignity of all people, including those in need, and a lasting service to a system of practice that has brought about great advances in medicine.



California Medical Association

NOTICES AND REPORTS

Council Meeting Minutes

Tentative Draft: Minutes of the 528th Meeting of the Council, Los Angeles, Airport Marina Hotel, 15 January 1967.

The meeting was called to order by Chairman Anderson at the Airport Marina Hotel, on Sunday, 15 January 1967 at 8:45 a.m.

A quorum present and acting (full roll call, including names of invited guests appears in item 34).

1. *Minutes for Approval*

The minutes of the 527th meeting of the Council, held 10 December 1966, were approved as distributed.

2. *Health Manpower Council*

President-Elect Morrison reported that the Health Manpower Council had met recently in a planning session for a plenary session of the Manpower Council to be held the first part of February. Doctors MacLaggan, Melone and Morrison will represent CMA. Following the February meeting of the Health Manpower Council, the CMA Committee on Committees will have recommendations for permanent CMA representatives on the Council.

3. *Committee on Committees Recommendations*

Chairman Morrison announced that there will be an all-day session of the Committee on Committees on 28 January 1967. At its recent meeting, the Committee considered recommendations of the Finance Committee which were presented to the Council at the 10 December meeting. The Committee on Committees presented two recommendations with these results:

ACTION: Voted to authorize disbandment of the CMA-CPS Advisory Study Committee.

ACTION: Voted that joint meetings between the CMA Delegation to the AMA and the Council should be held only when deemed necessary by the chairman of each.

The following nominations to various CMA Committees and Commissions were presented:

Ad Hoc Committee on Libraries—Chester Tancredi, San Diego.

JAMES C. MacLAGGAN, M.D. . . . . President
JOHN G. MORRISON, M.D. . . . . President-Elect
WILLIAM F. QUINN, M.D. . . . . Speaker
JOSEPH W. TELFORD, M.D. . . . . Vice-Speaker
CARL E. ANDERSON, M.D. . . . Chairman of the Council
ALBERT G. MILLER, M.D. . . Vice-Chairman of the Council
HELEN B. WEYRAUCH, M.D. . . . . Secretary
DWIGHT L. WILBUR, M.D. . . . . Editor
HOWARD HASSARD . . . . Executive Director
General Office, 693 Sutter St., San Francisco 94102 • 415 776-9400
JACK CURLEY . . . . Southern California Office
1515 N. Vermont Ave., Los Angeles 90027 • 213 663-8071
RICHARD W. LEMOS . . . . Sacramento Office
1127 11th St., Sacramento 95814 • 916 444-7496

Scientific Board—

a. *Scientific section representatives*—John B. Dillon, Los Angeles; Edward J. Zaik, Los Angeles; Jerome Bettman, San Francisco; Vernon L. Nickel, Los Angeles; David Rubin, Los Angeles; and Justin J. Stein, Los Angeles.

b. *Specialty Group representatives*—Sidney Rosin, Beverly Hills; Henry B. Brainard, San Francisco; Charles Grayson, Sacramento; Charles J. Tupper, Davis; Keith P. Russell, Los Angeles; and Victor Richards, San Francisco.

ACTION: Voted approval of the above nominations.

4. *Finance Committee*

Chairman Kay presented a draft of the proposed 1967-68 fiscal year budget to the Council for study. Approval will be asked at the next meeting of the Council. Doctor Kay gave some explanation of the new format of the proposed budget. He also expressed the thanks of the Finance Committee to Mr. Lytton Hetland for his assistance in the preparation of the budget.

On behalf of the Finance Committee, Chairman Kay presented the following recommendations to the Council:

a. That the pre-convention issue of CALIFORNIA MEDICINE not include the full program for the Annual Scientific Assembly, but that it give a general preview of the highlights of the meeting. The full program would be presented in the official hand program.

b. That only three CMA staff persons attend the two annual AMA conventions.

c. That no funds be allocated for joint meetings of the CMA Council and the CMA delegation to the AMA.

d. That the allocation of funds for the addition of a health educator be postponed in the present proposed budget.

e. That the Council recommend to the House of Delegates that CMA authorize reimbursement to CMA Alternate Delegates to the AMA for only one of the two annual AMA conventions.

f. That the Council approve a joint liaison effort by CMA-Blue Shield to meet with California Congressmen and Senators in Washington, D.C. (for which funds are available).

Only the latter two recommendations were voted upon, with the following results:

ACTION: The recommendation to reimburse CMA Alternate Delegates for only one of the two AMA annual conventions did not pass, thus continuing present CMA policy.

ACTION: Voted to approve the CMA-Blue Shield joint liaison effort with California legislators in Washington, D.C.

5. *Resolution No. 105-66, Medical Laboratory Study*

As Chairman of the Commission on Community Health Services, Councilor Kay asked for guidance regarding further action on Resolution No. 105-66, which concerns a joint study of current medical laboratory practices in California. Doctor Kay stated that attempts had been made to undertake such a study with other interested groups, but due to lack of response from the other groups, no action had been taken. He pointed out that such a study had been conducted in 1964, and it was the opinion of the Commission that the statement evolving from that study was still valid.

ACTION: Voted to reaffirm the 1964 policy statement previously approved by the Council (Published in* California Medicine, *100:303, April 1964).

It was suggested that the Commission continue its efforts to develop policies regarding laboratory practices with pathologists and other interested groups.

ACTION: Voted to authorize the Commission to continue cooperative efforts to study laboratory services in California.

6. *Physicians for Rural Areas*

Councilor Kay introduced Leopold J. Snyder, M.D., chairman of the CMA Committee on Rural Health, who reviewed for the Council the background of the Committee's efforts to help solve the problem of recruiting adequate health care personnel for rural areas. He said that several meetings had been held, and proposed a state-wide meeting to explore the situation. Doctor Snyder presented to the Council the proposed format for such a meeting, which would bring together the many allied health organizations that share a common interest in the problem.

ACTION: Voted to approve the concept of CMA's sponsoring a state-wide meeting to explore the problem of recruiting adequate health care personnel for rural areas.

After some discussion it was decided that the Commission on Community Health Services and its Committee on Rural Health should work in conjunction with the Commission on Allied Health Professions and Services and its committees (particularly the Committee on Paramedical Personnel) to avoid duplication of effort in this area.

7. *Commission on Hospital Affairs*

Chairman Halter reviewed for the Council the many activities in which the Commission has been involved during the past year. He said that the Commission worked to establish suggested *Guidelines for Utilization Review,* has participated in initial discussion on the advisability of entering into agreement to participate in a pilot study, through data processing, on various facets of hospital planning; has participated in preparing *Proposed Staffing in County Hospitals;* and negotiated with the State Department of Public Health regarding the feasibility of having CMA Medical Staff Survey teams inspect medical staffs in non-accredited hospitals for possible certification under P.L. 89-97. Doctor Halter reported that five resolutions had been assigned to the Commission, two of which required action. Recommendations on Resolution No. 1-66 (Standardization of Hospital Records) and Resolution No. 104-66 (Physician's Compensation for Administrative Requirements) will be presented by the Commission at the next meeting of the Council.

Chairman Halter stated that the Committee on Health Facilities Planning, under the leadership of Doctor Glenn Pope, has been very active and is in the process of producing a brochure on health facilities planning to stimulate interest and to educate physicians on the need for voluntary planning. The Committee also has kept abreast of the "CHIPS" Program and its implementation in Santa Clara County, and has followed closely the activities of the "543" Committee.

In reviewing the activities of the Medical Staff Survey Committee, Doctor Halter informed the Council that the Committee had surveyed 110 medical staffs in the last eight months, making the total number of staffs surveyed 371. He added that there are 40 projected surveys scheduled through March of this year.

8. *Committee on Legislation*

Chairman Kilroy reported to the Council on the effects of Proposition 1-A and said that, because the proposition provides for an annual session of the Legislature, CMA will probably have to expend considerably more time and effort in this area.

Chairman Anderson read a letter sent by the California Psychiatric Association, urging its members to join and support the Public Health League. Doctor Anderson commented that this was a fine indication of support and that CMA might discuss this type of endorsement with other specialty societies.

9. *L. Henry Garland Memorial Lecture*

Chairman Anderson brought before the Council a request submitted to the Scientific Board from the California Radiological Society for an annual lecture to be held in conjunction with the CMA Annual Scientific Assembly, in memory of Doctor L. Henry Garland. The lecture would be sponsored by the California Radiological Society each year, and if approved, would be held this year on Sunday, 16 April, at 2:00 p.m.

ACTION: Voted to approve the L. Henry Garland memorial lecture to be held at CMA Annual Sessions.

10. *Requests for Sponsorship of Meetings*

Chairman Anderson brought before the Council a request from Mr. Clinton Fair of the California Labor Federation, AFL-CIO, for CMA co-sponsorship of a Conference on Rehabilitation to be held 17-18 February in Sacramento.

ACTION: Voted to participate with the California Labor Federation in this effort as a cooperating organization.

Another request was made by the California Steering Committee on Arthritis and Rheumatism for cooperation in a state-wide conference on Rheumatoid Arthritis, to be held 21-23 April in Sacramento.

ACTION: Voted to encourage the California Steering Committee on Arthritis and Rheumatism to hold the conference.

It was suggested that, due to the number of requests received by CMA for co-sponsorship, cooperation and participation in various conferences, review should be made to establish current policy of CMA for the handling of such requests.

ACTION: Voted that the Scientific Board be designated to update the guidelines for establishing policy on requests to CMA for participation in joint efforts with other organizations.

11. *Smoking and Health*

A request from the San Diego County Council on Smoking and Health was brought before the

Council, asking CMA to endorse the policy that cigarettes not be sold in hospitals. The request also asked that CMA recommend to AMA that it seek sponsoring membership on the National Interagency Council on Smoking and Health.

ACTION: Voted to defer action.

12. *Status Report on 1966 House of Delegates Resolutions*

Chairman Anderson reviewed with the Council the "Status Report on 1966 House of Delegates Resolutions," which Council members had received for study at the 10 December 1966 meeting. Several additions and corrections were discussed. The Council agreed to distribute the Report, incorporating suggested amendments, to CMA Delegates and Alternates.

13. *Treatment of Workmen on UC Campuses*

On behalf of the Commission on Public Agencies and the Committee on Occupational Health and Rehabilitation, Vice-Chairman of the Council Miller presented a recommended policy statement regarding medical treatment for on-campus contract construction workers. The statement recommended "approval of industrial medical services on a university campus, but with the limitation that these services not be extended. In essence, they should be limited to major emergency care, and if private medical care is available, the university should not compete." Councilor Miller stated that, if approved, the statement should be transmitted through proper university channels to the various student health centers.

ACTION: Voted to approve the above statement on treatment of workmen on U.C. campuses.

14. *Utilization Review in Extended Care Facilities*

Councilor Kaiser, chairman of the recently established Subcommittee on Utilization Review in Extended Care Facilities, briefly discussed the activities of his subcommittee to date. He directed the attention of the Council to the letter and supporting materials which the subcommittee had sent to all presidents, secretaries and medical executives of component societies. In addition to explaining CMA's role and policies in this area, the letter urged physicians to become involved and provide leadership at the local level in providing utilization review in extended care facilities.

Accompanying the letter were a list of extended care facilities in California that had been certified by the Social Security Administration as of 30 December 1966, and 11 sample utilization review plans.

15. *Statement on Medi-Cal Potential Deficit*

Following discussion regarding anticipated fiscal problems under the State's Medi-Cal program, the following policy statement (as amended) was presented:

"The Council of the California Medical Association is profoundly concerned by the potential deficit of the Medi-Cal program as reported by Mr. Spencer Williams, Administrator of the State Health and Welfare Agency. It is the understanding of the Council that the anticipated deficit in this program is not due to over-spending the budgeted amounts for physicians' fees or hospital care but is primarily attributed to a greater than anticipated use of nursing home facilities and an unforeseen opinion of the State Attorney General which holds that certain funds expended by counties for health care must be charged against the Medi-Cal program.

"The Council believes that government financed health programs can provide the high quality of health care expected by the public to which patients are entitled, only if these programs are adequately funded. The Council offers the cooperation of the California Medical Association to the State Health and Welfare Agency and proposes the following three recommendations to overcome any excessive utilization of nursing homes:

"1. That no patient be admitted to a nursing home except on the direct order of a physician.

"2. That after a Medi-Cal patient has been in a nursing home for an appropriate period of time, but not longer than 21 days, his condition be medically reviewed and authorization for his further care in the nursing home be obtained from the county Medi-Cal medical consultant for a continuing stay.

"3. That a medical review of those patients now in nursing homes be made by a utilization committee to determine the medical necessity of each patient for continued stay.

"The Council of the California Medical Association believes that implementation of the above three recommendations will contribute significantly to the fiscal soundness of the Medi-Cal program and it urges the state to continue making available sufficient funds to accomplish the intent of this excellent program."

ACTION: Voted to approve the three recommendations for helping to curb any excessive utilization of nursing homes under the Medi-Cal program (as embodied in the above statement).

16. *Amendment to Statement on Compensation For Physicians' Services*

The Council received and discussed a proposed amendment to its policy statement on "Compensation for Physicians' Services" (adopted by the Council on 7 May 1966). The suggested change, requested by the California Society of Anesthesiologists, was in the form of an additional qualifying phrase. Below is an excerpted paragraph from the policy statement, with the suggested addition underlined:

"Long-accepted traditional, ethical and legal determinations indicate that all medical services provided by a physician for patients, whether individually performed by him or provided by him and under his supervision (such as the services of nurses, assistants, technologists *not involving medical decisions*) constitute an integral part of the physician's professional service in the practice of medicine."

ACTION: Voted to approve the suggested amendment, but to inform the California Society of Anesthesiologists that the action was taken to clarify the statement (which has particular reference to pathologists), **not** ***to change in any way the ethical statements adopted by the AMA, which emphasize that anesthesiology is a medical service.***

17. *Assistance to Societies without Executive Staff*

In the absence of Mr. Reuben Dalbec, chairman of the Medical Executives Conference, Doctor Anderson directed the attention of the Council to a survey regarding methods for assisting component medical societies without executive staff. The report was referred to the Medical Executives Conference and the CMA staff for further study and recommendations.

18. *Planning and Goal Conference in Continuing Education*

Doctor John Dillon presented to the Council for its information the proposed program for the CMA Planning and Goal Conference in Continuing Medical Education to be held 11-12 March 1967, in San Diego. The Conference is designed to try to develop an overall plan for the future direction of continuing medical education in California.

19. *Report on the 1967 Conference of Component Society Officers*

Doctor Dale Ritter, chairman of the program committee for the conference, reported that, in spite of the fog, there was a near-record attendance at the conference, with 309 participants. Doctor Ritter expressed his thanks to the committee and all those who helped with the conference. Doctor Morrison took this opportunity to suggest that the Council take steps to organize a meeting for the officers of component societies in conjunction with the Annual Session. He said he felt such a meeting would serve an important function in giving special recognition to these officers. It was decided to leave the implementation of the suggestion to the Speaker and Vice-Speaker of the House of Delegates.

20. *Extension of State Alcoholism Control Program*

Councilor Miller, chairman of the Commission on Public Agencies, brought before the Council the request of the State Department of Public Health that CMA endorse Senate Bill 25 (introduced 4 January 1967, by Senator J. Eugene McAteer). The bill relates to the continuation of the program sponsored by the Division of Alcoholism of the State Department of Public Health. Discussion followed.

ACTION: Voted to reaffirm CMA's position (House of Delegates resolution No. 30-66) favoring continuation of the State alcoholism control program and to offer the assistance of CMA in its reasonable implementation.

21. *Announcement of Doctor Dwight L. Wilbur's Candidacy for AMA President-Elect*

President MacLaggan read a proposed draft of a letter announcing the candidacy of Doctor Dwight L. Wilbur, currently a member of the AMA Board of Trustees, for the position of President-Elect of the American Medical Association.

ACTION: Voted to approve the letter (with amendment) for distribution to AMA Delegates and Alternates.

22. *AMA Board of Trustees*

Doctor Dwight L. Wilbur reviewed his recent activities on the National Advisory Commission on Health Manpower, and urged that CMA assume a major role in studying the health care system of the future. He said that much effort is needed in this area, and that CMA participation on the Health

Manpower Council should be supplemented by CMA's concerted evaluation and study of the problem with suggestions for solutions.

ACTION: Voted to approve active CMA participation in health manpower activities.

23. *Audio-Digest Foundation*

The Council recessed to hold the annual meeting of the administrative members of Audio-Digest Foundation, Doctor John Morrison presiding.

Vice-President and Editor Claron Oakley reported on the editorial progress of the organization and stated that a written report of the past five years' progress would be available by Annual Session. He expressed thanks to the CMA staff for its assistance in publicity for the Foundation. He informed the Council that there are over 4,000 California physician subscribers, out of a total of 15,000 subscribers. The renewal rate is approximately 77 per cent. Mr. Oakley commented on two innovations that may be tried by the Audio-Digest Foundation: The use of cartridge tapes for automobiles, recording six short courses in one package, which would be sold separately from the regular subscriptions; and the offering of subsidized subscriptions to medical schools in order to familiarize medical students with Audio-Digest before they leave medical school and start into practice.

Treasurer K. L. Hamman announced that as of November 1966, the Foundation had passed the $1 million mark for number of subscriptions sold in the previous 12 months. He stated that since 1960, the percentage of subscribers had doubled, in relation to the physicians in private practice in the specialties covered, and that of those who subscribed for at least three years, the renewal rate was 93 per cent. Mr. Hamman reported that as of 31 December 1966, Audio-Digest had contributed $309,750 to medical education. This past year, $110,000 was contributed and divided between AMA-ERF and CMERF.

The administrative members present complimented both Mr. Hamman and Mr. Oakley for the excellent work done during the past year.

Doctor John Morrison, chairman of the CMA Committee on Committees, offered a list of nominees for the Audio-Digest Board of Directors for 1967-68. These were: Doctors Gordon Beckner, Inglewood; William D. Evans, Los Angeles; Elmer F. Gooel, Beverly Hills; George C. Griffith, Los Angeles; Merlin A. Hendrickson, Rialto; Donald D. Lum, Alameda; Robert L. Marsh, Glendale; John W. Pender, Palo Alto; Keith P. Russell, Los Angeles; Edward B. Shaw, San Francisco; and Messrs. Claron Oakley, Los Angeles; K. L. Hamman, Oakland, and Jerry Pettis, Los Angeles.

ACTION: Voted to accept the list of nominees as submitted.

24. *Blue Shield-California Physicians' Service*

In the absence of the Blue Shield Board Chairman Richard Wilbur, M.D., Thomas C. Paton, president, reported to the Council. He stated that as of the end of the year, Blue Shield standard programs were in a strong financial position, even though underwriting losses had been experienced earlier in the year. Under government programs ten million Medi-Cal claims had been paid since 1 March, totaling about $116,000,000. About 40 to 45 per cent of this went to physicians. Under Medicare since 1 July approximately 600,000 claims, totaling about $13,000,000 had been processed.

Mr. Paton also said that utilization activities are being strengthened through additional internal activities, plus more sophisticated computer techniques. Lists of physicians submitted by various county medical societies, plus others who are selected by computer techniques, are undergoing special comprehensive audits. Simultaneously, additional studies of utilization habits of other vendors such as druggists, hearing aid dispensers, and the like, are underway.

Mr. Paton reported that the claims function within the Los Angeles office is being expanded and improved. Fifty-five new people have recently been hired, and in the near future it is anticipated that some keypunch activities, and perhaps a satellite computer will be added to the Los Angeles function.

Beginning 1 April, California Blue Shield will offer its new Major Medicare program supplementing the Federal Medicare plan. This will be aggressively supported by a promotional campaign, which also will include information that those who are not participants under Title XVIII can obtain Blue Shield coverage.

In other marketing activities, Mr. Paton reported that a new program has been developed for small groups with four to nine persons. In addition, the program offered to groups with 10 to 24 persons has been improved and a Major Medical program added.

With respect to claims handling under the Medicare program, Chairman Anderson pointed out, and Mr. Paton concurred, that it takes much more clerical work to process an unassigned claim than it does to process an assigned claim. This results primarily from the fact that on unassigned claims the doctor's license number is frequently missing and the RVS Procedure Code is sometimes lacking. To research and insert such information slows down the clerical process considerably. Chairman Anderson suggested that CMA inform California physicians of these problems so that faster claims handling might result.

25. *Woman's Auxiliary to the CMA*

Mrs. Samuel K. Bacon, president of the Auxiliary, reported that membership is now almost 10,000. She also informed the Council that the California Auxiliary has developed a new GEMS (Good Emergency Mother Substitutes) manual, which the national Auxiliary has requested permission to use as its teaching manual.

26. *California Hospital Association*

President Henry X. Jackson commented on Medicare reimbursement. He stated that a program, initiated by the California Hospital Association and later adopted by the American Hospital Association is analyzing the cost of providing care to patients 65 and over. The analysis has been ongoing in six regions of the United States and from the results so far, it was demonstrated that the cost of providing care for patients 65 and over is at least 25 per cent more expensive than providing care for patients under 65.

27. *California Nurses Association*

Mrs. Mary K. Stanley, president of the Association, thanked the CMA for inviting her to participate in the Conference of Component Society Officers. Mrs. Stanley also reported that CNA is very much in favor of the Health Manpower Council and has set up task forces to gather information that might prove helpful to the Manpower Council.

28. *University of California, Davis, School of Medicine*

Dean Tupper brought the Council up to date on the progress being made at Davis and said that the school had been primarily concerned with three major areas—curriculum design, faculty recruitment and building planning and construction. Doctor Tupper reported that one building is completed and occupied, and another is underway. A "curriculum retreat" is planned which will include as participants representatives of many schools in California and across the nation, and from this, the faculty at Davis hopes to gain knowledge of the best possible curriculum available. A four-year residency program in general surgery has been approved at the Sacramento County Hospital to supplement the existing residency program in general practice.

29. *UCLA School of Medicine*

Associate Dean John Dillon informed the Council that UCLA School of Medicine had decided to hold the incoming enrollment to a total of 96 students this year, rather than increase it to 128 as had been stated previously. Doctor Dillon said that this was partially due to budgetary considerations, but also involved the great current physical expansion. He said it was felt by the Chancellor that increasing the class number this year might jeopardize the quality of education of the students. Councilor Todd asked for the number of medical students graduating from each medical school in California, as well as the number of students admitted in schools just starting and dates when first classes will graduate. This information was not readily available and will be supplied to the Council.

30. *State Department of Public Health*

Director Lester Breslow reported that the Department had recommended more than 600 facilities to the Social Security Administration for certification as extended care facilities in California under Medicare. Of these 600 facilities, 440 already have been certified, which makes available more than 30,000 beds. With the certification of the rest of the facilities recommended, the total number of beds will reach more than 40,000 in extended care facilities. Doctor Breslow also stated that, by way of comparison, California has had certified more than twice as many facilities as any other state. Discussion followed on newly proposed regulations for nursing home utilization under Medi-Cal.

31. *State Department of Mental Hygiene*

Doctor Anthony Toto, representing the director of the Department, reviewed the current waiting list for institutional care. He stated that although the total number of applicants was 1,566, under the revised classification of applicants undertaken last July, only 806 of these were considered to need truly institutional care (24-hour nursing care under continuing close medical supervision). He reported that 171 of those on the waiting list were reasonable candidates for nursing home care, and the other 589 were considered to need some other sort of facility—neither institutional nor nursing home care.

32. *Department of Rehabilitation*

Director Richard Young stated that he would like to see the "Medical Advisory Committee" to the Department of Rehabilitation expanded and that any persons the Council might like to suggest for membership on the committee would be given consideration.

ACTION: Voted to recommend to the State Department of Rehabilitation that its "Medical Advisory Committee" be expanded and reactivated.

Considerable discussion ensued regarding the role of the State Department of Rehabilitation, rehabilitation centers and prevalent concepts of rehabilitation.

33. *Membership*

Four applicants were voted election to Associate Membership. These were: Oliver Stoddard Hayward, Morton Nelson, Robert Douglas Roller, III, Alameda-Contra Costa County; Morris M. Rubin, Riverside County.

Five members were voted election to Retired Membership. These were: Ernest O. Gunderson, Alameda-Contra Costa County; Ralph L. Schroeder, Butte-Glenn County; Robert W. Vance, San Diego County; Harvey F. Hendrickson, San Mateo County; Harry J. Arnold, Santa Clara County.

Reduction of dues was voted for seven members for reasons of prolonged illness or postgraduate education.

34. *Roll Call*

Present were President MacLaggan, President-Elect Morrison, Speaker Quinn, Vice Speaker Telford, Secretary Weyrauch, and Councilors Dwight L. Wilbur, Isenhour, Eastman, Melone, Todd, Gooel, Taw, Bullock, O'Connor, Ham, Rogers, Crum, Boyle, Maguire, Burnett, Miller, Watts, Fenlon, Kay, Kaiser, Anderson, Yant, Grunigen, and Immediate Past President Teall. Absent for cause were Councilors Richard S. Wilbur and Longmire.

Present by invitation were CMA staff members Becker, Belser, Bowman, Clark, E. Collins, J. Collins, Curley, Eberlein, Goldman, Griffith, Hetland, Jones, Klutch, Lemos, Miller, Price, Redfern, Thomas and Whelan; Messrs. Hassard and Huber, legal counsel; component society executives Scheuber of Alameda-Contra Costa, Rideout of Butte-Glenn, Garrick of Forty First, Geisert of Kern, Baker of Los Angeles, Walters of Riverside, Donmyer of San Bernardino, Nute of San Diego, Neick of San Francisco, Wood of San Mateo, Donovan of Santa Clara, Brown of Sonoma and Whitehall of Stanislaus; Messrs. Paton, Babb, Heller, Koch, L'Heureaux, Light, Potloff, and Purdy of Blue Shield-California Physicians' Service; Messrs. Read and Brown of the Public Health League; Doctor Dillon of UCLA School of Medicine; Doctor Tupper of UC-Davis; Doctor Breslow of the State Department of Public Health; Doctor Young of the State Department of Rehabilitation; Doctor Toto of the State Department of Mental Hygiene; Mr. Jackson of the California Hospital Association; Mrs. Bacon of the Woman's Auxiliary to the CMA; Mrs. Stanley of the California Nurses Association; Mrs. Fitzgerald of the California Medical Assistants Association; Mr. Gould of the American Medical Association; Messrs. Hamman and Oakley of the Audio-Digest Foundation; Doctors Besson, Doyle, Harrington, Halter, Howard, Kilroy, Ritter, and others.

35. *Adjournment*

The meeting was adjourned at 4:15 p.m.

CARL E. ANDERSON, M.D., *Chairman*
HELEN B. WEYRAUCH, *Secretary*

In Memoriam

EDWIN L. BRUCK, M.D.

The recent death* of Edwin L. Bruck terminated the career of one of California's most distinguished and devoted physicians.

A leader in his chosen field of internal medicine, he was a prime mover in the formation of the San Francisco and the California Societies of Internal Medicine. Despite the heavy load of an active teaching assignment and the demands of a large practice he found time to participate in the work of organized medicine.

He served as a member of the Board of Directors of the San Francisco Medical Society and as its President in 1939. He was a member of the Council of the California Medical Association from 1942 to 1949, and Chairman of the Council from 1946 to 1949.

Ed Bruck was dedicated to the provision of medical care of high standard for the people of California. He was uncompromising in his opposition to any influence which threatened its excellence.

He was vigorous and effective in the struggle to prevent the socialization of medicine in California in the era of 1938 to 1950. A medical statesman of the first order and not content only to oppose, he was one of the architects of the constructive policies of the California Medical Association from the formation of California Physicians' Service through the campaign to bring medical care more easily within the reach of our people by the wide expansion of voluntary health insurance.

Ed was a critic of the situation then existing in the American Medical Association which allowed essentially one man control and was the author of the California resolution that ultimately resulted in returning control of that body to its elected representatives. He was one those who initiated the policies which made the American Medical Association better able to withstand the challenge of 1948 to 1952.

In these sound, realistic and forward-looking activities he modestly kept himself in the background. He rejected repeated efforts to make him President of the California Medical Association and to project him into the national picture.

Ed was a Californian in every sense of the word. He was born on the Krug ranch near St. Helena in 1893. He was a descendant of Doctor Edward Turner Bale, an English ship's surgeon, whose vessel was wrecked near Monterey in 1837, and who practiced medicine briefly there. Doctor Bale married Maria Ignacia Soberanes, a niece of General Vallejo, and a member of a Spanish Grant family. Subsequently he was awarded a grant in the Napa Valley by Governor Juan Alvarado. Ed's paternal grandfather, Louis Bruck, who came to the United States with Charles Krug, was one of the early vintners in the Napa Valley. His father, Bismarck Bruck, served with distinction in the California Legislature as well as carrying on the Krug tradition.

Ed received his education at the University of California and was awarded his degree in Medicine in 1920. His graduate training in internal medicine was in the University Hospital. After a period of study in Europe, he became associated in practice with the late Herbert C. Moffitt and was one of the last of the splendid group of men trained by that illustrious teacher of medicine.

He was active on the faculty of the University of California until he became Clinical Professor of Medicine, Emeritus. Most of his teaching was done on the California Service of the San Francisco Hospital. He was respected, admired and beloved by his students and house staff because of his diagnostic acumen, his broad knowledge of medicine, his ability to impart that knowledge to others and to apply it to patient care.

He made contributions to medical knowledge and to the medical literature. Perhaps one of the most significant was in the treatment of pneumonia in the period before the advent of chemotherapy and antibiotics when pneumonia was less easily manageable.

Ed served as Chief of Medicine and Chief of Staff of St. Luke's Hospital, where he gave liberally of his talents. His services as a consultant were widely sought over Northern California. As one of those who benefited from them phrased it, "When the going is rough, Ed Bruck is a tower of strength."

Those who knew him well—his colleagues, patients and friends—regarded him with respect and affection as an individual as well as a physician. His intelligence and incisive mind could brush aside inconsequential matters, go directly to the heart of a problem and reach a rapid but thoughtful decision. His own forthrightness made him intolerant of sham and subterfuge.

Ed had a delightful sense of humor and an articulate and pungent way of expressing his thoughts. These and his capacity for warm friendship made him a delightful companion.

He loved the out of doors. He was a yachtsman, a hunter and a fisherman. One could feel closest to Ed in the isolation and quiet of a duck blind.

To those who knew him best his outstanding characteristic was courage. This was apparent in his entire life but was most evident in the two serious and protracted illnesses he mastered to return to the practice of medicine. Even during these he could laugh and make light of his predicament.

---

*Dr. Bruck died 5 January 1967.

His sometimes brusque exterior clothed a kindness and concern for others. None know this as well as the patients whom his skill and devotion carried through serious illnesses.

It is rare to encounter an individual with the many talents and fine characteristics of Ed Bruck. His colleagues, friends and patients mourn his passing.

Our utmost sympathy is extended to his widow, Ellen, and to his children, Ann, Kergan and Meredith.

John W. Cline, M.D.

Blondin, Edward A., La Mesa. Died 25 January 1967, in Laguna Hills, aged 75. Graduate of Washington University School of Medicine, St. Louis, Missouri, 1919. Licensed in California in 1922. Doctor Blondin was a retired member of the San Diego County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

◆

Craycroft, Charles Burr, San Jose. Died 2 February 1967, in San Jose, aged 52. Graduate of Stanford University School of Medicine, Palo Alto-San Francisco, 1940. Licensed in California in 1940. Doctor Craycroft was a member of the Santa Clara County Medical Society.

◆

Dale, Philip M., Los Angeles. Died 29 January 1967, in Los Angeles, aged 83. Graduate of Rush Medical College, Chicago, Illinois, 1912. Licensed in California in 1924. Doctor Dale was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

◆

Gattuccio, Bartholomew, San Jose. Died 21 January 1967, in San Jose, aged 73. Graduate of Hahneman Medical College of the Pacific, San Francisco, 1916. Licensed in California in 1916. Doctor Gattuccio was a retired member of the Santa Clara County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

◆

Greene, Clarence Orville, Lodi. Died 2 January 1967, aged 53, of heart disease. Graduate of the University of Louisville School of Medicine, Kentucky, 1948. Licensed in California in 1950. Doctor Greene was a member of the San Joaquin County Medical Society.

◆

Hilgesen, James, Campbell. Died 16 January 1967, in Palo Alto, aged 70, of bilateral bronchopneumonitis. Graduate of the University of California School of Medicine, Berkeley-San Francisco, 1927. Licensed in California in 1927. Doctor Hilgesen was a retired member of the Alameda-Contra Costa Medical Association and the California Medical Association, and an associate member of the American Medical Association.

◆

Hollingsworth, Loren D., Laguna Beach. Died 26 December 1966, in Laguna Beach, aged 84, of cerebral thrombosis. Graduate of the College of Medicine of the University of Southern California, Los Angeles, 1905. Licensed in California in 1912. Doctor Hollingsworth was a member of the Orange County Medical Association.

◆

McRae, Colin Christopher, San Francisco. Died 6 February 1967, in San Francisco, aged 64. Graduate of the University of Michigan Medical School, Ann Arbor, 1927. Licensed in California in 1932. Doctor McRae was a member of the San Francisco Medical Society.

◆

Medoff, Murray H., North Hollywood. Died 24 January 1967, aged 43, of coronary thrombosis. Graduate of Kansas City College of Osteopathy and Surgery, Kansas City, Missouri, 1950. Licensed in California in 1952. M.D. degree from California College of Medicine, 1962. Doctor Medoff was a member of the Los Angeles County Medical Association.

◆

Murphy, William K., Veterans Home. Died 6 February 1967, at the California Veterans Home in Yountville, Napa County, aged 68. Graduate of Creighton University School of Medicine, Omaha, Nebraska, 1928. Licensed in California in 1928. Doctor Murphy was a member of the Napa County Medical Society.

◆

Parry, Gronway R., Jr., Mountain View. Died 4 February 1967, aged 41, of leukemia. Graduate of Northwestern University Medical School, Chicago, Illinois, 1954. Licensed in California in 1957. Doctor Parry was a member of the Santa Clara County Medical Society.

◆

Pyle, Howard Reitz, San Mateo. Died 23 January 1967, in San Mateo, aged 40. Graduate of the University of Kansas School of Medicine, Lawrence-Kansas City, 1951. Licensed in California in 1955. Doctor Pyle was a member of the San Mateo County Medical Society.

◆

Riesenfeld, Fred Fritz, Los Angeles. Died 13 January 1967, in Los Angeles, aged 75, of astrocytoma of the brain. Graduate of Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Prussia, Germany, 1919. Licensed in California in 1940. Doctor Riesenfeld was a member of the Los Angeles County Medical Association.

◆

Roth, Harold Alan, Riverside. Died 5 January 1967, in Long Beach, aged 60. Graduate of the University of Illinois College of Medicine, Chicago, 1930. Licensed in California in 1954. Doctor Roth was a member of the Riverside County Medical Association.

◆

Schneider, Kenneth F., Los Angeles. Died 24 January 1967, in Los Angeles, aged 70, of myocardial infarction. Graduate of the University of Illinois College of Medicine, Chicago, 1935. Licensed in California in 1941. Doctor Schneider was a member of the Los Angeles County Medical Association.

◆

Schweitzer, S. Roland, Sacramento. Died 18 January 1967, in an automobile accident near Truckee, aged 41. Graduate of the University of Rochester School of Medicine and Dentistry, New York, 1953. Licensed in California in 1960. Doctor Schweitzer was a member of the Sacramento County Medical Society.

✣

SHEARER, WALLACE C., JR., Palos Verdes. Died 26 January 1967, in Los Angeles, aged 59, of lymphosarcoma. Graduate of the University of Oregon Medical School, Portland, 1936. Licensed in California in 1948. Doctor Shearer was a member of the Los Angeles County Medical Association.

✣

SORENSON, EINER IMMANUEL, Porterville. Died 21 January 1967, in Porterville, aged 62. Graduate of State University of Iowa College of Medicine, Iowa City, 1932. Licensed in California in 1937. Doctor Sorenson was an associate member of the Tulare County Medical Society.

✣

STEPHENS, BROOKS PALMER, Ben Lomond. Died 15 January 1967, in Bakersfield, aged 73. Graduate of the University of Kansas School of Medicine, Lawrence-Kansas City, 1921. Licensed in California in 1929. Doctor Stephens was a retired member of the Santa Cruz County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✣

STURGEON, CHARLES T., Santa Barbara. Died 20 January 1967, in Santa Barbara, aged 86. Graduate of the University of Michigan Medical School, Ann Arbor, 1904. Licensed in California in 1915. Doctor Sturgeon was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

✣

THOMPSON, EDGAR ALLEN, Glendora. Died 27 January 1967, in Glendora, aged 50, of heart disease. Graduate of Indiana University School of Medicine, Bloomington-Indianapolis, 1942. Licensed in California in 1946. Doctor Thompson was a member of the Los Angeles County Medical Association.

✣

VOORSANGER, WILLIAM C., San Francisco. Died 24 January 1967, in San Francisco, aged 90. Graduate of Cooper Medical College, San Francisco, 1899. Licensed in California in 1899. Doctor Voorsanger was an honorary member of the San Francisco Medical Society.

A Mechanism to Assure Continuing Medical Education

JOHN B. DILLON, Associate Dean and Professor of Surgery/Anesthesiology, UCLA School of Medicine; Chairman, Committee on Scientific Assemblies, and member, Committee on Continuing Medical Education, Scientific Board of the California Medical Association.

THE CONCEPT THAT SOME form of continuing education is needed for all physicians in today's society is generally recognized, although not completely accepted. It does seem highly probable and, indeed, almost necessary that some evidence of "continued intellectual competence" be required for a physician to continue practice of medicine perhaps within the next five or 10 years. It seems desirable to consider methods by which such evidence of continued competence can be demonstrated, and what mechanisms can be suggested that would accomplish this with the least trauma and dislocation to the individuals involved.

With the development of the idea of the "primary physician" or family physician as a specialist in the medical field and with the present tremendous interest of the American Academy of General Practice in this area, it would seem that medicine as a whole could borrow heavily upon the concept experience of the Academy of General Practice for future methods of certification of education. The Academy of General Practice requires a certain amount of documented postgraduate work over a period of time in order to maintain membership in the Academy. If the physician fails to comply with the rules he is dropped from membership. This, of course, in no way affects his ability to continue the practice of medicine, but it does remove from him some aura of professional respectability and experience indicates that very few physicians are willing to lose their membership.

It would seem that a similar method of operation could be applied to all specialties of medicine in much the same way that the Academy of General Practice has pioneered. It is not inconceivably difficult nor prohibitively expensive for "colleges" or "academies" to assume the function that has been assumed in the past by the Academy of General Practice. It would seem that it would be possible as a requirement for continued membership in the organizations to insist that the members offer, periodically, evidence of some form of postgraduate education which might be different with each group, but, nevertheless, organized in such a way as to assure that the individual members were indeed fulfilling their obligation. To put this in somewhat of a more meaningful frame of reference, let us assume that the American College of Physicians sets up so many credit hours to be acquired by Dr. Jones. Dr. Jones during the calendar year of 1970 fulfills these requirements and submits evidence to the Society of Internal Medicine or the American College of Physicians that he has indeed complied with the requirements for continued membership. The College then submits to Dr. Jones a little card or sticker which could be applied to his certificate of membership indicating that he has fulfilled his 1970-1971 requirements and is in good standing. In addition, the College or Dr. Jones submits to the State Board of Medical Examiners a copy of his recertification or updating stamp. The Board, in turn, issues to Dr. Jones another small seal to be applied to his license. This indicates that Dr. Jones has updated his medical knowledge to the year in question. It would be required of all physicians licensed in such a state to display their license as, indeed, most all physicians do as a matter of general policy, in a place which is prominent and easily seen by all patients entering the physician's office.

It does not take a great deal of imagination to come to the conclusion that over a period of a very few years this evidence of continued education on the license and on the college membership would be very well recognized and noted by all patients entering any physician's office.

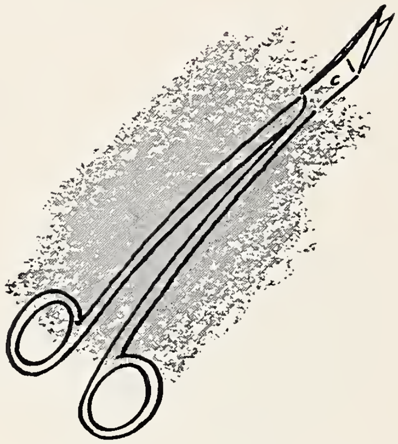
The suggestion of reexamination poses many problems. If periodic examinations were to be given by the state boards of medical examiners the numbers of physicians involved would pose an essentially unsolvable problem of manpower as well as scheduling. Subject matter would certainly have to vary with the special interests of the examined, for it would be unreasonable to expect knowledge in basic sciences to be refreshed and brought up to date to the degree expected on initial licensure.

Reexamination by specialty board would necessitate that all physicians be board certified. There is some question whether this is necessary or desirable. Universal board certification would lessen the meaning of such certification. In addition, reexamination would again require logistics of examiners and examinees that would be exceedingly difficult.

It is my opinion that examinations both for original licensure and specialty certification denote that a series of courses have been successfully completed and by their nature denote a point in time.

Membership in a college or academy would not need the degree of initial preparation of specialty board certification, as indeed it does not now, and would appear to be reasonable in its extension for continued membership to require a degree of effort by members to remain members.

The solution of updating licenses by means of continued certification on submission of evidence of taking adequate amount of postgraduate education depending upon the various fields of medicine would, I think, solve the problem in a realistic manner and would indicate to the public in a very real way the seriousness of the physician in maintaining his professional competence.



CALIFORNIA MEDICAL ASSOCIATION

1967 ANNUAL SCIENTIFIC ASSEMBLY

Biltmore Hotel • Los Angeles • April 15-19

Plan NOW to Attend
Three General Sessions on

ADVANCES IN DIAGNOSTIC TECHNIQUE

(See Program in February Issue)

DISTINGUISHED SPEAKERS INCLUDE:

- ★ William A. Altemeier, M.D., Chief, Department of Surgery, University of Cincinnati College of Medicine, Cincinnati
- ★ Irvine H. Page, M.D., Director, Research Center, Cleveland Clinic, Cleveland
- ★ Herbert M. Stauffer, M.D., Professor and Chairman, Diagnostic Section, Department of Radiology, Temple University Science Center, Philadelphia
- ★ Jon V. Straumfjord, Jr., M.D., Professor and Chairman, Department of Clinical Pathology, Medical College, University of Alabama, Birmingham
- ★ George Zur Williams, M.D., Chief, Clinical Center, National Institutes of Health, Bethesda

SPECIAL CONFERENCES ON:

Cancer of the Colon and Rectum
Research Advances in Veterinary Medicine
The Impact of Medicare—Titles 18 and 19
Medicine and Religion—Moral Aspects of New Procedures

PLUS . . .

EIGHTEEN SCIENTIFIC SECTION PROGRAMS
SCIENTIFIC AND TECHNICAL EXHIBITS
COLOR TV AND FILM SYMPOSIA

17th annual regional postgraduate institute

KONOCTI HARBOR INN
Clear Lake
Thurs. noon—Sat. noon
April 27-29, 1967

REDWOOD REGIONAL CONFERENCE

Presented cooperatively by North Coast Counties Medical Societies, Stanford University School of Medicine, and California Medical Association Committee on Continuing Medical Education. A 12-hour course.

HOST: Mendocino-Lake County Medical Society

Regional Chairman: **D. E. Coolahan, M.D.**

Institute Fee: $15.00. For additional information contact Continuing Medical Education office, California Medical Association, 693 Sutter Street, San Francisco 94102. All California Medical Association members and their families are cordially invited to attend.

PROGRAM THEMES:

CARDIOVASCULAR PROBLEMS . . . EAR, NOSE AND THROAT . . . ALLERGY . . . EYE PROBLEMS . . . MYOSITIS

THURSDAY, APRIL 27

Afternoon Session

2:00—**Office Procedures Involving the Ear, Nose and Throat**—John M. Fredrickson, M.D.

2:45—**Office Procedures Involving the Eye**—Frank C. Winter, M.D.

3:15—**Problems in the Therapy of the Allergic State**—William McKee, M.D.

4:15—CONCURRENT WORKSHOPS (you may go to one of your choice)

- A. **Demonstration of ENT Techniques Usable in the Office**—John M. Fredrickson, M.D.
- B. **Demonstration of Fundoscopic Examination**—Frank C. Winter, M.D.
- C. **Seminar on the Specific Problems of the Allergic Patient**—William McKee, M.D.

FRIDAY, APRIL 28

Morning Session

8:30—**Cardiovascular Problems**—The Useful Studies for Patients Selected for Surgical Therapy

- A. **Peripheral Vascular Disease**
- B. **Cerebral Disease and Stroke**
- C. **Cardiac Valvular Replacement**

Panel: Roy B. Cohn, M.D.
Edward J. Hurley, M.D.
Alfred P. Spivack, M.D.

11:00—CONCURRENT WORKSHOPS

- A. **Use of the Defibrillator**—Edward J. Hurley, M.D.
- B. **Wet Clinic: Patients with Cardiac Disease**—Alfred P. Spivack, M.D.

Afternoon Session

1:30—**Luncheon: New Horizons in Research**

DNA MOLECULES WITHOUT ENDS IN TUMOR VIRUSES AND TEMPERATE PHAGES—Dale Kaiser, Ph.D., Professor of Biochemistry, Stanford University School of Medicine

SATURDAY, APRIL 29

Morning Session

8:30—**Coronary Care Unit: Should a Small Hospital Have One?**—Alfred P. Spivack, M.D.

9:30—**Recent Additions to Laboratory Examinations Useful to the Clinician**—Frederick A. Fox, M.D.

10:15—**Useful Physiotherapeutic Measures in Rehabilitation**—Leslie W. Knott, M.D.

11:15—CONCURRENT WORKSHOPS

- A. **Wet Clinic Patients with Peripheral Vascular Disease**—Roy B. Cohn, M.D.
- B. **Cerebral Vascular Disease**—Edward J. Hurley, M.D.
- C. **Physiotherapeutic Procedures**—Leslie W. Knott, M.D.

17th annual regional postgraduate institute

AHWAHNEE HOTEL
Yosemite
May 5-6, 1967

SAN JOAQUIN VALLEY COUNTIES

Presented cooperatively by San Joaquin Valley Counties Medical Societies, Continuing Education in Medicine and Health Sciences, University of California Medical Center, San Francisco, and the Committee on Continuing Medical Education, California Medical Association. A 12 hour course.

HOST: Fresno County Medical Society
Regional Chairman: **Irwin Barg, M.D.**

Institute Fee: $15.00. For additional information contact Continuing Medical Education office, California Medical Association, 693 Sutter Street, San Francisco 94102. All California Medical Association members and their families are cordially invited to attend.

GUEST SPEAKER: Victor McKusick, M.D., Chief, Division of Medical Genetics, Johns Hopkins University School of Medicine.

PROGRAM THEMES:

PROGRESS IN CLINICAL PRACTICE: THE EMERGING GENETIC ASPECTS . . . GASTROENTEROLOGY

FRIDAY, MAY 5

Morning Session

Presiding:

9:00—**Constitutional and Environmental Interactions**—Piero Mustacchi, M.D.

9:20—**Genetic Determinants of Health and Disease**—Victor McKusick, M.D.

10:00—**Manifestations of Genetic Disorders in Growing Children**—Melvin M. Grumbach, M.D.

10:30—**Coffee Break**

10:45—CONCURRENT WORKSHOPS (you may go to one of your choice)
- A. **Pinpointing the Causes of Maternal Mortality**—Gilbert A. Webb, M.D., and Joseph H. Kushner, M.D.
- B. **Metabolic Problems in Daily Practice**—Felix O. Kolb, M.D., and Melvin M. Grumbach, M.D.

12:15—**Wine Tasting and Get Acquainted Session**—Doctors and their wives are invited

Afternoon Session

2:00—**Genetic Aspects of Childhood Neoplasia**—Joseph H. Kushner, M.D.

2:30—**Cancer of the Stomach: Genetic and Environmental Interaction**—Leon Goldman, M.D.

3:00—**The Implications of Intestinal Polyposis**—Walter Birnbaum, M.D.

3:30—**Coffee Break**

3:50—CONCURRENT WORKSHOPS
- A. **An Overview of Contemporary Genetics**—Victor McKusick, M.D., and Nicholas L. Petrakis, M.D.
- B. **The Patient with a "Gastric Ulcer"**—Hugo C. Moeller, M.D., and Leon Goldman, M.D.

SATURDAY, MAY 6, 1967

Morning Session

9:00—**Genetic Control of Hypertension**—Victor McKusick, M.D.

9:30—**Genetic Factors in Renal Stone Formation**—Felix O. Kolb, M.D.

10:00—**Genetic Factors in Anemia**—Nicholas L. Petrakis, M.D.

10:30—**Coffee Break**

10:45—CONCURRENT WORKSHOPS
- A. **The Patient with Pancreatic Disease**—Leon Goldman, M.D., Hugo C. Moeller, M.D., and Melvin M. Grumbach, M.D.
- B. **Common Endocrinologic Problems in Daily Practice**—Gilbert A. Webb, M.D., and Felix O. Kolb, M.D.

Afternoon Session

2:00—**Ulcerative Colitis: The Point of View of the Internist**—Hugo C. Moeller, M.D.

2:45—**Ulcerative Colitis: The Point of View of the Surgeon**—Walter Birnbaum, M.D.

3:30—**Question Period**

3:45—**Coffee Break**

4:00—CONCURRENT WORKSHOPS
- A. **The Colostomy Patient, a Combined Approach**—Hugo C. Moeller, M.D., and Walter Birnbaum, M.D.
- B. **The Patient with an Abnormal Blood Count**—Joseph H. Kushner, M.D., Nicholas L. Petrakis, M.D., and Piero Mustacchi, M.D.

application for HOTEL ACCOMMODATIONS

NINETY-SIXTH *Annual Session*

CALIFORNIA MEDICAL ASSOCIATION • APRIL 15-19, 1967

BILTMORE HOTEL, LOS ANGELES

House of Delegates Opening Session Saturday evening, April 15; Scientific Programs begin Sunday morning, April 16.

---

1. Fill in the form below **completely** for room accommodations at the CMA's 1967 Annual Session. There is only a limited number of rooms available. Your choice of accommodations will be better if your request is for rooms to be occupied by two or more persons.
2. Your reservation request should include the definite date and hour of your arrival and departure.
3. All reservations must be made through the CMA Housing Bureau, 693 Sutter Street, San Francisco, California 94102.
4. **DEADLINE** for housing: April 1, 1967.

HOTEL ROOM RATES*

	Single	Double and Twins	Suites
BILTMORE HOTEL			
515 So. Olive Street	10.50-17.50	15.50-21.50	30.00-55.00
MAYFLOWER HOTEL			Triples
535 So. Grand Avenue	8.50-13.50	15.50-17.50	22.00
STATLER-HILTON			Suites
930 Wilshire Boulevard	11.00-17.50	16.00-22.50	30.00-65.00

*Rates subject to change

Send to: California Medical Association—*Housing Bureau*
693 Sutter Street, San Francisco, California 94102

Please reserve the following accommodations for the CMA's 1967 Annual Session in Los Angeles, April 16-19, 1967. The first meeting of the House of Delegates begins Saturday evening, April 15; Scientific Programs begin April 16.

Single Bedroom $.................... Twin-Bedded Room $.................... Double Bedroom $....................

Small Suite $.................... Large Suite $.................... Other $....................

First Choice Hotel.................... Second....................

Arrival (date) .... Hours.................... a.m. p.m. Departure (date) .... Hour.................... a.m. p.m.

THE NAME OF EACH HOTEL GUEST MUST BE LISTED. Include the names and addresses of both persons for each twin-bedded or double room, and names and addresses of all other persons for whom you are requesting reservations.

....

....

....

....

—PLEASE PRINT—

Your Name .... Officer? .... Delegate? .... Alternate? .... Speaker? ....

Address .... County ....

City and State.................... Zip No. ....

PUBLIC HEALTH REPORT

Lester Breslow, M.D., M.P.H.
Director, State Department of Public Health

SINCE THE ANNUAL influenza surveillance network went into operation the first week of October, and through the middle of February, no influenza activity has been reported in California.

School absenteeism has remained substantially at normal rates of 3 to 8 per cent. So far this season, very few specimens have been received with requests for testing for pheumotropic viruses. And to date there has been no laboratory confirmation of influenza in California.

Some increase of unspecified respiratory infections was noted by physicians in Colusa County during January. Illness rates ascribed to the "flu syndrome" have remained under 4 per cent in the California Medical Facility at Vacaville, and the weekly total admissions to Cowell Memorial Hospital at the University of California, Berkeley, for respiratory infections have remained below 10.

Pneumonia-influenza deaths have remained at or below the epidemic threshold so far this season. Eleven cities report on pneumonia-influenza deaths, and 14 local health departments participate in the surveillance of school and industrial absenteeism.

Thus far the 1967 experience is in sharp contrast to that of the previous influenza season when a widespread and severe epidemic occurred and the number of deaths directly or indirectly related to influenza was the highest since 1960. As observed in previous epidemics, deaths from causes other than pneumonia and influenza were also elevated during the 1965-66 epidemic, reflecting the well known increased risk of mortality from cardiovascular, renal and other causes during periods of epidemic influenza. Total "excess" pneumonia-influenza deaths—that is, over the normal expectations—in the 11 reporting cities during this epidemic numbered over 320.

The epidemic was severe in its intensity and in the number of persons affected. Statewide the epidemic was short, lasting approximately two and a half months, and came to an end by the first week of April.

* * *

The familiar yellow booklet, "International Certificates of Vaccination," has been revised and the new form went into effect the first of the year. Smallpox, cholera and yellow fever vaccinations recorded on the old form before 1 January will remain valid until the expiration date of the certificate. For example, if the physician vaccinated a traveler against smallpox on 31 December 1966, this certificate is valid for three years.

The new Certificate of Vaccination or Re-vaccination against smallpox now requires the physician to record the origin and batch number of the vaccine.

The Certificate of Vaccination or Re-vaccination against yellow fever was amended to extend the validity from six years to 10. Certificates already issued are automatically extended to be valid for 10 years from the date of vaccination or re-vaccination.

INFORMATION

Your Medical Assistant

HENRY BODNER, M.D., *Los Angeles*

THE WELL QUALIFIED, highly trained medical assistant of today is a vitally important part of the art and practice of medicine. Good public relations are usually well exemplified in an efficient assistant.

The organizational pattern of the Medical Assistants Association is very much like that of the American Medical Association and its component state and county medical associations. There are county, state (California Medical Assistants Association) and national bodies (American Association of Medical Assistants). The American Association of Medical Assistants has now grown to an organization of approximately 15,000 members in 44 state chapters. The California Medical Assistants Association has approximately 2,100 members in 45 local chapters. This organization is still dedicated to the basic principles and guidance indicated in the constitution of the Long Beach Medical Assistants Association which was organized more than 30 years ago. The original Long Beach Chapter constitution stated:

> The object of this organization shall be to aid in the advancement and elevation of the medical professions by encouraging women employed as medical assistants in ethical medical offices to form societies whereby they may secure the educational advantages of lectures, discussions and instruction in the details of their duties; to bring to its members a desire to render more efficient service and to be truthful, honest and loyal to the profession which they serve; to create a feeling of fellowship and cooperation among its members and promote among them a desire for mutual improvement, and it shall be non-profit, non-union, non-partisan and non-sectarian.

Component medical assistant chapters have physicians as "medical advisors." These advisors are requested to serve by the respective chapters subject to approval by the local county medical association. The state medical assistants association and the national organization also have physicians to serve in an advisory capacity, and their appointment also must be approved by the state medical society and the American Medical Association.

The medical assistant has gained much stature in recent years as the result of studies over the past few decades. It is obvious that a good assistant can be of real help to her employer in providing good medical care. The future value of the medical assistant was all the more emphasized when a recent survey showed that most physicians spend one-third of their time in administrative details (with such things as writing records in longhand, explaining hospital costs, discussion of insurance coverage and accounts) which can easily be handled by a qualified aide.

The medical assistants organization now has established a certification board analogous to the various medical specialty boards. The medical assistants chapters throughout the United States have a continuous educational program carried on at monthly meetings. In addition, most state associations, as well as local chapters, sponsor one-to two-day educational symposia through the year. Under the guidance or stimulation of component medical societies, more than 20 junior colleges in the state of California now offer courses in medical assistant training. The California Medical Association has established a Liaison Committee to the California Medical Assistants Association to cooperate and work with California junior college administrators. The California Industrial Education Association, California Medical Assistants Association president, state educators and hospital administrators attended California Medical Association council meetings this past year to advise and attempt to standardize the course of study for medical assistants.

The Maricopa County Medical Society in Arizona recently established a five-month training school for medical assistants. This school has a faculty of professional educators as well as a group of more than 25 active practicing physicians. The fifth month of training in this school consists of an internship in a physician's office. Medical subjects offered to the students include anatomy, physiology, patient relations, preparing patients for examination, operation of the electrocardiograph, reading the blood pressure and temperature, professional ethics, medical terminology and spelling, medical transcription and corre-

---

Dr. Bodner is a member of the California Medical Association Advisory Committee to the California Medical Assistants Association.

spondence. The non-medical subjects include use of dictating machines, medical office accounting, collections and credit, preparation of insurance and industrial commission forms, office management, social security and taxes, telephone techniques, purchasing supplies and services, malpractice insurance, role of the detail man and grooming and personal development.

In the near future, there may be set up national computer systems for all patients in the United States to facilitate more complete patient histories. Automated billing is already in effect in some areas. Central record controls are obviously around the corner. Computerized and electronic equipment will obviously continue to be increasingly important in day-to-day practice. The Medical Assistants Association is well aware of the need for all aides to be familiar with the operation of these varied types of electronic equipment.

The physician's partner in the care of the sick warrants the utmost interest and cooperation by the physician. Much closer liaison between individual physicians and medical assistants associations is needed. More physicians should encourage their assistants to be members of an organization dedicated to improving themselves by constant education and training. A physician investing dues for his aides in this organization would receive countless returns on his investment.

Although numerous attempts have been made to unionize our assistants, they still insist on staying out of labor organizations. As physicians individually and as members of our county, state, and national medical association, we must continue to support the Medical Assistants Association. If we expect loyalty from our assistants, we need to be loyal to them. Have you urged and sponsored your aides to membership in the Medical Assistants Association?

Dentist-Physician Relationship In Extended Care Facilities

THE HOSPITAL DENTAL SERVICE Committee of California Dental Association and Southern California Dental Association, meeting in joint session on 9 November 1966 in Los Angeles, developed the following statement defining the obligations of the dentist concerning services for patients in Extended Care Facilities, Nursing Homes, Convalescent Hospitals, etc., under the Medicare and Medi-Cal programs. A firm understanding of the dentist-physician relationship will obviate the need for stringent controls by government agencies.

Dentists rendering care to patients in extended health care facilities, nursing and convalescent homes who need other medical services should consult with the patient's physician so that the total health of the patient may be considered.

Procedures to implement dentist-physician consultations should be established in each facility so as to allow the dentist freedom to carry out his responsibility for the dental health of the patient without jeopardizing the physician's concern for the patient's physical and emotional condition. Special emphasis should be placed on consultation where the administration of drugs and anesthetic agents is contemplated.

CHESTER W. MERRILL, *Chairman*
Hospital Dental Service Committee
California Dental Association

CHARLES YOON, *Chairman*
Hospital Dental Service Committee
Southern California Dental Association

Federalized Health Care Systems in Western Europe And Australia

A Report of the Bureau of Research and Planning, California Medical Association

CURRENT LEGISLATIVE action, including passage of P.L. 89-97, which contains provisions for health benefits for the aged, necessitates review of the responsibility assumed in other countries by government for the financing and provision of health care, if only to become acquainted with programs of these other countries. The object of this *Report* is to describe the nature, organization, and distribution of health benefits in several countries, largely in Western Europe, but including also the unique system found in Australia. No attempt is made to compare in a critical fashion the varied systems

---

Reprint requests to: 693 Sutter Street, San Francisco 94102.

discussed, although descriptions of programs in selected countries may provide some clues to future directions in the United States.

As of May 1964, 64 countries throughout the world, including all European nations, had some type of sickness insurance and benefit program.[11] Generally, in Western Europe, these benefits include old age, medical care, cash sickness, survivor, maternity, invalidity, family allowance, and unemployment benefits. This *Report,* however, considers only the financing of medical care and cash provisions for illness; no qualitative judgment is made on the various systems. The intent of this paper is to provide a summary and comparison of certain aspects of medical provision in selected countries. However, a brief outline of some of the problems common to these systems may be useful to the reader in evaluating the information presented.

Difficulties have been encountered in some countries in coordinating different aspects of health services, particularly where responsibility is shared by two or more governmental agencies; the reconciliation of professional freedom with government supervision has occasionally proved troublesome; and provision of the best possible medical care may conflict with the need for economy in public expenditure.[3] (For a discussion of professional problems in federalized health care abroad, the reader is referred to CALIFORNIA MEDICINE, 104:146-151, February 1966.)

Payment of the physician may be handled by a number of methods, from salaried service to fee-for-service, typified respectively by the U.S.S.R. and the United States. Other systems include: capitation, a regular payment to the physician for each of a group of patients, with free contractual relationships, as found in the United Kingdom; case payment, where the doctor is paid by the financing organization according to the number of patients seen in a given period; and fee-for-service, where payment based on a set scale is given for actual services performed. In this case, payment may be made either by the patient, who is then reimbursed in whole or in part, or directly by the financing organization.[5]

Certain aspects of health care services are covered in more depth in most European countries than under P.L. 89-97. For example, dental care, eye glasses and examinations, prosthetic devices, and hearing aids are regularly covered under most European systems but are excluded under Medicare. Most European systems provide unlimited hospitalization benefits from the onset of illness, while Medicare provides 90 days of hospitalization per illness, with a $40 deductible, and $10 per day from the 61st through the 90th days of hospitalization. Medicare covers a much more narrow segment of the population—those 65 and over—than do European systems.

Organization and financing of health services in France, Germany, Italy, Sweden, the United Kingdom, and Australia[1,2,4,6-10] are described briefly below.

In *France,* comprehensive state-controlled health insurance applies automatically to about nine-tenths of the population, including all paid workers, their families, and all pensioners and the unemployed (although to be eligible for cash benefits the recipient must have worked at least 60 hours in the three months prior to the onset of illness). Participation by individuals not covered automatically is voluntary. Employees and employers participate in the administration of the program, which is organized on three levels: local funds make benefit payments and receive deposits; medical and hospital rates to be paid are set by agreement with the medical association at the regional level; and payments to local groups and coordination of the entire program are made at the national level.

The patient has free choice of doctor and hospital; all doctors and public hospitals and many private hospitals participate.

The program is financed with funds from the single payment made monthly to the overall social security program. This payment averages 20.25 per cent of the worker's income (6 per cent paid by the worker and 14.26 per cent by his employer), and may run as high as 35 per cent. Although the State makes no contribution, it may make advances to the fund. Deficits in the national fund are made up by general taxation.

Unlimited health benefits begin at the onset of illness; the patient pays the doctor directly and is reimbursed for 80 to 100 per cent of physician and hospital expenditures. However, after one month in the hospital, or, in case of long-term illness or three months absence from work, the patient pays no hospital bills at all. Dental, orthodontic, and drug bills are also reimbursed in large measure. Cash payments during illness are made up to 12 months in any three year period, amounting to 50 to 66.67 per cent of salary, based on severity of illness and number of dependents. Pensioners continue to receive full benefits during illness.

In *West Germany,* the health insurance program, although privately run, is state supervised. Local organizations receive contributions, make payments, and supervise the operations, under a Federal association. The individual belongs to the compulsory plan at his place of employment or residence; provisions are also made for voluntary membership of those not automatically enrolled. The local organization makes a lump sum payment on a capitation basis to associations representing the physician; these associations pay the individual physician for items of service under an official fee schedule. The patient has a free choice of approved doctors and hospitals.

Employer and employee each contribute one-half of an amount equal to 8 to 11 per cent of the worker's salary, up to $165 a month. The Federal Government contributes only for the unemployed; the state does not contribute, except to make up deficits.

In order to be eligible for benefits, the worker must earn less than $1,980 a year; there are no prior work requirements.

Coverage extends from the onset of illness, with benefits for a maximum of 18 months of hospitalization. Dental work, hearing devices, and most drugs are covered completely. Cash benefits of up to 69 per cent of the worker's salary are available up to 78 weeks in any three-year period; the employer pays the difference between the benefits and the full salary for the first six weeks of illness. All workers in Germany receive these benefits regardless of nationality.

In *Italy,* approximately 85 per cent of the population, including all non-government workers, their families, pensioners, and the unemployed are covered by the health insurance program, which is operated by an autonomous administration under the National Government. Management is done by governmental representatives, employers, and the insured. In the framework of this scheme, the National Council supervises the entire program, regional organizations collect and administer funds, and local branches assure availability of medical care and make benefit payments. Benefits are paid from the onset of illness, but for a maximum of six months a year; there is no ceiling on income of participants, and no required period of work prior to illness.

The State contributes nothing to the benefits. Blue collar workers pay 15 per cent of their wages, their employers 7.15 per cent. White collar workers pay 15 per cent, employers 5.15 per cent of wages. These payments are made to the Social Security Administration, and cover all benefits such as old age, maternity, and survivors benefits, in addition to health insurance.

Physicians are either under contract with the Social Security Administration or are paid separately for each patient. In the former case, the patient must select a physician and a hospital associated officially with the health plan; in the latter case he may choose any doctor or hospital, but must pay some of the medical costs himself.

Dental care and most prescription drugs are free, and allowances are made for prosthetic devices, hearing aids, and eyeglasses. There is a separate system for tuberculosis patients, under which complete payment of sanitorium fees and up to 50 per cent of other costs may be paid.

Cash payments of from 22 to 54 per cent of wages are made for a maximum of six months, depending on whether or not hospitalization is necessary, and on number of dependents.

In *Sweden,* the National Social Insurance Board operates the health insurance program through regional and municipal offices, hospitals, and public assistance committees. Public officers at regional and national levels are responsible for policy making and administration. All persons, including housewives and the self-employed, are eligible to participate. Under this system, the patient pays the physician, and is reimbursed 75 per cent of the standard fee by the regional office. Fee schedules are worked out by the government, in cooperation with the Swedish Medical Association. Social assistance service is available for those who cannot afford to make use of the regular service.

There is no time limit on hospitalization or medical services, except for the patient over 67 years of age or on an old age or retirement pension, in which case maximum duration of hospitalization is 180 days. Cash benefits vary from 45c to $4.50 a day, depending on income and level of contributions.

Contributions by the worker cover about half the flat premium rate, and the Government and the employer each pay about one-fourth. Cost of insurance varies regionally and with the amount of earnings. Fifty per cent of the cost of cash benefits, and refunds on doctors' fees, as well as part of the cost of drugs and medicine, most hospital costs, and contributions for the unemployed are paid by the Government.

All doctors and public hospitals participate in the program, and the patient has a free choice among them. Reduced charges are made in municipal sanitoriums for tuberculosis, mental illness and other protracted conditions, and dental care is free to children.

The *United Kingdom* has a health plan which differs from those found in the rest of Western Europe, in that medical care is not a part of a social insurance system, but a free public service. All residents of Great Britain and Northern Ireland are eligible, without qualification, for health services of all kinds. The Government covers about 80 per cent of the cost of the National Health Service; of the remaining 20 per cent, the employee pays about four-fifths and the employer about one-fifth. An annual fixed fee per patient is paid to the participating physician, regardless of services actually rendered. The patient has a free choice of doctors. Most hospitals are government owned. There is a charge of one shilling (14c) for prescribed medicines. Dental services, eyeglasses, hearing aids, etc., are dispensed through the National Health Service, but the patient shares the cost.

Cash benefits for illness are extended automatically to virtually all employed and self-employed individuals. Housewives and the low-income self-employed may insure if they wish. On the average, workers contribute about $1.34 and employers about $1.60 weekly, to the entire national insurance program (including old-age, survivors, maternity, and unemployment benefits) with the Government paying about one-third of the cost. There is no limit on duration of payments for the worker who has made at least 165 weekly contributions.

Disability payments are made of $11.20 a week, with additional payments for dependents.

Australia combines voluntary insurance with benefit payments from Commonwealth Funds. Payments are administered through voluntary insurance agencies under the Australian Medical Plan. Only a small Government staff is utilized; most paper work is conducted by the insurance agencies. No special or separate taxes are levied to support the plan, and all money withheld from checks is voluntary. Under this system, the patient pays the doctor, and receives insurance and Commonwealth benefits on the basis of a statutory schedule, agreed upon by physicians, pharmacists, and the Government, providing fees for specified services. The total charge is always slightly higher than the combined payments of the Commonwealth and insurance.

In order to receive benefits, the patient must be a member of a registered "medical benefits organization," contributing approximately 35c a week for the single person, and about 70c for families. In theory, the Commonwealth and insurance fund should pay a total of 90 per cent of medical expenses, and the patient 10 per cent. In practice, the Commonwealth averages payments of 28 per cent, insurance of 34 per cent, and the patient of 38 per cent. In 1964, approximately 75 per cent of the population was covered under this scheme.

Under this system, the patient has a free choice of physician; he makes his own arrangements, pays the bill, obtains a claims form from his benefit organization, and sends it in, with the doctor's receipt, to his insurance agency. Payment of both Commonwealth and insurance benefits is made by the organization, usually by mail, but sometimes on the spot. If the patient is unable to pay the physician, the organization may handle payment directly.

Sickness benefits of up to about $11.55 a week are payable after a waiting period of one week, for the duration of the illness.

REFERENCES

1. European Social Security Systems, Economic Policies and Practices, Paper No. 7, Joint Economic Committee, Congress of the U.S., U.S. Government Printing Office, Washington, D.C., 1965.

2. Health services in Western Europe, Economic Newsletter, Canadian Medical Association, January 1966.

3. Ibid, p. 2.

4. Hogarth, James: The Payment of the Physician: Some European Comparisons, The Macmillan Company, New York, 1963.

5. Ibid, p. 3.

6. Medical care protection under social security schemes: A statistical study of selected countries: International Labour Review, 89:570-593, 1964.

7. Recent Foreign Social Security Developments, Research and Statistics Note No. 4, U.S. Department of Health, Education, and Welfare, November 1964.

8. Schoeck, Helmut: Financing Medical Care: An Appraisal of Foreign Problems, Caxton Printers, Idaho, 1962, pp. 227-305.

9. Social security programs of foreign countries: Social Security Bulletin, 27:16-26, September 1964.

10. Social Security Programs Throughout the World, 1964, U.S. Department of Health, Education, and Welfare, U.S. Government Printing Office, Washington, D.C., 1964.

11. Ibid, p. xvii.

LETTERS *to the Editor*

Spider Bites, Tick Bites

THE CASE REPORT of Doctors Lewis and Regan, CALIFORNIA MEDICINE, Vol. 105, p. 457, is of interest in that the photographs and clinical description accurately portray the results of the bite of the venomous tick known as the pajaroello, a rather unpleasant resident of California, in particular, the Salinas Valley.

The pajaroello, also spelled pajahuello and variously pronounced ("pock-a-whale-oh" is heard most frequently), encounters its victim in a rural field, one which appears quite pleasantly dotted with oak trees, and which has been grazed by cattle. The tick frequents the ground beneath the oak trees where the traveler is wont to take a rest from driving, or a short picnic. The bite of the pajaroella is not usually felt immediately, in common with many other ticks it apparently produces some local anesthesia before becoming firmly attached.

A severe local reaction begins a few hours later and progresses with startling speed from a small bite with perhaps 2 cm of induration to one with deep central cyanosis of 5 cm to 10 cm surrounded by thick, edematous skin within 24 or 36 hours, and later by a very distressing total slough of the cyanotic area. The sloughed area resembles that which would be cut out by a roughly wielded plug-cutter through all layers of the skin down to the fascia. Fever is uncommonly present, and despite the appearance of the lesion, pain is not prominent and there is little if any lymphangitis.

Treatment is not standardarized, however, local wound care is given consisting of a bland soapy wash, large doses of tetracycline commenced immediately, immobilization (if an extremity is involved), and warm moist packs of diluted sterile Magnesium Sulfate solution. Early treatment in this manner has prevented slough in cases seen as long as six hours after presumed time of exposure.

The pajaroello does not appear like ordinary more familiar ticks in that it is rather small, about 0.6 cm in size, and instead of appearing somewhat leathery it resembles a shed piece of oak bark, grey in color and rough in texture. The underside resembles the more familiar ticks. Although Doctors Lewis and Regan stated that they were unfamiliar with insects which could cause injury similar to that in their case report and ascribed the injury to a variety of Loxosceles, the pajaroello is defined in Dorland's Illustrated Medical Dictionary, 24th Ed. as, "pajaroello: Ornithodoros coriaceus. The pajaroello of California which is greatly feared because of its bite. It is not known to transmit disease." Another dictionary describes it as "a venomous tick," and a desk standard dictionary gives an alternate spelling as "pajahuello," which closely resembles the local pronunciation.

In that no known vector of injury was identified in the above-described case, and knowing of the common existence of the pajaroello, it would be interesting to learn if the patient had been in an area where he could have picked up this very undesirable hitch-hiker and to collect some pajaroellas and see if they would produce lesions similar to those described.

JAMES H. DOOLEY, M.D.

I HAVE REVIEWED Dr. Dooley's letter with great interest. According to Dr. Dooley the venomous tick pajaroello can cause necrotic lesions similar to but smaller than those produced by the Loxosceles spider.

This tick usually encounters its victims in rural fields and is commonly found in areas grazed by cattle, and in the vicinity of oak trees. Therefore, this tick would be classified mainly as an outdoor insect, and encounters its victims in open areas.

As stated in our paper the Loxosceles spider is predominantly an indoor spider. It usually inhabits storage areas around the house and within stored bedding and clothing. It is a nocturnal predator; its victims are therefore encountered in bed during the night or after wearing clothing that has been stored for several months.

We have encountered three patients, who have presented with large necrotic lesions following a bite of a small insect. All three patients have shown large, deep ulcerations similar to the ulcerations seen in the young man from San Diego. The most significant finding in all these patients is that they received the bites in bed while sleeping. In the latter two patients, which I examined after publication of our paper, the patients actually found small spiders on their beds after awakening the following morning. Unfortunately, both of these patients disposed of the spiders, making it impossible to positively identify it as the Loxosceles spider. The entomologist, who examined our first patient, is familiar with the pajaroello tick and feels that the lesion produced by this venomous tick is not as extensive as the lesions in our first reported case.

A controlled experiment similar to the one used by Dr. Wingo in identifying the Loxosceles spider in Missouri, should be performed using both the venomous tick as well as the Loxosceles species found in the Southern California area.

MARTIN I. LEWIS, M.D.

THE INTERESTING article on necrotic arachnidism by Lewis and Regan (December 1966[1]) warrants some comment.

All spiders with the exception of two small groups are venomous.[2] Fortunately, the fangs of most species are too short or too fragile to penetrate the human skin. Nevertheless, at least 50 species of spiders have been implicated in bites on humans in the United States. The venoms of most of the spiders have not been studied in any detail, but what observations have been made indicate that many spider venoms have common chemical and physiopharmacological properties.[3] This would be expected, for most spiders use their venom apparatus for food-gaining purposes. Some of the so-called "salivary" enzymes are not only common to many of the spiders but are also present in some snake venoms.[3,4] It is, therefore, possible that if a species of spider can inflict a bite on man its venom might produce signs and symptoms similar to those seen following bites by other spiders, including *Loxosceles reclusa.* As Lewis and Regan indicate in their paper,[1] poisoning by this species was not recognized until 1957, even though many cases were seen prior to that date.

Non-*Latrodectus* (black widow) spider venom poisoning is very common in Southern California. In an 11-year period (1955-66) the senior author has seen 44 cases of suspected spider bite at this hospital and an additional 29 cases on consultation at other hospitals in the greater Los Angeles area. In addition, 87 suspected spider bites were reported to us through the Poison Information Center at the Children's Hospital in Los Angeles. The junior author has received almost 400 reports of other suspected spider bites in Los Angeles County during the past 10 years.

In only 16 of the cases seen by the senior author was the offending spider brought to the hospital for identification. The spiders were:

Phidippus formosus
Misumenoides aleatorius
Liocranoides sp.
Neoscona vertebrata
Drassodes sp.
Aranea sp.
Chiracanthium inclusum
Peucetia viridans
Pamphobeteus sp.
Lycosa raptoria
Cupiennius sallei
Phoneutria sp.
Bothriocyrtum californicum
Aphonopelma sp.

All of these spiders are known to bite, and on some occasions to produce lesions. Bites of *Phidippus sp.* in Southern California were apparently known to practitioners in this area at the turn of the century,[5] and no less than 416 suspected spider bites are recorded in the hospital records studied by us in preparation for a bibliography.[6]

In seven of the cases seen by one of us (FER), a necrotic lesion not unlike that described by Lewis and Regan has been observed. In one of these cases, *Loxosceles unicolor* was the offending species. The bite was inflicted on a Southern Pacific Railroad brakeman enroute to Los Angeles from Tucson, Arizona, and was subsequently treated at the Southern Pacific Yard Hospital in Los Angeles. In the other six cases the offending arthropod was not caught, although it was seen by two of the patients. The description given by these patients did not fit that of a *Loxosceles* species. In one case the offending arthropod was probably not a spider, although at the time the patient thought it was. It was probably a Pajaroello tick, *Ornithodoros coriaceus,* which has been implicated in numerous bites in this area, and whose toxin sometimes produces a vesicular lesion which on occasions may give rise to necrosis and ulceration. The ulcers are particularly painful and slow to heal. These cases will be reported elsewhere.

These various reports and cases indicate that spider bites are very common in Southern California, and that some may result in lesions not wholly unlike those produced by *L. reclusa.* We have also seen flea, bedbug and biting-fly bites that have developed into bullous lesions which have subsequently ruptured and ulcerated.

Finally, we are not aware of any "pronounced westward ecologic migration of this spider . . . as far west as the Pacific Coast." The migrations of *L. reclusa* have been carefully studied and recorded, and what few spiders of the species that have been found west of Texas have been "introduced" species; that is, they have been brought into the area by a collector or on personal belongings, in boxcars or in autos. One such specimen has recently been found in Southern California.[7] It was introduced on the personal belongings of a family visiting from the midwest.

The possibility that *L. unicolor* might be implicated in some cases of arachnidism in the southwest cannot be excluded in view of the case we have noted. However, it is difficult to accept the supposition that the numerous spider-bite cases seen in Southern California, only a few of which have resulted in localized necrosis and ulceration, can be attributed to *L. unicolor* (since this spider is exceedingly rare in this area), or to an introduced *L. reclusa* specimen (since only one has ever been collected in Southern California). Unless the offending spider is identified by an arachnologist or a competent zoologist, we feel that a diagnosis based upon the lesion alone is very hazardous, and that the physician should be cautious in accepting the assumption that *Loxosceles* alone is responsible for necrotic arachnidism.

FINDLAY E. RUSSELL, M.D.
Director, Laboratory of Neurological Research, Los Angeles County Hospital
WILLIAM G. WALDRON
Los Angeles County Health Department

BIBLIOGRAPHY

1. Lewis, M. I., and Regan, J. F.: Necrotic arachnidism, Calif. Med., 105:457-462, 1966.
2. Gertsch, W. J.: Personal communication to the senior author.
3. Kaiser, E., and Michl, H.: Die Biochemie der tierischen Gifte, F. Deuticke, Wien, 1958.
4. Russell, F. E.: Phosphodiesterase of some snake and arthropod venoms, Toxicon, 4:153-154, 1966.
5. Browning, C. C.: Original investigations of spider bites in Southern California, Southern California Practitioner, 16:291-300, 1901.
6. Russell, F. E.: Bibliography of venomous arthropods and their venoms, in preparation.
7. Waldron, W. G., and Russell, F. E.: Loxosceles reclusa in Southern California, Toxicon, in press.

NEWS & NOTES

NATIONAL • STATE • COUNTY

LOS ANGELES

Post-doctoral fellowships in cancer for practicing physicians are available immediately at the University of Southern California School of Medicine and the Los Angeles County Hospitals, under sponsorship of the National Cancer Institute. The fellowships are for one month each, with a stipend of $1500. They are for board-eligible or certified physicians in **tumor surgery, tumor pathology, therapeutic radiology and cancer chemotherapy.** Applicants should send a brief resumé to: Dr. David Stolinsky, 2025 Zonal Avenue, Los Angeles, California 90033.

SAN DIEGO

Mr. Cliff Smith, medical writer for the San Diego Union, was honored recently by the San Diego County Medical Society for his outstanding reporting of medical affairs. Mr. Smith is the fourth newsman to win this recognition since it was established by the medical society in 1960.

SAN FRANCISCO

The 48th annual session of the **American College of Physicians** will be held 10 to 14 April at the Civic Auditorium in San Francisco. Attendance is expected to exceed 4,500.

The five-day scientific program will feature original papers on clinical medicine, clinical investigation, basic sciences and psychiatry, as well as two symposia, color television programs and 12 panel discussions.

Four award lectures will be presented during the week. Sir John C. Eccles, Canberra, Australia, will give the annual Lilly address. Maxwell M. Wintrobe, M.D., Salt Lake City, Utah, will present the John Phillips Memorial lecture; David T. Smith, M.D., Durham, N.C., the James D. Bruce Memorial lecture and Marshall Nirenberg, Ph.D., Bethesda, Md., the American College of Physicians lecture.

* * *

Dr. William K. Hamilton, professor of the Department of Anesthesia at the University of Iowa College of Medicine, has been appointed **professor and chairman of the Department of Anesthesia** in the School of Medicine at the University of California San Francisco Medical Center. The appointment is effective 1 July.

* * *

The San Francisco academy of General Practice is sponsoring for the fourteenth year its series of **medical and surgical postgraduate lectures** to be held at French Hospital, San Francisco, Tuesday nights 8-10 p.m., 4 April through 2 May. These lectures were formerely called the Ft. Miley Symposium. Further information may be obtained from Dr. George E. Welles, 2386-20th Avenue, San Francisco 94107.

GENERAL

Two new appointments to the **State Board of Medical Examiners** were announced recently. **Dr. James C. MacLaggan,** San Diego, president of the California Medical Association, was appointed for a four-year term. He succeeds Dr. Donald H. Abbott of Riverside. **Dr. Marco R. Rago,** Los Angeles, has been named to complete the term of Dr. Francis E. West, of San Diego, who recently resigned.

Dr. George C. Griffith, Los Angeles, was presented the 1967 Gifted Teacher's Award of the American College of Cardiology, and **Dr. E. Grey Dimond,** of Jolla, received the Distinguished Fellowship Award, at the organization's annual scientific session held recently in Washington, D.C.

Scientists who would like to present papers at the **American Heart Association's 1967 Scientific Sessions** to be held 20 to 22 October should submit abstracts no later than 15 May, according to announcement from Dr. Rodman D Starke, chairman of the San Francisco Heart Association's Professional Education Committee. Papers should be based on original investigations in the cardiovascular or related fields.

Forms for submitting abstracts and applications for scientific exhibit space can be obtained by writing directly to: Department of Medical Education, American Heart Association, 44 East 23rd Street, New York, N.Y. 10010.

The Physician's BOOKSHELF

PROGRESS IN CLINICAL PSYCHOLOGY—Vol. VII—Edited by Lawrence Edwin Abt, Ph.D., and Bernard F. Riess, Ph.D. Editorial Board: Henry P. David, Ph.D.; George S. Klein, Ph.D.; Bruno Klopfer, Ph.D.; Rollo May, Ph.D.; James G. Miller, M.D., Ph.D., and David Wechsler, Ph.D. Grune & Stratton, Inc., New York, 1966. 309 pages, $11.75.

This volume contains sixteen review articles by fifteen different sets of authors in three general areas relevant to the current status of clinical psychology.

First of these general areas is entitled "Problems and Development in the Practice of Clinical Psychology" and deals generally with the variety of dimensions which define the current struggle of clinical psychology for its identity.

The second area entitled, "Research Problems and Progress in Clinical Psychology," presents a series of selected relevant findings which influence the practice of clinical psychology and, in a broader scope, psychiatry and medicine as well.

The final area is entitled, "Clinical Psychological Practice in Other Lands," and contains articles describing the activities and functionings of psychologists in five selected European countries.

It must be emphasized that the volume does not represent necessarily a comprehensive cross section through all of clinical psychology. In the total of seven editions of this book selected topics each year will cover the total span of the major areas of psychology when summed over several consecutive volumes although certainly not within each volume. Related topic headings may appear only once every two or three volumes or if a major central area of interest it would appear each year in the pattern of the Annual Reviews of Psychology or the Annual Reviews of Physiology and Medicine.

The articles themselves are somewhat mixed both in quality and in the manner in which they approach the topic. Some contain a comprehensive and careful review of the literature with summary conclusions from that review which can be interesting and most useful. Others represent a coherent statement of a philosophic or theoretical position regarding a relevant psychological issue such as Dr. James Bugental's article on Humanistic Psychology. Many of the articles are of parochial interest to psychologist alone but the major portions of the volume are written in sufficiently broad terms to be of general interest to physicians who wish to acquaint themselves with some current attitudes, beliefs and concerns of Medical Psychologists. One of the most interesting comparisons comes from juxtaposition of an article by Joseph Wolpe and on direct behavior therapies, and that of James Bugental on humanistic psychology. Both of these approaches to the psychotherapeutic endeavor are less well known to medical practitioners and should make stimulating reading. Of interest to many may be the first chapter in the book dealing with manpower and training which can give some facts to correct the beliefs held regarding the activities and functions of psychologists.

That chapter points out that some 37 per cent of psychologists in America responding to the National Science Foundation questionnaire of 1964 identified themselves as Clinical Psychologists with the remaining psychologists scattering themselves through the fields of Experimental, Comparative, Psysiological, Educational, etc. Approximately 6 per cent of the total membership of the American Psychological Asociation are in private clinical practice and the remainder are employed for their major professional activities in educational institutions, a variety of governmental agencies, industry, business, the military and other assorted pursuits. Similarly of the 292 former NIMH Trainees in Psychology as polled in 1962, only 3 per cent were in private practice with some 73 per cent employed in hospitals, clinics, medical schools and other educational institutions. From these figures it would appear that the psychologist remains largely a teacher, administrator or institutional employee.

Despite its variability both in quality and intensity, the volume would be a useful one for physicians to read who wish to inform themselves about psychology.

ALEXANDER C. ROSEN, PH.D.

* * *

PRIMER ON PREMATURITY AND HIGH-RISK PREGNANCY—By S. Gorham Babson, M.D., Associate Professor of Pediatrics, University of Oregon Medical School, Hospitals, and Clinics, Portland, Oregon; Director of Premature Nursery, Doernbecher Memorial Hospital for Children, University of Oregon Medical School, Portland, Ore.; and Ralph C. Benson, M.D., Professor of Obstetrics and Gynecology and Chairman, Department of Obstetrics and Gynecology, University of Oregon Medical School, Hospitals, and Clinics, Portland, Ore. The C. V. Mosby Company, St. Louis, Mo., 1966. 194 pages, $10.50.

This short text covers a topic which is attracting the attention of many obstetricians currently. To quote directly from the text, "The death rate of the low-weight neonate is 40 times that of the full-sized infant born at term." Therefore, any practical suggestions for the management of these problems would be most welcome.

In this short text there is adequate coverage of definition and description of the problem, discussion of the major contributing factors, and their management, and proper treatment of the infants. The discussion of the role of the infectious diseases is an excellent compilation. As stated in the preface, personal judgments have been made by the authors in some instances. Thus, not all would agree that gamma globulin should be used in patients exposed to rubella. Dührssen's incisions and prophylactic forceps for the delivery of twins are not common practices in many areas. The abdominal approach for circlage of the incompetent cervix is not advocated by many.

There is a very helpful division between those conditions in which much is to be gained by prolonging the pregnancy, and those conditions in which it is important to rescue the fetus from an unfavorable environment.

Thus, in chapter 5, such conditions as multiple pregnancy, placenta previa, incompetent cervix, slight premature placental separation and thyroid dysfunction are discussed. In Chapter 6, one may read about conditions in which early delivery may be advisable—such as premature rupture of membranes, toxemia, premature separation of the placenta, erythroblastosis fetalis, diabetes and urinary tract infection.

The section concerning management of the infants is well written and complete. It contains several excellent tables for quick reference, such as Table 12, Drugs, Nutrients, and Blood Replacements and Their Dosage and Table 15, Biochemical Values For The Premature Infant. There is excellent coverage of the differential diagnostic possibilities of most of the symptoms exhibited by newborn infants.

There is a wealth of information in this brief publication, and it is most helpful to have it all brought together and correlated so well. Ordinarily, one must consult a great number of sources to acquire this same information. The practical suggestions are many and clear cut. This volume will be of great assistance and value to all physicians caring for obstetrica patients, or their infants.

* * *

CLINICAL OBSTETRICS AND GYNECOLOGY—Volume 9, Number 3, September 1966—Teratology, edited by L. C. R. Carrington, M.D., and Adolescent Gynecology, edited by Russell Ramon de Alvarez, M.D. Published quarterly by Hoeber Medical Division, Harper and Row, Publishers, New York. Subscription $18.00 per year.

The series of articles on teratology is a rather successful attempt to bring up-to-date what is known about this important subject. The introductory article by Peter Gruenwald discusses basic mechanisms of teratogenesis at the embryological level. The mechanisms discussed will not be familiar to all readers of this volume but are helpful in our thinking of how defects develop. The discussion on fetal risk associated with maternal systemic infection by Ralph Moloschok is a very helpful and succinct discussion of this controversial subject. While most of the emphasis is rightly given to the discussion of rubella other infections commonly acquired by the obstetric patient are also discussed with respect to the fetus. Our serious lack of knowledge about the effects of drugs on the intrauterine patient is pointed out by Dr. Virginia Apgar. Some mention is also made of other harmful categories of drugs, but this article is by no means exhaustive in its discussion. The reader will find his appetite whetted for more specific information on the danger of drugs in pregnancy and what abnormalities may result. The emphasis on the difficulty in applying animal data to humans is creditable. The discussion on the teratogenic phase of radiation is helpful and tries hard to define the minimum radiation dose which can damage fetal neuroblasts, the most radiosensitive cells. Perhaps the best in this series of articles is by Auerbach and Digeorge on the subject of inborn errors of metabolism. This greatly expanding field is of increasing interest to obstetrician and pediatrician alike. In the not so distant past diabetes was the major if not the only inborn error with which the obstetrician had to deal, but the authors point out that this is no longer the case and conditions such as phenylketonuria, cystic fibrosis, virilizing adrenal hyperplasia and numerous others will be complicating pregnancy in increasing numbers. Included is a listing of the inborn errors known at present which requires five pages merely to enumerate. Another paper summarizing chromosome abnormalities is included and will be found quite adequate. For those who have not yet familiarized themselves with this expanding field the article is recommended. The author discusses briefly the subject of chromosomal defects in human abortuses. Genetic counseling is quite adequately handled except that the subject can hardly be covered in a single short article and the remarks can be considered no more than introductory. Taken as a whole this series of articles will be of great interest to the physician dealing with the reproductive female. For the most part the information is supplied at the practical level and indeed supplies answers which patients are daily seeking from their obstetrician or pediatrician. The symposium on adolescent gynecology attempts to look at the developing female from seerval points of view. The initial discussion by Michael Joseph Daly on physical and psychological development in the adolescent female is surprisingly practical and thought provoking. He expresses, no doubt, a personal opinion but a well thought out one. Dr. Carrington succinctly summarizes the problem of vaginitis in the adolescent although no new information is presented. Also provided, somewhat redundantly, is discussion of the genetics of sexuality and intersexuality. The outstanding article in this group appears to be that by Lundy in which he deals with endocrinology of adolescence and the interplay of the other endocrine organs including the hypothalamus, pituitary, adrenal cortex and ovaries. Those who have not been in close touch with these subjects recently will find this discussion to be of outstanding interest. No new information on disorders of menstruation or gynecologic tumors in adolescence is presented although subjects are dealth with in a very readable fashion. In general the symposium on adolescent gynecology is worth while although not packed with new information.

Leo D. Lagasse, M.D.

* * *

DISORDERS OF THE RESPIRATORY TRACT IN CHILDREN (by 29 Authorities)—Edited by Edwin L. Kendig, Jr., M.D., Professor of Pediatrics, Medical College of Virginia; Director of Child Chest Clinics, Medical College of Virginia Hospitals, Richmond. W. B. Saunders Company, Philadelphia and London, 1967. 834 pages, $26.00.

This newly published volume is edited by Dr. Edwin Kendig, Professor of Pediatrics at the Medical College of Virginia. Dr. Kendig has personally contributed chapters on tuberculosis, sarcoidosis and diseases caused by atypical mycobacteria. He has also lined up an impressive panel of 29 authorities to write the other chapters. These include such familiar names as Mary Ellen Avery who wrote the opening chapter on respiratory physiology in collaboration with Victor Chernick, Harry Schwachman on cystic fibrosis, Amos Christie on histoplasmosis, Margaret Smith on bacterial pneumonias, and many, many others.

The design of the book is to cover physiology of respiration, first, then aspects of history, physical examination, special diagnostic and therapeutic procedures including the roentgenogram. The neonate is dealt with in a separate chapter by Mildred Stahlman. After that the various disorders of the upper and lower respiratory tract are covered in considerable detail. There are 67 chapters and 837 pages.

On the whole the approach is that of a good textbook of pediatrics but in a great deal more detail than most textbook editors would permit. The book is lavishly illustrated though entirely in black and white. There are many tables, graphs and diagrams. Among them are full page, very legible diagrams of distribution of lobar and segmental bronchi, position of children for bronchography as well as for bronchial drainage, a table of tracheostomy tube sizes suitable for various ages, and many, many others. The style of writing varies with the different authors as one would expect. On the whole almost all the chapters are quite up to date. Most authors provide a lengthy and updated list of references for further reading.

A single textbook of pediatrics no longer seems to be able to deal adequately with the various subspecialty areas. The cardiovascular system, the endocrine system and many others have been covered by separate texts which are becoming fairly standard. This book does the same service for the respiratory tract. The book is thoroughly recommended for hospital libraries, clinics and those physicians who can afford to spend $26.00 for a text covering one body system in children. They will find this book very worthwhile.

* * *

APPRAISAL OF CURRENT CONCEPTS IN ANESTHESIOLOGY—Volume 3—Edited and assembled by John Adriani, M.D., Professor of Surgery, Tulane University School of Medicine, New Orleans, La.; Clinical Professor of Surgery and Pharmacology, Louisiana State University Schoool of Medicine, New Orleans, La.; Director, Department of Anesthesiology, Charity Hospital of Louisiana, New Orleans, La. 522 pages, $10.85.

Volume three is in the same format as that used for volumes one and two published in 1961 and 1964 respectively. The contents are composed of 50 short reviews of various current subjects prepared by 28 members from the Anesthesia Department of Charity Hospital of Louisiana. The reviews have been capably edited by Dr. John Adriani and are intended to bridge the time gap between presentation of medical information as articles in medical journals, each usually dealing with only a narrow aspect of the broad subject, and later incorporation in more coordinated monographs and textbooks. As explained in the preface, the presentations are not intended to be exhaustive reviews but are planned for the clinician who desires "to be brought up to date" on a pertinent subject without expending the time required for searching out a large number of articles. A reasonable list of references appears at the end of each subject so an excellent beginning for further investigation is available. Through this well indexed, handy sized volume of 503 pages, a reader can participate in the seminar program of a well organized anesthesiology residency.

John W. Pender, M.D.

* * *

SCIENTIFIC YEARLY OF THE LINCOLN CHIROPRACTIC COLLEGE—1966, Volume I—By Earl A. Rich, D.C., Editor. Owned and published by the Lincoln Chiropractice College as its official scientific journal, Indianapolis.

Cineradiography has recently been introduced by the chiropractors as a diagnostic method. In the report by Rich on the cineroentgenological observations of the human systems, teleologic reasoning is evident based on rather inadequate observations and without any supporting evidence of anatomic or pathologic nature. This is illustrated in the comments regarding "hypermotoricity" in degenerative change involving intervertebral discs. The basic work by Fielding regarding mobility of the cervical spine has been overlooked.

The discussion on the chiropractic management of diseases of the heart incorporates printed material available to the public by the various heart associations regarding the overall management of heart disease. The chiropractic management of patients with heart disease is not a specific but, apparently, a general relaxing experience, not dissimilar to massage and rubdowns, designed to relieve physicial expression of disease and tension.

In the sections on lumbosacral degeneration and postural disorders, adequate references are made to supporting authorities. Postulates of the actual internal derangements or anatomic defects which exist remain undocumented. The categorization of permanent versus reversible subluxations in the lumbosacral spine is arbitrary and again lacks adequate documentation. In postural disorders, much emphasis is placed on what is considered the neurofunctional cause of scoliosis and the chiropractic methods which should be directed toward correcting this. While some basic orthopedic management is described, such as heel lifts for short legs, chiropractic measures are described "for the mechanical and neural integration of morphopathologically altered vertebral structures like in osteoarthritis of the pelvis and the vertebral column" or "in secondary neuromuscular and vertebral symptoms due to irreversible and dynamically inadaptable disorders of posture as in the occipital neuralgia of Bechterew's disease." In the same area, arteriosclerosis, particularly of the cervical arteries, is described as a disease that is rather often neglected in the judgment of chiropractic indications. Substantiating evidence of the efficacy of chiropractic therapy is missing.

The section on the functional radiography of the cervical spine reports extensive studies of small groups of patients divided into multiple categories. The method of angular mensuration used had been previously reported and relates total cervical spine motion to a baseline of the cervico-thoracic junction. This method suffers from the unreliability of impact of shoulder height on the total cervical mobility and reflects minor degrees of voluntary guarding. Evaluation of normal range of mobility is not described in completely normal individuals, although 50 patients without cervical complaint formed the baseline for comparison. Approximately a 3° greater mobility was observed in this "normal group" interpreted as indicating presence of hypertonicity of anterior neck musculature or a static imbalance of the spine in those cases suffering from neck complaints. Interpretation of the pre-manipulation and post-manipulation radiographs taken in flexion and extension is reported as indicative of the effectiveness of chiropractic management. Without a control group of similarly aged individuals with cervical symptomatology followed for a similar length of time but without manipulation, substantiation of effectiveness of the chiropractic method is not acceptable.

In the section on precoxarthrosis and coxarthrosis incipient, by Lindner, a reasonably complete listing of varying abnormalities of the hip is given with relatively complete description of radiologic findings in the standard diseases of the hip. The therapeutic approach is summarized. "Whereas the great majority of precoxarthrotic conditions are cases for the orthopedic specialist, coxarthrosis is of direct interest to the chiropractor. The therapeutic endeavors will have to include measures to relieve the pain, restoration of normal or almost normal joint function, and proper advice to the patient as to his future behavior." Diathermy, electrotherapy, vibrations, spinal manipulations, passive, and, finally, active exercises all are listed as methods of approach in coxarthrosis.

Malcolm D. Jones, M.D.

A SURVEY OF SURGICAL SHOCK WITH SPECIAL REFERENCE TO FLUIDS, ELECTROLYTES AND METABOLITES—By Joseph H. Pollock, B.S., M.D., F.I.C.S., Cedars of Lebanon Hospital, Los Angeles, Calif., Charles C Thomas, Publisher, Springfield, Ill., 1966. 710 pages, $28.75.

The stated purpose of the author in developing this monograph is to provide a readily available and easily understandable reference source on the present knowledge of surgical shock. Dr. Pollock, in introducing the book, states that it is his intent to survey the subject from the practitioner's point of view and to derive an "intelligent approach."

The author's intent is ambitious and the more than 700 pages of text reflect devoted interest and study. However, the stated purpose is not achieved. The text reads like a student's notebook, incorporating page length extractions from various sources, including Medical World news, Antibiotic News and Medical Tribune. Little has been predigested and the practitioner will search in vain for practical guides systematically presented. The volume contains errors of interpretation and errors of fact. For example, the author regards the measurement of serum angiotensinase and determination of cardiac output by thermal dilution technique as foremost among tests to be used preoperatively in the prevention of shock. The definitions of alkalosis and plasma volume, among others, are incorrect. There is an extensive bibliography which might attract the serious student; yet, the unnumbered bibliography stands by itself and the citations of published works, which form the very basis of the survey, are essentially undocumented.

* * *

CIBA FOUNDATION SYMPOSIUM—The Thymus: Experimental and Clinical Studies in honour of Sir MacFarlane Burnet, O.M. Edited by G. E. W. Wolstenholme, O.B.E., M.A., F.R.C.P., F.I.Biol., and Ruth Porter, M.R.C.P. Little, Brown and Company, Boston, Mass., 1966. 538 pages, $15.00.

Within the past decade the thymus has emerged from a lymphoid organ of uncertain status to one of prime importance in a functioning immune system. However, in spite of intense investigation on the role of the thymus, there are still many more unresolved problems than clearly defined functions. The *Ciba Foundation Symposium: The Thymus: Experimental and Clinical Studies* attempts to resolve some of the divergent views by presenting an outstanding compilation of recent research by leading investigators. This book is designed primarily for individuals with a basic interest in the investigational aspects of the thymus. Except for the last section which deals with the human thymus in pathological states, the book is oriented towards current animal research rather than clinical problems. For the interested reader there are enlightening reports dealing with many areas of thymic physiology including the thymus as a humoral organ, the function of the neonatal and adult thymus, cellular traffic through the thymus, the relation of the thymus to peripheral lymphatic tissues and the role of the thymus in both animal leukemia and autoimmune disorders. Most of the invited participants presented data on current research and attempted to integrate this into a composite picture covering all aspects of thymic function. As with any series of reports, some are of excellent quality, introducing either new concepts or presenting new data to support previously advanced hypotheses. Others cover nothing more than a repetition of data or concepts previously published. Fortunately, most of the papers represent the former and their data should help to clarify certain aspects of thymic physiology. At the same time, these reports raise some new and interesting issues which remain to be adequately explained. One of the most interesting features of this symposium is the critical discussion by noted authorities which follows each paper. These are frequently as illuminating as the report itself. For the person interested in recent advances in this stimulating field of investigation, I recommend this symposium as an outstanding colloquium on the role of the thymus.

ALAN WINKELSTEIN, M.D.

* * *

CLINICAL MANAGEMENT OF BEHAVIOR DISORDERS IN CHILDREN — Third Edition, illustrated — By Harry Bakwin, M.D., Professor of Clinical Pediatrics, New York University; Visiting Physician, Bellevue Hospital; and Ruth Morris Bakwin, M.D., Professor of Clinical Pediatrics, New York University; Visiting Physician, Bellevue Hospital; Director Emeritus, Department of Pediatrics, New York Infirmary. W. B. Saunders Company, Philadelphia and London, 1966. 652 pages, $14.50.

The Bakwins, husband and wife, have produced a worthy third edition. The first edition was published in 1953 and the second seven years later. The present edition follows the second by six years. This edition is larger than the previous ones and numerous changes have been made, although the organization is the same. There is considerable new information and up-to-date references have been included. The Bakwins present a critical and systematic appraisal of normal and abnormal behaviour in the child. They have added their own evaluation of the vast literature. They report a long and carefully documented personal experience. The book presents a "common-sense" approach, and is comprehensive, practical and easily read.

BENJAMIN M. KAGAN, M.D.

"Yes, Doctor, the pain is gone."

🟥 Despite introduction of synthetic substitutes, efficacy of 'Empirin' Compound with Codeine remains unchallenged.

'Empirin'® Compound with Codeine Phosphate gr. ½ No. 3

Each tablet contains: Codeine Phosphate gr. ½ (Warning—May be habit forming), Phenacetin gr. 2½, Aspirin gr. 3½, Caffeine gr. ½.

Keeps the Promise of Pain Relief

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N. Y.

REFERENCES AND REVIEWS

PERCUTANEOUS NEEDLE—J. H. Bates, and J. S. Adamson, Jr. (VA Hosp., Little Rock, Ark.), Arch. Intern. Med., 119:164-169 (February) 1967.

A report of 71 percutaneous lung biopsies performed in 62 patients is presented. Tissue adequate for examination was obtained in 83 per cent of the patients. Complications consisted of one death, 12 pneumothoraces, and three instances of hemoptysis.

* * *

HYDROTIC ECTODERMAL DYSPLASIA: CLOUSTON'S FAMILY REVISITED—M. Williams, and F. C. Fraser (Human Genetics Sector, McGill Univ., Montreal), Canad. Med. Assoc. J., 96:36-38 (January 7) 1967.

Several families segregated for hydrotic ectodermal dysplasia are described. Included are the later generations of a family originally reported by Clouston (1929). No deviations from an autosomal dominant pattern of inheritance were found.

PHYSICIAN PLACEMENT SERVICE

The California Medical Association offers free placement assistance through the Physician Placement Service, 693 Sutter Street, San Francisco, California 94102. This service is for the use of all physicians seeking practice opportunities in California and for CMA members who are seeking an assistant or associate. A bulletin is published every other month. Opportunities in the Bulletin must be sponsored by a CMA member, an accredited California hospital or responsible persons in a California community. The Bulletin does not carry listings describing real estate for sale or office space for rent or lease or blind listings with box numbers.

* * *

NASOPHARNYGEAL CARCINOMAS IN CHILDREN—R. H. Nishiyama, J. G. Batsakis, and D. K. Weaver (1405 E. Ann St., Ann Arbor, Mich.), Arch. Surg., 94:214-217 (February) 1967.

Nasopharyngeal carcinomas are reported in nine children with ages ranging from 7 to 15 years at the time of histological diagnosis. The lymphoepithelioma is the most common carcinoma of the nasopharynx in childhood. Radio therapy is the treatment of choice. However, prognosis is best gauged by clinical staging of the disease rather than the cell type. An interesting and possibly significant finding in childhood nasopharyngeal carcinomas is the relatively high familial incidence of the neoplasm.

* * *

AN ELECTRON MICROSCOPIC STUDY OF MYOTONIC DYSTROPHY—G. H. Klinkerfuss (Washington Univ. School of Medicine, St. Louis), Arch. Neurol., 16:181-193 (February) 1967.

Light and electron microscopic study of eight muscle biopsies from patients with myotonic dystrophy demonstrated coarse filamentous destruction of myofilaments which usually extend from Z disks and may involve all or part of a fibril. Other fibers showed proliferation of myofilaments to form new sarcomeres which are randomly arranged to the long axis of the fiber. The newly formed fibrils gave the appearance of "Ringbinden" when appropriately sectioned and can explain muscle fiber hypertrophy and central nuclei.

CLASSIFIED ADVERTISEMENTS

USE THIS ORDER BLANK TO ADVERTISE PHYSICIAN OPPORTUNITIES, SITUATIONS WANTED, PRACTICES, OFFICES, REAL ESTATE, EQUIPMENT, ETC.

CLOSING DATE for copy and advance payment: 5th of month PRECEDING date of issue.

RATES PER INSERTION: $10.00 for the first 50 words or less, 10c for each additional word; box number charge of 50c.

California Medicine • 693 SUTTER STREET, SAN FRANCISCO, CALIFORNIA 94102

Name____________________ Address____________________

Enclosed is $__________ for __________ (No. Mos.) insertion(s).

Check one: ☐ **Please include my name and address in ad**
☐ **Please assign a box number (50c additional for each month's insertion)**

Photo professionally posed

**Mike expects a penicillin injection.
He's about to be pleasantly surprised.**

His physician is going to prescribe an oral penicillin—Pen • Vee® K (potassium phenoxymethyl penicillin). It's usually so rapidly and completely absorbed that therapeutic serum levels are produced in 15 to 30 minutes. Higher serum levels generally last longer than with oral penicillin G.

Indications: Infections due to pathogens susceptible to oral penicillin G. Prophylaxis of rheumatic fever in patients with previous history of the disease.
Precautions: Skin rash, symptoms resembling those of serum sickness, or other manifestations of penicillin-allergy may occur. Measures for treating anaphylaxis should be readily available: epinephrine, oxygen and pressor drugs for relief of immediate allergic reactions; antihistamines and corticosteroids for delayed effects. Penicillin may delay or prevent the appearance of primary syphilitic lesions. Patients with gonorrhea who are suspected of concurrent syphilitic infections should be tested serologically for at least 3 months. Where lesions of primary syphilis are suspected, dark-field examination should precede use of penicillin. As with other antibiotics overgrowth of nonsusceptible organisms may occur; if so, discontinue and take appropriate measures. Treat β-hemolytic streptococcal infections with full therapeutic dosage for at least 10 days to prevent development of rheumatic fever or glomerulonephritis.

Contraindications: Infections caused by nonsusceptible organisms; history of penicillin sensitivity.

Composition: Tablets—125 mg. (200,000 units) and 250 mg., (400,000 units); Liquid—125 mg. (200,000 units) and 250 mg. (400,000 units) per 5 cc. Wyeth Laboratories Philadelphia, Pa.

ORAL **PEN · VEE® K**
(potassium phenoxymethyl penicillin) Wyeth®

Officers OF THE CALIFORNIA MEDICAL ASSOCIATION

General Officers

President: JAMES C. MACLAGGAN, 525 Hawthorn Street, San Diego 92101. 714 233-6519
President-Elect: JOHN G. MORRISON, 333 Estudillo, San Leandro 94577. 415 483-1332
Past President: RALPH C. TEALL, 2626 L Street, Sacramento 95816. 916 448-1851
Speaker of the House of Delegates: WILLIAM F. QUINN, 1401 S. Hope Street, Los Angeles 90015. 213 749-0181
Vice-Speaker of the House of Delegates: JOSEPH W. TELFORD, 3255 Fourth Avenue, San Diego 92103. 714 295-5151
Chairman of the Council: CARL E. ANDERSON, 1150 Montgomery Drive, Santa Rosa 95405. 707 546-1922
Secretary: HELEN B. WEYRAUCH, 450 Sutter Street, San Francisco 94108. 415 392-6909
Editor: DWIGHT L. WILBUR, 655 Sutter Street, San Francisco 94102. 415 673-4080

Executive Director: HOWARD HASSARD, 693 Sutter Street, San Francisco 94102. 415 776-9400
General Counsel: PEART, HASSARD, SMITH and BONNINGTON, 44 Montgomery Street, San Francisco 94104. 415 982-8585

* * *

District Councilors

- FIRST DISTRICT—Roger C. Isenhour, (1967), 2881 Fourth Avenue, San Diego 92103. San Diego County. 714 295-2137
- SECOND DISTRICT—Imperial, Inyo, Mono, Orange, Riverside and San Bernardino Counties—
  - Office No. 1—Henry V. Eastman (1967), 661 W. First Street, Tustin 92680. 714 544-4560
  - Office No. 2—Frank C. Melone (1968), 527 N. Palm Avenue, Ontario 91761. 714 986-1104
- THIRD DISTRICT—Los Angeles County—
  - Office No. 1—Malcolm C. Todd (1968), 2865 Atlantic Avenue, Long Beach 90806. 213 426-1355
  - Office No. 2—Elmer F. Gooel (1969), 465 N. Roxbury Drive, Beverly Hills 90210. 213 271-5745
  - Office No. 3—Richard L. Taw (1967), 3741 Stocker Street, Los Angeles 90008. 213 293-7157
  - Office No. 4—Lewis T. Bullock (1968), 3875 Wilshire Boulevard, Los Angeles 90005. 213 382-2209
  - Office No. 5—Joseph P. O'Connor (1969), 595 E. Colorado Boulevard, Pasadena 91101. 213 792-2154
  - Office No. 6—Franklin F. Ham (1967), 14556 Hamlin Street, Van Nuys 91401. 213 786-5711
  - Office No. 7—Wilbur G. Rogers (1968), 1122 N. Brand Boulevard, Glendale 91202. 213 241-3125
  - Office No. 8—Jean F. Crum (1969), 8355 E. 3rd Street, Downey 90241. 213 869-1091
  - Office No. 9—Joseph F. Boyle (1967), 1136 W. Sixth Street, Los Angeles 90017. 213 482-9891
- FOURTH DISTRICT—Joseph F. Maguire (1967), 2755 Loma Vista Road, Ventura 93003. San Luis Obispo, Santa Barbara and Ventura Counties. 805 648-2567
- FIFTH DISTRICT—Ralph W. Burnett (1969), 515 Truxton Avenue, Bakersfield 93301. Calaveras, Fresno, Kern, Kings, Madera, Mariposa, Merced, San Joaquin, Stanislaus, Tulare and Tuolumne Counties. 805 327-4411
- SIXTH DISTRICT—Monterey, San Benito, San Mateo, Santa Clara and Santa Cruz Counties.
  - Office No. 1—Richard S. Wilbur (1967), 300 Homer Avenue, Palo Alto 94301. 415 321-4121
  - Office No. 2—Albert G. Miller (1968), 77 San Mateo Drive, San Mateo 94401. 415 343-9074
- SEVENTH DISTRICT—San Francisco County—
  - Office No. 1—Malcolm S. M. Watts (1969), 909 Hyde Street, San Francisco 94109. 415 776-3838
  - Office No. 2—Roberta F. Fenlon (1967), 490 Post Street, San Francisco 94102. 415 986-1391
- EIGHTH DISTRICT—Alameda and Contra Costa Counties—
  - Office No. 1—Harold Kay (1969), 411 Thirtieth Street, Oakland 94609. 415 832-4822
  - Office No. 2—William F. Kaiser (1967), 3000 Colby Street, Berkeley 94705. 415 843-6515
- NINTH DISTRICT—Carl E. Anderson (1967), 1150 Montgomery Drive, Santa Rosa 95405. Del Norte, Humboldt, Lake, Marin, Mendocino, Napa, Solano and Sonoma Counties. 707 546-1922
- TENTH DISTRICT—James H. Yant (1968), 2600 Capitol Avenue, Sacramento 95816. Alpine, Amador, Butte, Colusa, El Dorado, Glenn, Lassen, Modoc, Nevada, Placer, Plumas, Sacramento, Shasta, Sierra, Siskiyou, Sutter, Tehama, Trinity, Yolo and Yuba Counties. 916 446-5278
- ELEVENTH DISTRICT—Forest J. Grunigen (1967), 3919 Beverly Boulevard, Los Angeles 90004. Forty First Medical Society. 213 663-1161

SCIENTIFIC BOARD REPRESENTATIVE—William P. Longmire, Jr. (1967), UCLA Department of Surgery, Los Angeles 90024. 213 272-8911

* * *

COMMISSION ON *Medical Services*

Ralph Burnett, Bakersfield
Elmer F. Gooel, Beverly Hills (Secty.)
Arthur F. Howard, Fresno (Chmn.)
Charles B. Hudson, Oakland
Homer C. Pheasant, Los Angeles
Milo A. Youel, San Diego.

Committee on Fees

H. Dean Hoskins, Oakland	1967
Wallace A. Gerrie, Jr., Newport Beach	1967
Robert L. Watson, Jr., Los Angeles	1967
Roger C. Isenhour, San Diego	1968
Alfred J. Murrieta, Jr., Los Angeles	1968
Maurice S. Salomon, San Francisco	1968
Homer C. Pheasant, Los Angeles (Chmn.)	1969
Glenn A. Pope, Sacramento	1969
Leo Bell, San Mateo	1969
Joseph F. Boyle, Los Angeles	1969

CONSULTANT: William H. Thompson, San Mateo.

Committee on Federal Medical Care Programs

Charles B. Canby, Van Nuys	1967
Merlin A. Hendrickson, Rialto	1967
Ralph L. Hoffman, San Diego	1967
John S. McKee, Jr., San Diego	1968
Arthur Howard, Fresno (Chmn.)	1968

Committee on Veterans Affairs

Charles B. Hudson, Oakland (Chmn.)
Ben D. A. Miano, San Bernardino
Robert L. Watson, Los Angeles
CONSULTANT: Lloyd E. Gould, Napa.

Committee on Mediation

Eli A. Layon, Brawley	1967
Milo A. Youel, San Diego (Chmn.)	1967
Richard L. Taw, Los Angeles	1968
Albert G. Clark, San Francisco	1969
George Babbin, Sacramento	1969

Committee on Insurance and Prepayment

Oscar W. Hills, San Mateo	1967
Dave F. Dozier, Sacramento	1967
Ralph W. Burnett, Bakersfield (Chmn.)	1968
Stanley J. Murasky, Reseda	1968
Ivan Neubauer, San Jose	1969
Thayer A. Smith, Norwalk	1969

* * *

COMMISSION ON *Public Agencies*

Lewis T. Bullock, Los Angeles
Joseph P. Cosentino, Sacramento (Secty.)
William F. Kaiser, Berkeley
Howard W. Lindsey, Jr., San Mateo
Albert Miller, San Mateo (Chmn.)
Stanley A. Skillicorn, San Jose
Howell E. Wiggins, San Diego
Joseph P O'Connor, Pasadena
CONSULTANT: Malcolm C. Todd, Long Beach.

Committee on Public Health

Robert L. Garrett, Vallejo	1967
Karl L. Schaupp, Jr., San Francisco	1967
Allen F. Sterling, San Bernardino	1967
Austin W. Matthis, Sacramento	1967
Albert G. Miller, San Mateo	1968
Stanley A. Skillicorn, San Jose (Chmn.)	1968
Alfred F. Kandlbinder, Monterey	1969
Robert Watson, Jr., Los Angeles	1969
Samuel S. Woolington, Long Beach	1969

Committee on Mental Health

Emmanuel Silver, Palo Alto	1967
Ira H. Degenhardt, San Rafael	1967
Rudolph B. Toller, Stockton	1967
John A. Stroud, III, Sacramento	1967
Alfred Auerback, San Francisco	1968
David H. Payne, Los Angeles	1968
Jack B. Lomas, Beverly Hills	1968
Joseph J. Furlong, San Francisco	1969
Stuart C. Knox, Los Angeles (Chmn.)	1969

Committee on Welfare Medical Care Programs

Clyde L. Boice, Palo Alto	1967
Edgar Rosen, Oakland	1967
Thomas N. Elmendorf, Willows	1967
Vincent P. Carroll, Laguna Beach	1967
Orrin S. Cook, Jr., Sacramento	1968
Robert F. Schell, San Rafael	1968
William F. Quinn, Los Angeles	1969
James Dalton, Santa Barbara	1969
Arthur F. Howard, Fresno	1969
Howard W. Lindsey, Jr., San Mateo (Chmn.)	1969

SUBCOMMITTEE ON ADOPTIONS: Walter Beckh, San Francisco; Fred M. Kay, Fullerton; Robert J. McNeil, Los Angeles, (Chmn.); Donald Minkler, Berkeley; Dale W. Ritter, Chico; Ben J. Siebenthal, Sacramento.

Committee on Occupational Health and Rehabilitation

John H. Leimbach, Jr., San Francisco	1967
Herbert A. Holden, San Leandro	1967
Howell E. Wiggins, San Diego (Chmn.)	1967
Edward Zaik, Los Angeles	1968
Packard Thurber, Jr., Los Angeles	1968
Thomas Gucker, III, Los Angeles	1968
Herbert A. Lints, Oakland	1969
Joseph N. Vizzard, Los Gatos	1969
Patrick J. Maloney, Merced	1969

* * *

COMMISSION ON *Community Health Services*

Wayne P. Chesbro, Berkeley
Charles G. Hutter, Jr., Hollywood
Gerald G. Jampolsky, Tiburon (Secty.)
Elmer R. Jennings, Long Beach
Harold Kay, Oakland (Chmn.)
George A. Martin, Redding
Pierre Salmon, San Mateo
Leopold J. Snyder, Fresno
Beverly G. Stewart, Bakersfield

Committee on Rural Health

Mitry P. Ajalat, Calexico	1967
Richard W. Johnson, Arbuckle	1967
Leopold J. Snyder, Fresno (Chmn.)	1968
Erwin P. Brauner, Visalia	1968
Carrol W. Goss, Lamont	1968
Franklin L. Murphy, Oroville	1969
Thomas Rossitto, Auburn	1969
M. Marlin Clark, Delano	1969

Committee on School Health

B. Otis Cobb, Martinez	1967
Gerald G. Jampolsky, Tiburon (Chmn.)	1967
Byron H. Demorest, Sacramento	1967
William A. Leovy, La Mesa	1968
Tom W. Robinson, Newport Beach	1968
Glenn Austin, Los Altos	1968
Ruth A. Frary, Watsonville	1968
Leslie M. Holve, Los Angeles	1969
Henry B. Bruyn, Berkeley	1969

Committee on Health Care of the Aged

Franklin F. Ham, Van Nuys	1967
Pierre Salmon, San Mateo (Chmn.)	1967
Charles E. Schoff, Jr., Sacramento	1968
Raymond D. Manchester, Visalia	1968
Leo A. Wagner, Novato	1969
Lloyd E. Gould, Napa	1969
Walter Batchelder, San Mateo	1969

Committee on Disaster Medical Care

Charles C. Henderson, San Mateo	1967
Donald E. Barker, Van Nuys	1967
Wayne P. Chesbro, Berkeley (Chmn.)	1968
Michael J. Hitchko, Eureka	1968
J. T. McNally, Stockton	1969
John Heald, San Francisco	1969

CONSULTANTS: Charles N. Earl, Arcata; Frank F. Schade, Los Angeles; Robert S. Johnson, Chico; Stephen Bruny, Fresno.

Committee on Automotive and Traffic Safety

George C. Snively, Sacramento	1967
Beverly G. Stewart, Bakersfield (Chmn.)	1967
Arnold R. Friesen, Pasadena	1968
Joseph F. Maguire, Ventura	1969
Thomas W. Lyons, La Mesa	1969
Robert L. Tour, San Francisco	1969

CONSULTANTS: Julian A. Waller, Berkeley; Julian M. Sether, Los Angeles.

Committee on Medical Aspects Of Sports and Physical Fitness

John F. Blinn, Stockton	1967
Robert K. Kerlan, Los Angeles	1967
George S. Buehler, Whittier	1968
Philip H. McFarland, Fullerton	1968
Tom W. Robinson, Newport Beach	1968
Charles G. Hutter, Jr., Hollywood (Chmn.)	1969
Arnold A. Nutting, San Rafael	1969

Committee on Environmental Health

George A. Martin, Redding	1967
Robert L. Hippen, San Diego	1967
Grace M. Talbott, San Francisco	1968
Francis M. Pottenger, Jr., Monrovia	1968
Reginald H. Smart, Los Angeles	1968
Douglas Campbell, San Francisco	1969
John R. Lee, Mill Valley	1969

Committee on Blood Banks

Robert W. Purvis, Modesto	1967
Osman W. Hull, Monterey	1967
James H. Yant, Sacramento	1967
Elmer Jennings, Long Beach (Chmn.)	1968

CONSULTANT: Melvin L. Sommer, Beverly Hills

COMMISSION ON *Communications*

Frederick Ackerman, Pleasant Hill
L. Morgan Boyers, Sacramento (Secty.)
John A. E. Bullis, Los Angeles
Roberta F. Fenlon, San Francisco
Elmer F. Gooel, Beverly Hills
Walter W. Hopps, Los Angeles
George Schwartz, Beverly Hills
Malcolm C. Todd, Long Beach (Chmn.)
Richard S. Wilbur, Palo Alto
EX-OFFICIO: James C. MacLaggan, San Diego; John G. Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Scientific Board

David A. Rytand, Palo Alto	1967
William W. Belford, San Diego	1967
Jerome W. Bettman, San Francisco	1967
John B. Dillon, Los Angeles	1967
Justin J. Stein, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
R. Kirklin Ashley, San Francisco	1967
Victor Richards, San Francisco	1967
David Rubin, Los Angeles	1967
Keith P. Russell, Los Angeles	1967
Edward J. Zaik, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Seymour J. Brockman, Los Angeles	1968
S. Austin Jones, Los Angeles	1968
Hobart M. Kelly, Riverside	1968
Saul J. Robinson, San Francisco	1968
Eugene Ziskind, Los Angeles	1968
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. deC. M. Saunders, San Francisco	1968
Herbert A. Holden, San Leandro	1968
David A. Wood, San Francisco	1968
Arthur A. Clinco, Los Angeles	1968
R. Theodore Bergman, Los Angeles	1968
Leland B. Blanchard, San Jose	1969
L. S. Goerke, Los Angeles	1969
Bert L. Halter, San Francisco	1969
Ellis Jones, Pasadena	1969
Carl McCandless, San Francisco	1969
J. Blair Pace, Oceanside	1969
Donald W. Petit, Los Angeles	1969
Glenn Pope, Sacramento	1969
Robert J. Prentiss, San Diego	1969
Edward B. Shaw, San Francisco	1969
Ralpb E. Worden, Los Angeles	1969

COUNCIL REPRESENTATIVES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.
EX-OFFICIO: Dwight L. Wilbur, San Francisco.

Executive Committee

William W. Belford, San Diego	1967
Victor Richards, San Francisco	1967
Justin J. Stein, Los Angeles	1967
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. Dillon, Los Angeles	1969
Donald Petit, Los Angeles	1969

COUNCIL DESIGNEES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.

Committee on Nominations

Justin J. Stein, Los Angeles (Chmn.)	1967
Edward B. Shaw, San Francisco	1969
Robert J. Prentiss, San Diego	1969

Committee on Scientific Assemblies

John B. Dillon, Los Angeles (Chmn.)	1967
Victor Richards, San Francisco	1967
David A. Rytand, Palo Alto	1967
John B. deC. M. Saunders, San Francisco	1968
Ellis Jones, Pasadena	1969
Donald Petit, Los Angeles	1969

CONSULTANT: Claron Oakley, Los Angeles

Committee on Continuing Medical Education

John B. Dillon, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
Saul J. Robinson, San Francisco	1968
Donald W. Petit, Los Angeles (Chmn.)	1969
Bert L. Halter, San Francisco	1969
J. Blair Pace, Oceanside	1969

Committee on CALIFORNIA MEDICINE

David Rubin, Los Angeles	1967
R. Kirklin Ashley, San Francisco	1967

Committee on Scientific Information

Justin J. Stein, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Arthur A. Clinco, Los Angeles	1968
Edward B. Shaw, San Francisco (Chmn.)	1969
L. S. Goerke, Los Angeles	1969

Committee on Maternal and Child Care

Russell W. Mapes, Beverly Hills	1967
Keith P. Russell, Los Angeles (Chmn.)	1967
Forrest H. Howard, Garden Grove	1967
Charles M. Blumenfeld, Sacramento	1968
Herbert A. Holden, San Leandro	1968
Hobart M. Kelley, Riverside	1968
Leland Blancbard, San Jose	1969

Committee on Cancer

John W. Cline, San Francisco	1967
Robert W. Jamplis, Palo Alto	1967
David A. Wood, San Francisco (Chmn.)	1967
John G. Walsh, Sacramento	1968
Lewis W. Guiss, Los Angeles	1969
Robert Prentiss, San Diego	1969

SUBCOMMITTEE ON CANCER EDUCATION: Sol R. Baker, Beverly Hills; Robert W. Jamplis, Palo Alto (Chmn.); Maurice Galante, San Francisco; Justin J. Stein, Los Angeles.

SUBCOMMITTEE ON TUMOR TISSUE REGISTRY: John W. Cline, San Francisco (Chmn.); Lewis W. Guiss, Los Angeles; James E. Kahler, Los Angeles; Carl M. McCandless, San Francisco; Weldon K. Bullock, Los Angeles.

SUBCOMMITTEE ON CONSULTATIVE TUMOR BOARDS: Bert L. Halter, San Francisco; Carl L. Moore, Bakersfield; Victor Richards, San Francisco; John G. Walsh, Sacramento (Chmn.).

Committee on Adverse Drug Reactions

Edward R. Bloomquist, Glendale	1967
Ralph O. Wallerstein, San Francisco	1967
John F. Murray, Fresno (Chmn.)	1968
Russell J. Tat, San Francisco	1969
William W. Thayer, Stockton	1969

CONSULTANT: John W. Brown, Berkeley; Ralph W. Weilerstein, Berkeley.

SUBCOMMITTEE ON DANGEROUS DRUGS: Edward Bloomquist, Glendale; Norman A. Gale, San Diego; Edward T. Jewett, Santa Rosa; William F. Quinn, Los Angeles (Chmn.); Helen B. Weyrauch, San Francisco.
CONSULTANT: Ralph W. Weilerstein, Berkeley.

Committee on Cardiovascular Diseases

Harold I. Griffeath, San Francisco (Chmn.); Lewis T. Bullock, Los Angeles; David J. New, Modesto; George C. Griffith, Los Angeles.
CONSULTANT: John E. Connolly, Los Angeles.

* * *

BUREAU OF *Research and Planning*

J. Brandon Bassett, Oakland
John Sheehy, Riverside
Samuel Sherman, San Francisco (Chmn.)
H. Russell Fisher, Glendale
G. Roger Ellison, Pomona
EX-OFFICIO: Malcolm S. M. Watts, San Francisco; Ralph C. Teall, Sacramento.
CONSULTANT: Gerald W. Shaw, Santa Monica.

* * *

COMMISSION ON *Professional Welfare*

Bert L. Halter, San Francisco
George K. Herzog, Jr., San Francisco (Chmn.)
Francis E. West, San Diego
James H. Yant, Sacramento (Secty.)

Committee on Physicians Group Insurance

Robert J. Wescott, El Centro	1967
Robert X. DeMangus, Glendale	1967
John B. Long, Sacramento	1967
George K. Herzog, Jr., San Francisco (Chmn.)	1968
John Hogan, San Diego	1969
Homer C. Pheasant, Los Angeles	1969

Liaison Committee to Medical Schools

Samuel R. Irvine, Beverly Hills	1967
Francis E. West, San Diego	1967
Frank J. Novak, Menlo Park	1968
Stanley Cowell, Los Angeles	1968
Bert L. Halter, San Francisco (Chmn.)	1969
Alonzo Neufeld, Los Angeles	1969
Herbert Jenkins, Sacramento	1969

EX-OFFICIO: James C. MacLaggan, San Diego; John G. Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Medical Review and Advisory Committee

Eugene M. Webb, San Francisco	1967
James H. Yant, Sacramento (Chmn.)	1967
John B. Dillon, Los Angeles	1968
Carl Goetsch, Berkeley	1968
Donald Donisthorpe, Los Angeles	1969

CONSULTANT: Don Harper Mills, Long Beach.

Liaison Committee to State Bar of California

David Rubsamen, Berkeley	1967
John V. Fiore, Westchester	1967
Francis E. West, San Diego (Cbmn.)	1968
Carl Goetsch, Berkeley	1968
Robert J. McNeil, Los Angeles	1969
Henry A. Brown, San Mateo	1969

* * *

COMMISSION ON *Allied Health Professions and Services*

Roger W. Barnes, Los Angeles (Secty.)
Forest J. Grunigen, Los Angeles (Chmn.)
Frank C. Melone, Ontario
Thomas W. Ledwich, Napa
C. Gerald Scarborough, San Jose
Richard H. Schug, Long Beach

Committee on Paramedical Personnel

Frank C. Melone, Ontario (Cbmn.)	1967
Gregory Bard, San Francisco	1968
Elmer R. Jennings, Long Beach	1968
Gerson R. Biskind, San Francisco	1969
Joseph Schaefer, Santa Rosa	1969
Leonard M. Asher, Los Angeles	1969
Richard H. Schug, Long Beach (Chmn.)	1969

Committee on Other Professions

George S. Buehler, Whittier	1967
Burt L. Davis, Palo Alto	1967
C. Gerald Scarborough, San Jose (Chmn.)	1968
William D. Evans, Los Angeles	1968
J. Gordon Epperson, Oakland	1969
Lewis T. Bullock, Los Angeles	1969

Liaison Committee to California Medical Assistants Association

Leon O. Burke, Sacramento	1967
John C. Brennan, Pico Rivera	1967
H. Russell Irwin, San Diego	1967
Peary Benn Berger, Inglewood	1968
Judith D. Smith, Vallejo	1969
Sanford E. Feldman, San Francisco	1969
Richard H. Schug, Long Beach (Chmn.)	1969

CONSULTANTS: Leland W. Bonar, Redding; Henry Bodner, Van Nuys.

Committee on Medicine and Religion

Albert E. Long, Daly City	1967
Roger W. Barnes, Los Angeles (Chmn.)	1967
Max Nelson, National City	1968
Frank J. Glassy, Sacramento	1969
Richard J. Lescoe, Torrance	1969

Committee on Voluntary Health Agencies

H. Mason Hohl, Beverly Hills	1967
Thomas W. Ledwich, Napa (Chmn.)	1968
Harvey May Cordua, Jr., San Diego	1969
J. Brandon Bassett, Oakland	1969

* * *

Judicial COMMISSION

Donald A. Charnock, Los Angeles	1967
Albert E. Long, Daly City	1967
Donald Cass, Hollywood	1967
Floyd O. Due, Oakland	1968
Sidney J. Shipman, San Francisco	1968
Francis E. West, San Diego (Chmn.)	1968
Douglas G. Campbell, San Francisco	1969
W. Philip Corr, Riverside (Secty.)	1969
L. E. Wilson, Anaheim	1969

* * *

COMMISSION ON *Hospital Affairs*

William L. Argo, Fresno (Secty.)
James C. Doyle, Beverly Hills
Bert L. Halter, San Francisco (Chmn.)
John C. Lungren, Long Beach
Glenn Pope, Sacramento
John T. Saidy, San Mateo
Joseph W. Telford, San Diego
Frederic N. Tyroler, Los Angeles

Medical Staff Survey Committee

Robert M. Adams, San Leandro
W. L. Argo, Fresno
Daniel W. Black, Castro Valley
Rush M. Blodget, Jr., Redding
Bert L. Halter, San Francisco (Chmn.)
J. Ralph Hughes, San Diego
Wm. Kenneth Jennings, Santa Barbara
Donald O. Lagerlof, Los Angeles
John C. Lungren, Long Beach
William A. Newsom, San Francisco
James L. Rhee, Oakland
John T. Saidy, San Mateo
Wilfred J. Snodgrass, Santa Monica
Alexander Hatoff, Oakland
Shepard F. Palitz, Bakersfield
William L. Sumption, Santa Monica
A. Jerome Hurter, Pasadena
Ernest R. Barnett, Alhambra
Richard L. Taw, Los Angeles
Richard H. McDonald, Garden Grove
Robert M. Zweig, Riverside
L. Morgan Boyers, Sacramento
James O. Farley, Sacramento
Joseph W. Telford, San Diego
Paul Deeb, Redlands
Maxwell Goldstein, Fontana
Ray Seavers, Jr., San Bernardino
William Soldmann, La Mesa
Lloyd Milburn, San Francisco
Frank Solomon, San Francisco
Sanford Feldman, San Francisco
John Bazzano, San Francisco
Kenneth Allen, San Mateo
Martin Kohn, San Bruno
William Stephenson, Santa Barbara
Merlin Hendrickson, Rialto
Andrew D. Lucine, Campbell
Fred Haven Draper, San Jose
Wayne O. Buck, San Jose
William A. Reeves, Mt. View
A. A. Thurlow, Jr., Santa Rosa
John A. Carollo, Sabastopol
William Rector, Sacramento
Gerald W. Shaw, Santa Monica
Charles L. Lear, Norwalk
John R. Heckman, Marysville
Robert Buckley, Santa Monica
Woodrow W. Burgess, Sacramento
Ira H. Degenhardt, San Rafael
Arthur E. Gladman, Oakland
Norman Graff, San Mateo
Frank V. Hoffman, Huntington Beach
Robert E. James, Belmont
Clement O. Juul, Oakland
Seymour Kolko, San Mateo
Melvin M. Lipsett, Berkeley
Jack B. Lomas, Los Angeles
Edward A. Macklin, San Francisco
D. Stuart MacRobbie, Berkeley
Felix H. Ocko, Berkeley
William R. Sheehy, Berkeley
John A. Stroud, Sacramento
R. J. Sutherland, Oakland
Rudolph B. Toller, Stockton
Albert T. Voris, Belmont
G. Cameron Cray, Palo Alto

(Continued on Page 51)

Officers of Scientific Sessions

Members who contemplate presentation of scientific papers should promptly address the secretary of the proper section at the address shown below.

Allergy

Leo N. Meleyco.......................Chairman
2040 Forest Avenue, San Jose 95128
Ernest J. Saslow.......................Secretary
2011-18th Street, Bakersfield 93301
Samuel Ross ....Assistant Secretary
2946 Fresno Street, Fresno 93706

Anesthesiology

Thomas W. McIntosh.......................Chairman
686 East Union St., Pasadena 91101
Donald B. Dose.......................Secretary
5625 Linfield Ave., San Diego 92120
Donald A. Walker.......................Assistant Secretary
35 Spring Lane, Tiburon 94920

Dermatology

Robert G. Walton ....Chairman
1700 McHenry Village Way, Modesto 95350
Samuel Ayres, III.......................Secretary
405 N. Bedford Rd., Beverly Hills 90210
George T. Wilson.......................Assistant Secretary
Welch and Pasteur Rd., Stanford Medical Plaza
Palo Alto 94304

General Practice

J Blair Pace.......................Chairman
408 Cassidy St., Oceanside 92054
Alfred F. Kandlbinder.......................Secretary
302 Via Paraiso St., Monterey 93940
Allen F. Sterling.......................Assistant Secretary
1285 D St., San Bernardino 92045

General Surgery

Seibert Pearson ....Chairman
2865 Atlantic Ave., Long Beach 90807
Albert G. Clark.......................Secretary
450 Sutter St., San Francisco 94108
Robert T. Hood, Jr.......................Assistant Secretary
2010 Wilshire Blvd., Los Angeles 90057

Industrial Medicine and Surgery

Leon R. Rudnick.......................Chairman
15921 E. 14th St., San Leandro 94578
Raymond O. Oja ....Secretary
525 Golden Gate Ave., San Francisco 94102
James G. Forgerson.......................Assistant Secretary
Metropolitan Life Insurance Company
600 Stockton St., San Francisco 94108

Internal Medicine

Robert L. Paver.......................Chairman
384 Post St., San Francisco 94108
John L. Benton, Jr.......................Secretary
1216 Wilshire Blvd., Los Angeles 90017
Edgar Rosen ....Assistant Secretary
1340 K St., Sacramento 95814

Obstetrics and Gynecology

Lester T. Hibbard.......................Chairman
142 S. Windsor Blvd., Los Angeles 90004
Warren E. Jones.......................Secretary
1380-45th Street, Sacramento 95819
James W. Ravenscroft.......................Assistant Secretary
2330 First Ave., San Diego 92101

Ophthalmology

Victor G. Fellows.......................Chairman
595 Buckingham Way, San Francisco 94117
Wendell C. Irvine ....Secretary
3875 Wilshire Blvd., Los Angeles 90005
Robert E. Hopkins.......................Assistant Secretary
4255 Pacific Ave., Stockton 95207

Orthopedics

David C. Monsen.......................Chairman
2300 S. Hope St., Los Angeles 90007
Arthur R. Hartwig.......................Secretary
490 Post St., San Francisco 94102
Vernon L. Smythe.......................Assistant Secretary
3875 Wilshire Blvd., Los Angeles 90005

Otolaryngology

G. Howard Gottschalk ....Chairman
8618 S. Sepulveda Blvd., Los Angeles 90045
Charles P. Lebo ....Secretary
490 Post St., San Francisco 94102
Irving L. White.......................Assistant Secretary
911 San Antonio Dr., Long Beach 90807

Pathology

Frank R. Dutra.......................Chairman
20103 Lake Chabot Rd., Castro Valley 94546
William H. Kern.......................Secretary
679 S. Westlake Ave., Los Angeles 90057
Donald L. Bittner.......................Assistant Secretary
Bay Area Laboratories, 2245 Post St.,
San Francisco 94115

Pediatrics

Richard L. Anderson.......................Chairman
636 Harris St., Eureka 95501
Sherrod C. Swift.......................Secretary
14619 Whittier Blvd., Whittier 90605
Robert J. Harvey.......................Assistant Secretary
909 Hyde St., San Francisco 94109

Physical Medicine

Rene Cailliet ....Chairman
4900 Sunset Blvd., Los Angeles 90027
Anton A. Tratar.......................Secretary
2360 Clay St., San Francisco 94115
Elizabeth Austin ....Assistant Secretary
Los Angeles County Hospital
1200 N. State St., Los Angeles 90033

Preventive Medicine

Henrik L. Blum.......................Chairman
School of Public Health, U.C., Room 409, Earl Warren Hall, Berkeley 94720
Maxwell Rosenblatt ....Secretary
Los Angeles County Health Department,
220 N. Broadway, Los Angeles 90012
Russell B. Watson.......................Assistant Secretary
Orange County Health Department, Santa Ana 92701

Psychiatry and Neurology

Werner M. Mendel.......................Chairman
1934 Hospital Place, Los Angeles 90033
Ames Fischer ....Secretary
Langley Porter Inst., 401 Parnassus Ave.,
San Francisco 94122
Howard S. Barrows.......................Assistant Secretary
2025 Zonal Ave., Los Angeles 90033

Radiology

Victor G. Mikity.......................Chairman
1200 State St., Los Angeles 90033
Vernon R. Gee.......................Secretary
2020 Court St., Redding 96001
Richard P. Storrs.......................Assistant Secretary
2131 West 3rd St., Los Angeles 90057

Urology

Carl K. Pearlman.......................Chairman
1518 N. Sycamore, Santa Ana 92701
James L. Goebel.......................Secretary
51 Loma Linda, Ross 94957
Robert O. Pearman.......................Assistant Secretary
5353 Balboa Blvd., Encino 91316

Delegates and Alternates

TO THE AMERICAN MEDICAL ASSOCIATION

DELEGATES		ALTERNATES
Francis E. West, San Diego	(1966-1967)	Vincent P. Carroll,* Laguna Beach
Samuel R. Sherman, San Francisco	(1966-1967)	Albert G. Miller, San Mateo
Henry Gibbons, III, San Francisco	(1966-1967)	A. B. Sirbu, San Francisco
John M. Rumsey, San Diego	(1966-1967)	Carl E. Anderson, Santa Rosa
Eugene F. Hoffman, Los Angeles	(1966-1967)	Joseph F. Boyle, Los Angeles
Warren L. Bostick, Los Angeles	(1966-1967)	Walter H. Brignoli, St. Helena
J. B. Price, Santa Ana	(1966-1967)	Donald C. Dodds, Oakland
Ralph C. Teall, Sacramento	(1966-1967)	Robb Smith, Orange Cove
James C. Doyle, Beverly Hills	(1966-1967)	Herman H. Stone, Riverside
Wilbur G. Rogers, Glendale	(1966-1967)	Carl M. Hadley, San Bernardino
Charles B. Hudson, Oakland	(1966-1967)	Robert L. Watson, Jr., Los Angeles
John G. Morrison, San Leandro	(1967-1968)	Frederick Ackerman, Pleasant Hills
William F. Quinn, Los Angeles	(1967-1968)	Dudley M. Cobb, Los Angeles
Robert C. Combs, San Francisco	(1967-1968)	Emmett Rixford, San Francisco
Homer C. Pheasant, Los Angeles	(1967-1968)	James C. MacLaggan, San Diego
Alfred J. Murrieta, Los Angeles	(1967-1968)	Harold Kay, Oakland
Burt L. Davis, Palo Alto	(1967-1968)	Leon P. Fox, San Jose
Arlo A. Morrison, Ventura	(1967-1968)	Henry Brown, San Mateo
James E. Feldmayer, Exeter	(1967-1968)	Charles Grayson, Sacramento
O. W. Wheeler, Riverside	(1967-1968)	Wilbur Bailey, Los Angeles
Malcolm C. Todd, Long Beach	(1967-1968)	Neil C. Hamel, Encino
J. E. Vaughan, Bakersfield	(1967-1968)	Joseph P. O'Connor, Pasadena

(Delegates and Alternates to the AMA are elected for terms of two calendar years. Each office is scheduled for election at the Annual Session of the second year shown above.) *•Council appointee.*

Other Organizations and Medical Schools

Board of Medical Examiners of the State of California

Secretary.......................Shelby M. Hicks, M.D.
1021 O Street, Room A202, Sacramento 95814
San Francisco.......................30 Van Ness Ave., 94102
Room 2100
Los Angeles.......................107 South Broadway 90012
Sacramento.......................1021 O Street, Room A547 95814

The Public Health League of California

Executive Secretary.......................Ben H. Read
955 South Western Ave., Los Angeles 90006
Assistant Executive Secretary.......................Al Paul Putnam
693 Sutter Street, San Francisco 94102
San Francisco.......................693 Sutter Street 94102
415 474-1061
Los Angeles.......................955 South Western Ave. 90006
213 731-6397
Sacramento.......................1127-11th Street, 95814
916 444-7496

Department of Public Health of the State of California

Director.......................Lester Breslow, M.D.
2151 Berkeley Way, Berkeley 94704
Berkeley.......................2151 Berkeley Way 94704
415 843-7900
Sacramento.......................1927-13th Street 95814
916 445-2264
Los Angeles.......................417 South Hill Street 90013
213 620-2900

Medical Schools in California

University of California School of Medicine, Medical Center, San Francisco 94122. Dean: Stuart C. Cullen, M.D.
Stanford University School of Medicine, 300 Pasteur Drive, Palo Alto 94304. Dean: Robert J. Glaser, M.D.
University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 90033. Dean: Roger O. Egeberg, M.D.
Loma Linda University School of Medicine, Loma Linda 92354. Dean: David Burdg Hinshaw, M.D.
University of California at Los Angeles, School of Medicine, Hilgard Avenue, Los Angeles 90024. Dean: Sherman M. Mellinkoff, M.D.
University of California, California College of Medicine, 1721 Griffin Avenue, Los Angeles 90031. Dean: Warren L. Bostick, M.D.
University of California, San Diego, School of Medicine, La Jolla 92038. Acting Dean: Robert D. Tschirgi, M.D.
University of California, Davis, School of Medicine, Davis 95616. Dean: Charles John Tupper, M.D.

California Medical Education and Research Foundation

Directors: Ralph C. Teall, M.D., President; Carl E. Anderson, M.D., Vice-President; Howard Hassard, Secretary; James C. MacLaggan, M.D.; Harold Kay, M.D.; William F. Quinn, M.D.; E. B. Shaw, M.D.

Roster — CALIFORNIA COMPONENT MEDICAL SOCIETIES

Society secretaries are requested to notify California Medicine promptly when changes are indicated in their roster information

ALAMEDA-CONTRA COSTA Medical Assn., 6230 Claremont Ave., Oakland 94618. Meets quarterly.
Frederick W. Ackerman..........President
2100 Monument Blvd., Pleasant Hill 93542
Herbert A. Holden..........Secretary
1436 Clarke St., San Leandro 94577

BUTTE-GLENN Medical Society, P. O. Box 1008, Chico 95927. Meets Fourth Thursday.
Walter S. Mazen..........President
1617 Esplanade, Chico 95926
John B. Copeland..........Secretary
1423 Magnolia, Chico 95926

FORTY FIRST Medical Society, Suite 821, 1515 N. Vermont Ave., Los Angeles 90027
Paul D. Yates..........President
844 Hermosa Ave., Hermosa Beach 90254
Jordan M. Phillips..........Secretary
11239 S. Lakewood Blvd., Downey 90241

FRESNO County Medical Society, 1118 Divisadero St., Fresno 93721. Meets Second Tuesday, 6:30 p.m., California Hotel.
Theodore Steinberg..........President
155 N. Thesta St., Fresno 93701
Jack Schiff..........Secretary
167 N. Thesta St., Fresno 93701

HUMBOLDT-DEL NORTE County Medical Society. Meets Second Thursday.
Halvor J. Braafladt..........President
3115 G St., Eureka 95501
Max Todd..........Secretary
616-14th St., Arcata 95521

IMPERIAL County Medical Society, P. O. Box 518, Imperial 92251. Meets Second Tuesday, 8 p.m., Pioneer Memorial Hospital, Brawley.
O. G. Fais..........President
1745 S. Imperial Ave., El Centro 92243
Elmer C. Werner..........Secretary
1745 S. Imperial Ave., El Centro 92243

INYO-MONO County Medical Society. Meets Fourth Tuesday except December, January, February.
Milton R. Jones, Jr..........President
Box 38, Lone Pine 93545
Robert W Denton..........Secretary
611 W. Line St., Bishop 93514

KERN County Medical Society, 2603 G Street, Bakersfield 93301. Meets Third Tuesday, 7:30 p.m., Society Office, 2603 G Street, except June, July, August.
Keith W. Spaulding..........President
2521 "G" St., Bakersfield 93301
James R. Friend..........Secretary
P. O. Box 2087, Bakersfield 93303

KINGS County Medical Society, P. O. Box 1003, Hanford 93230. Meets Second Thursday, 7:00 p.m. Place to be announced.
James Dean..........President
912 N. Douty St., Hanford 93230
Willard S. Bridwell..........Secretary
1507 N. 11th Ave., Hanford 93230

LASSEN-PLUMAS-MODOC-SIERRA County Medical Society Meets on call
James M. McCullough..........President
50 N. McDow St., Susanville 96130
Charles W. Brown..........Secretary
Western Pacific Hospital, Portola 96122

LOS ANGELES County Medical Assn., 1925 Wilshire Blvd., Los Angeles 90057.
Joseph F. Boyle..........President
1136 W. Sixth St., Los Angeles 90017
Homer C. Pheasant..........Secretary
2300 S. Hope St., Suite 300,
Los Angeles 90007

MARIN Medical Society, 1601 Second St., Suite 106, San Rafael 94901. Meets First Thursday, 7:00 p.m.
Robert Mills..........President
711 D St., San Rafael 94901
William A. Galeno..........Secretary
712 D St., San Rafael 94901

MENDOCINO-LAKE County Medical Society.
Frank H. Dailey..........President
719 S. Dora, Ukiah 95482
Patrick R. Allanson..........Secretary
765 S. Dora St., Ukiah 95482

MERCED-MARIPOSA Medical Society, P. O. Box 586, Merced 95340. Meets Fourth Thursday; meeting place changes.
Patrick J. Maloney..........President
P. O. Box 586, Merced 95340
Robert J. Schiffler..........Secretary
P. O. Box 586, Merced 95340

MONTEREY County Medical Society, P. O. Box 308, Salinas 93903. Meets First Thursday.
Joseph E. Turner..........President
1010 Cass St., Monterey 93940
Thomas S. Elliott..........Secretary
101 Maple St., Greenfield 93927

NAPA County Medical Society, P. O. Box 2158, Napa 94559. Meets Second Wednesday.
Richard W. Neil..........President
1375 Main St., St. Helena 94574
Victor J. Bouillon..........Secretary
3020 Beard Rd., Napa 94558

ORANGE County Medical Association, 300 S. Flower, Orange 92666. Meets First Tuesday, 7:00 p.m.
Wallace A. Gerrie..........President
1901 Westcliff Dr., Newport Beach 92660
David I. Nielsen..........Secretary
400 Westminster Blvd., Newport Beach 92660

PLACER-NEVADA County Medical Society. Meets Second Wednesday.
F. Harold Johnson..........President
305 Oak Ridge Dr., Roseville 95678
Arthur R. Weaver..........Secretary
1212 High St., Suite 106, Auburn 95603

RIVERSIDE County Medical Association, 4175 Brockton Ave., Riverside 92501. Meets Second Monday, 7:30 p.m., Spanish Arts Gallery, Mission Inn.
Roger W. Ridley..........President
6969 Brockton Ave., Riverside 92506
Richard W. Trotter..........Secretary
6876 Magnolia Ave., Riverside 92506

SACRAMENTO County Medical Society, 5380 Elvas Ave., Sacramento 95819. Meets Third Tuesday, 8:30 p.m. Location of meetings varies.
James W. Martin..........President
2811 L St., Sacramento 95816
James O. Farley..........Secretary
3428 Watt Ave., Sacramento 95821

SAN BENITO County Medical Society. Meets once a month, except July and August. Time and place to be announced.
Robert D. Quinn..........President
930 Sunset Dr., Hollister 95023
Roderick L. Yip..........Secretary
891 Sunset Dr., Hollister 95023

SAN BERNARDINO MEDICAL SOCIETY, 369 Court St., San Bernardino 92401. Meets First Tuesday, 7:30 p.m., Harold's Club, Fontana.
Richard W. Judd..........President
1700 Waterman Ave., San Bernardino 92404
Daniel J. Rothenberger..........Secretary
321 Cajon St., Redlands 92373

SAN DIEGO County Medical Society, 3427-4th Ave., San Diego 92103. Meets Second Tuesday. Location of meetings varies.
John Wanless..........President
3427 Fourth Ave., San Diego 92103
W. C. Herrick..........Secretary
233 A St., San Diego 92101

SAN FRANCISCO Medical Society, 250 Masonic Ave., San Francisco 94118.
Albert G. Clark..........President
450 Sutter St., San Francisco 94108
Harry W. Sawyer..........Secretary
909 Hyde St., San Francisco 94109

SAN JOAQUIN County Medical Society, 445 W. Acacia St., P. O. Box 230, Stockton 95201. Meets First Thursday, 8:15 p.m.
Thomas Huff..........President
512 W. Harding Way, Stockton 95204
Dora A. Lee..........Secretary
2420 N. California St., Stockton 95204

SAN LUIS OBISPO County Medical Society, P.O. Box 319, San Luis Obispo 93402. Meets Third Saturday, 7:00 p.m., Madonna Inn, San Luis Obispo 93402.
Robert Buck..........President
P. O. Box 319, San Luis Obispo 93402
Billy W. Mounts..........Secretary
P. O. Box 319, San Luis Obispo 93402

SAN MATEO County Medical Society, 122 El Camino Real, San Mateo 94401. Meets Third Tuesday.
Stanford B. Rossiter..........President
Sequoia Hospital, Redwood City 94602
Eldon E. Ellis..........Secretary
2900 Whipple Ave., Redwood City 94602

SANTA BARBARA County Medical Society, 300 W. Pueblo St., Santa Barbara 93105. Meets Second Monday of the month, University Club, 1332 Santa Barbara St., Santa Barbara.
T. K. Hill..........President
1421 State St., Santa Barbara 93105
Casimir Domz..........Secretary
317 W. Pueblo St., Santa Barbara 93105

SANTA CLARA County Medical Society, 700 Empey Way, San Jose 95128. Meets First Monday.
Gerald Besson..........President
700 Empey Way, San Jose 95128
Alexander MacKenzie..........Secretary
700 Empey Way, San Jose 95128

SANTA CRUZ County Medical Society. Meets every Second Month, Second Tuesday. Time, place to be announced.
W. W. Hoskins..........President
30 Brennan St., Watsonville 95076
William Blade..........Secretary
1510 Seabright Ave., Santa Cruz 95062

SHASTA-TRINITY County Medical Society. Meets Second Monday of the month. Place to be announced.
Joseph T. DeWitte..........President
1129 Hartnell Ave., Redding 96001
Eugene A. Nutley..........Secretary
2625 Edith Ave., Redding 96001

SISKIYOU County Medical Society. Meets Sunday on call.
Robert L. Anderson..........President
504 Hillcrest Dr., Yreka 96097
John P. Geyman..........Secretary
705 S. A St., Mt. Shasta 96067

SOLANO County Medical Society, 631 Tennessee St. Vallejo 94599.
Tuesday. 8:00 p.m., at different meeting places.
Robert L. Garrett..........President
1512 Napa St., Vallejo 94590
Keith E. Vincent..........Secretary
2121 Redwood St., Vallejo 94594

SONOMA County Medical Society, 1625 Franklin Ave., Santa Rosa 95404. Meets Second Thursday.
John S. Dunn..........President
576 B St., Santa Rosa 95401
Elizabeth Buckley..........Secretary
1140 Sonoma Ave., Santa Rosa 95405

STANISLAUS County Medical Society, 1125-13th St., Modesto 95354. Meets Third Tuesday of the month, 7 p.m., Hotel Covell, Modesto.
Howard Kuhl..........President
1700 McHenry Ave., Modesto 95350
Jeanne I. Miller..........Secretary
709-18th St., Modesto 95354

TEHAMA County Medical Society. Meets at call of President.
W. Scott Davis..........President
P. O. Box 118, Los Molinos 96055
M. Blair McGee..........Secretary
217 Corona Ave., Corning 96021

TULARE County Medical Society, 1640 West Mineral King. P. O. Box 16, Visalia 93278.
Irving M. Schor..........President
P. O. Box 1151, Visalia 93277
George Tiss..........Secretary
P. O. Box 7, Visalia 93277

VENTURA County Medical Society, 3291 Loma Vista Rd., Room 40, Ventura 93003. Meets Second Tuesday each month, 7:30 p.m., Los Posas Country Club, Camarillo.
Joseph F. Gstettenbauer..........President
3003 Loma Vista Rd., Ventura 93003
Franklin K. Helbling..........Secretary
3291 Loma Vista Rd., Room 40, Ventura 93003

YOLO County Medical Society. Meets First Wednesday.
Benjamin R. Robinson..........President
650 Third St., Woodland 95695
Corbin Young..........Secretary
101 Main St., Winters 95694

YUBA-SUTTER-COLUSA County Medical Society.
John R. Heckman..........President
714-4th St., Marysville 95901
Norman K. Noordhoff..........Secretary
320 G St., Marysville 95901

Officers — CALIFORNIA PHYSICIANS' SERVICE — 1966-67
BLUE SHIELD

Officers:

Chairman of the Board	Richard S. Wilbur
Vice-Chairman of the Board	Milo A. Youel
Secretary	Wilbur G. Rogers
Treasurer	Bert L. Halter

BOARD OF *Trustees:*

Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Roberta F. Fenlon*	San Francisco
Donald R. Fitch	Glendale
Mr. Thomas Hadfield	San Francisco
Bert L. Halter	San Francisco
Donald C. Harrington	Stockton
M. M. Haskell	Long Beach
Carl Goetsch	Berkeley
Mr. Philip S. Magruder	Flintridge
Gregory C. Murray	Los Angeles
Wilbur G. Rogers*	Glendale
Mr. Paul Schrade	Los Angeles
William H. Thompson	San Mateo
Richard S. Wilbur*	Palo Alto
Milo A. Youel	San Diego

---

*Appointed by CMA Council

BOARD COMMITTEES:

Executive Committee

Richard S. Wilbur, Chairman	Palo Alto
Carl Goetsch	Berkeley
Bert L. Halter	San Francisco
Mr. Philip S. Magruder	Flintridge
Wilbur G. Rogers	Glendale
William H. Thompson	San Mateo
Milo A. Youel	San Diego

Finance Committee

Bert L. Halter, Chairman	San Francisco
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Donald R. Fitch	Glendale
Mr. Philip S. Magruder	Flintridge

Medical Policy Committee

Wilbur G. Rogers, Chairman	Glendale
Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Roberta F. Fenlon	San Francisco
Carl Goetsch	Berkeley
Mr. Thomas Hadfield	San Francisco
M. M. Haskell	Long Beach
Donald C. Harrington	Stockton
Gregory C. Murray	Los Angeles

When the problem is only skin deep

USE 'POLYSPORIN'® brand POLYMYXIN B-BACITRACIN OINTMENT

for topical antibiotic therapy with minimum risk of sensitization

Caution: As with other antibiotic products, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

Supplied in ½ oz. and 1 oz. tubes.

Complete literature available on request from Professional Services Dept. PML.

BURROUGHS WELLCOME & CO. (U.S.A.) INC.
Tuckahoe, N.Y.

'POLYSPORIN'
POLYMYXIN B-BACITRACIN
OINTMENT
help prevent infection in
burns, and abrasions;
aid in healing.

JAMA Evaluates Contraceptive Methods

"The population explosion and the frightening magnitude and complexity of the problems it is creating continue to be of worldwide concern," says an editorial in the February 27 *Journal of the American Medical Association.*

Within little more than a decade, the editorial notes, two highly effective methods of conception control have been developed: Oral contraceptives, and more recently, intrauterine contraceptive devices (IUDs).

Both types of conception controls are evaluated in this issue of the *Journal* in statements by the AMA Committee on Human Reproduction and the AMA Council on Drugs.

"Because of their convenience and proven effectiveness, the oral contraceptives have been widely accepted by both physicians and patients, and it is estimated that approximately seven million women (five million in the United States) are now using them," the editorial said. "'However, it has also been recognized that much information is lacking about the actions of these preparations."

Study groups operating under auspices of the World Health Organization and the U.S. Food and Drug Administration have published reports that present a balanced opinion on oral contraceptives, the editorial said.

Opinions differ on the oral contraceptives' relationship to cancer, liver function changes, and circulatory disease, the editorial notes.

"These sharp differences of opinion should be welcomed, since they help to stimulate further research directed toward obtaining answers to these problems," the editorial said. "In the meantime, an uneasy responsibility rests with the practicing physician who must properly guide his patients in the use of these preparations without engendering undue alarm, and yet be alert to the possibility that some unusual response may signal a serious adverse reaction.

In contrast to the oral contraceptives which prevent pregnancy by a complex hormonal action, the action of the IUDs seems to be a simple mechanical phenomenon (although much information is lacking about how IUDs actually work). Side effects of the IUDs are generally due to the problems posed by placing a foreign body in the uterus.

"The IUDs and the oral contraceptives represent another of man's successful efforts toward intelligent control of his environment," the editorial said. "Undoubtedly these methods presage the development of even better methods of fertility control.

"In the meantime, to extend their usefulness, convenience, and safety, more clinical investigations . . . are needed to resolve the major problems that exist. Any delay should be avoided, and the physician should be repeatedly urged to report the nature of all adverse reactions he might see."

One by one the family's downed Because the G.I. bug's around

Parepectolin for quick relief of acute diarrhea
...soothes colicky pain with paregoric*
...consolidates fluid stools with pectin
...adsorbs irritants with kaolin,
and protects intestinal mucosa

Whether it's a 24-hour "bug", a food problem, or simply nervousness and anxiety, Parepectolin will bring the diarrhea under control until etiology can be determined. In some cases, Parepectolin may be all the therapy necessary.

Parepectolin®

Each fluid ounce of creamy white suspension contains:
*Paregoric (equivalent) .... (1.0 dram) 3.7 ml.
Contains opium (¼ grain) 15 mg. per fluid ounce.
warning: may be habit forming
Pectin .... (2½ grains) 162 mg.
Kaolin (specially purified) .... (85 grains) 5.5 Gm.
(alcohol 0.69%)
Usual Adult Dose: One or two tablespoonfuls three times daily.
Usual Children's Dose: One or two teaspoonfuls three times daily.

RORER
WILLIAM H. RORER, INC.
Fort Washington, Pa.

For adolescents, acne is "...an affliction which immediately separates these people from their confreres."* Washing with pHisoHex, from the first sign of acne, can help your patients—even with severe forms—through the "acne decade" (12-22). Three or four thorough daily washes with powerful, antibacterial pHisoHex enhance any acne regimen. Faster, better results help restore self-confidence.

pHisoHex builds a cumulative bacteriostatic film of hexachlorophene to protect acne skin between washings, helps check the "infection factor." pHisoHex is especially effective against Staph—a frequent invader of acne lesions.

A much more surface-active detergent than soap, pHisoHex removes dirt, emulsifies oil and cleanses acne skin rapidly and thoroughly, including orifices of the sebaceous glands, sweat glands and hair follicles. pHisoHex is nonalkaline, hypoallergenic and "kind" to skin.

pHisoAc®, a keratolytic skin-toned cream, is recommended with pHisoHex for local treatment in acne. pHisoAc dries, peels and masks lesions, helps prevent comedones, pustules and scarring. It contains colloidal sulfur 6 per cent, resorcinol 1.5 per cent and hexachlorophene 0.3 per cent. Odorless, nongreasy and pleasant to use.

pHisoHex is supplied in squeeze bottles of 5 oz. and 1 pint and in unbreakable bottles of 1 gal.

pHisoAc is supplied in tubes of 1½ oz., also available in a combination package with a 5-oz. squeeze bottle of pHisoHex.

*Kligman, A. M., in Ludwig, G. D., and Elsom, Katherine O. (Eds.): *Am. Pract.* 13:200, March, 1962.

pHisoHex and pHisoAc, trademarks reg. U.S. Pat. Off.

Winthrop Laboratories, New York, N.Y. 10016

In acne
pHisoHex®
antibacterial skin detergent with 3% hexachlorophene
is always "right"
Winthrop

For the tense patient 1 tab.
t.i.d.

who can't sleep
h.s.
remember the
extra tablet at bedtime

Valium®

(diazepam)

Roche®

Before prescribing, please consult complete product information, a summary of which follows:

Contraindications: Infants, patients with history of convulsive disorders or glaucoma.

Warning: Not of value in the treatment of psychotic patients, and should not be employed in lieu of appropriate treatment.

Precautions: Limit dosage to smallest effective amount n elderly patients (not more than 1 mg, one or two imes daily) to preclude ataxia or oversedation. Advise patients against possibly hazardous procedures until correct maintenance dosage is established; driving during therapy not recommended. In general, concur- ent use with other psychotropic agents is not recommended. Warn patients of possible combined effects with alcohol. Safe use in pregnancy not established. Observe usual precautions in impaired renal or hepa- ic function and in patients who may be suicidal; periodic blood counts and liver function tests advisable in long-term use. Cease therapy gradually.

Side Effects: Side effects (usually dose-related) are fatigue, drowsiness and ataxia. Also reported: mild nausea, dizziness, blurred vision, diplopia, headache, incontinence, slurred speech, tremor and skin rash; paradoxical reactions (excitement, depression, stimulation, sleep disturbances, hallucinations); changes in EEG patterns. Abrupt cessation after prolonged overdosage may produce withdrawal symptoms similar to those seen with barbiturates, meprobamate and chlordiazepoxide HCl.

Dosage—Adults: Mild to moderate psychoneurotic reactions, 2 to 5 mg b.i.d. or t.i.d.; severe psychoneurotic reactions, 5 to 10 mg t.i.d. or q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; muscle spasm with cerebral palsy or athetosis, 2 to 10 mg t.i.d. or q.i.d. Geriatric patients: 1 or 2 mg/day initially, increase gradually as needed.

Supplied: Tablets, 2 mg, 5 mg and 10 mg; bottles of 50 for convenience and economy in prescribing.

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, N.J. 07110

Institute for Biomedical Research Releases New Booklet

"To House the Dreams of Those Who Delve into Life Itself" is the title of a new, 16-page pamphlet describing the role of the AMA-ERF Institute for Biomedical Research. It focuses upon fields of interest and gives thumbnail sketches of staff members. Copies may be obtained by writing the Institute for Biomedical Research, American Medical Association, 535 North Dearborn Street, Chicago, Illinois 60610.

Health and Safety Tips from The American Medical Association

"Safe at home" means scoring a run and possibly winning the game to a baseball player.

The same phrase means something entirely different to the American family.

Every year we are greeted with a new set of statistics telling us that we are by no means "safe at home" in the average household. In fact, the statistics show that home can be downright dangerous.

Accidents in the home caused 28,500 deaths in 1956, according to the *Archives of Environmental Health,* a professional journal published by the American Medical Association.

Each year about 25 million home accidents cause injuries serious enough to require medical attention or to restrict the activity of the victim a day or longer.

A host of physical factors are involved in home accidents, such as electrical and power equipment, inadequate lighting, glass doors, abandoned ice boxes, medicines, and many others. But by far the major causes of home accidents are two things—falls and fires.

Falls are the second most common cause of accidental death, next to highway accidents. Falls in and around the home each year cause injuries to nearly 7 million people and 12,000 deaths.

Falls on stairs cause nearly 2,000 fatal injuries a year. Proper lighting and sturdy handrails on stairs are highly important to home safety. A good ladder to aid in reaching high places is much safer than standing on boxes or chairs. And the ladder should be firmly placed and braced at the foot. Spilled water or grease or improper waxing of floors cause falls. Loose rugs, scattered toys and trailing extension cords also are hazards. A light switch near the door of each room is a safety measure.

Most of these hazards can be eliminated or minimized by the head of the household.

Get your family together this evening and organize a tour of the house to check for possible "fall hazards." Then set about to correct these hazards as quickly as possible. Make your home as nearly accident-proof as possible.

Tandearil®
oxyphenbutazone

Therapeutic Effects: Tandearil is a nonhormonal compound which may rapidly resolve inflammation and help restore normal joint function. Its action does not affect pituitary-adrenal function or impair immune responses. Its value in osteoarthritis is especially noteworthy because this disorder responds inconsistently to steroids and is often resistant to salicylates. Further, indomethacin is limited only to osteoarthritis of the hip, whereas oxyphenbutazone is effective in all forms of the disease.

Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently.

Warning: If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with great caution in the first trimester of pregnancy.

Precautions: Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patient should be closely supervised and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools or other evidence of intestinal hemorrhage. Make regular blood counts. Discontinue the drug and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.

Adverse Reactions: The most common are nausea, edema and drug rash. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. Infrequently, agranulocytosis, or a generalized allergic reaction may occur and require withdrawal of medication. Stomatitis, salivary gland enlargement, vomiting, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported but cannot definitely be attributed to the drug. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hyperglycemia, hepatitis, jaundice, and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently. Moderate lowering of the red cell count due to hemodilution may occur.

Dosage in Osteoarthritis: The initial daily dosage in adults is 300-600 mg. in divided daily doses. When improvement occurs, dosage should be decreased to the minimum effective level; this should not exceed 400 mg. daily, and is often achieved with only 100-200 mg. daily.

For complete details, please refer to full prescribing information. 6562-VI(B)R

Availability: Tablets of 100 mg.

Geigy
Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York

250
200
150
100
50
40
30
20
DETECTO
Doctor's scale

help tip the scales for your "should-but-can't" dieters...

- ☐ controls the appetite
- ☐ provides gentle 'lift' to strengthen patients' determination to stick to dietary instructions
- ☐ simple, easy-to-remember dosage
- ☐ special timed-release mechanism ensures smooth, uniform, 10-12 hour therapeutic effect
- ☐ proved clinically safe in over 80 million doses
- ☐ the preferred HCl salt for more available amphetamine

AMODEX®

TIMED CAPSULES

AMODEX® Timed capsules

Each AMODEX TIMED CAPSULE contains:

dextro-amphetamine HC1	15 mg.
amobarbital (barbituric acid derivative)	60 mg.

WARNING: may be habit forming

DOSAGE: One capsule on arising or at breakfast. Drugs are released gradually over 6 to 8 hours, providing therapeutic effect for 10 to 12 hours.

INDICATIONS: AMODEX Timed Capsules elevate the mood, relieve nervous tension, restore emotional stability and emotional capacity for physical and mental effort. AMODEX Timed Capsules are extremely useful in the treatment of anxiety states and may be used to control appetite in the management of the obese patient — without nervous excitation.

SIDE EFFECTS AND PRECAUTIONS: Frequent or continued use may cause nervousness, sleeplessness, or restlessness. Individuals suffering from high blood pressure, heart disease, diabetes, thyroid disease, lung ailments, or kidney disorders should not take this product. It should not be taken over a long period of time.

CONTRAINDICATIONS: Hyperexcitability, agitated pre-psychotic states. Sensitivity to Amphetamines or Barbiturates.

CAUTION: Federal Law prohibits dispensing without prescription.

SUPPLIED: In bottles of 30, 100, and 1000 capsules.

Fellows Testagar
DIVISION OF FELLOWS MEDICAL MFG. CO., INC.
pharmaceuticals since 1866
Detroit, Michigan

Classsifying Youngsters For Physical Education

Physical education is so important that great care should be used in excusing youngsters from a school's program in this area.

Schools should establish comprehensive programs that provide physical education for students with varying degrees of physical fitness. It's then the task of physicians, educators, and parents to cooperate in a program that classifies students according to physical ability.

These are some of the recommendations in a statement by the American Medical Association's Committee on Exercise and Physical Fitness that appeared recently in the *Journal of the American Medical Association.*

"A good physical education program offers opportunity for the student to participate in a wide variety of activities with differing degrees of intensity. This enables the physician to guide a student with a health handicap into a program of physical education adapted to his individual needs," the statement said.

A broadly based program also enables the physician to encourage healthy students to participate fully in a program of interest and value to them.

"Each youth—healthy or handicapped—can be helped to discover activities in which he can take part with benefit to his health," the statement said.

Generally, students are placed in one of four categories for physical activity. These include:

1. Unrestricted activity — full participation in physical education and athletic activities.
2. Moderate restriction—participation in designated physical education and athletic activities.
3. Severe restriction—participation in only a limited number of events at a low level of activity.
4. Reconstructive or rehabilitative—participation in a prescribed program of corrective exercises or adapted sports.

Classifying students for physical education has frequently been discussed at the National Conference on Physicians and Schools, held biennally under auspices of the AMA. Physicians, educators, and public health personnel attending these meetings have generally concluded:

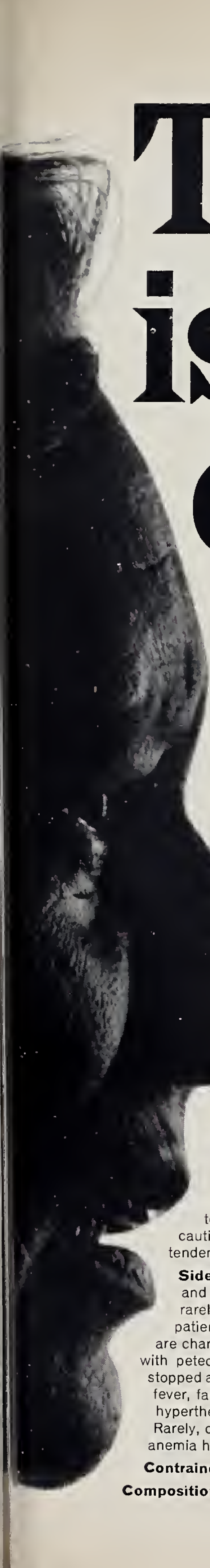
—Better communication is needed among all parties in such a classification program.

—The physician should provide all pertinent information on why an excuse from exercise has been issued. This can be done without divulging privileged information by providing an interpretation of the physical condition rather than medical findings.

—Inadequate physical education programs must

(Continued on Page 36)

"COCA-COLA" AND "COKE" ARE REGISTERED TRADE-MARKS WHICH IDENTIFY ONLY THE PRODUCT OF THE COCA-COLA COMPANY.
For the taste
you never
get tired of.



This pain is getting on my nerves.

Patients in pain often experience concomitant anxiety and tension, which may add to the burden of pain.

For such patients, you may want to prescribe a preparation that offers more than simple analgesia.

A good choice is often EQUAGESIC® (meprobamate and ethoheptazine citrate with aspirin). It helps relieve pain. And anxiety. And skeletal muscle spasm as related to pain or anxiety and tension.

Equagesic® TABLETS (meprobamate and ethoheptazine citrate with aspirin)

Wyeth®

Precautions: Keep out of reach of children. Carefully supervise dose and amounts prescribed, especially for patients prone to overdose themselves. Excessive prolonged use of meprobamate may result in dependence or habituation in susceptible persons—as ex-addicts, alcoholics, severe psychoneurotics. Withdraw gradually after prolonged high dosage to avoid possibly severe withdrawal reactions including epileptiform seizures. Warn patients of possible reduced alcohol tolerance. If drowsiness, ataxia or visual disturbances occur, reduce dose. If symptoms persist, caution patients against operating machinery or driving. Give cautiously to patients with suicidal tendencies. Treat attempted suicide with immediate gastric lavage and appropriate supportive therapy.

Side Effects: Ethoheptazine and aspirin may occasionally cause nausea, vomiting, epigastric distress, and rarely dizziness and CNS depression. Overdosage may result in salicylate intoxication. Meprobamate rarely causes allergic or idiosyncratic reactions. These reactions, sometimes severe, can develop in patients receiving only 1 to 4 doses who have had no previous contact with meprobamate. Mild reactions are characterized by urticarial or erythematous maculopapular rash. Acute nonthrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever have been reported. Meprobamate should be stopped and not reinstituted. Severe reactions, observed very rarely, include angioedema, bronchial spasms, fever, fainting spells, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case) and hyperthermia. A few cases of leukopenia, usually transient, have been reported following prolonged dosage. Rarely, cases of aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis, and hemolytic anemia have been reported; almost always, in the presence of known toxic agents.

Contraindications: History of sensitivity or severe intolerance to aspirin or meprobamate.

Composition: 150 mg. meprobamate, 75 mg. ethoheptazine citrate and 250 mg. aspirin per tablet.

Wyeth Laboratories Philadelphia, Pa.

advance notice

NINTH ANNUAL
NEVADA CANCER SEMINAR

RENO/SPARKS NUGGET
CONVENTION CENTER

MAY 11 & 12, 1967

ENJOY RENO, A COSMOPOLITAN CITY (Deluxe Facilities, Stores, Entertainment, Excellent Transportation),—COMBINED WITH THE ATMOSPHERE OF THE TRUE WEST (Alpine Scenery, Desert Ranges, Ghost Towns) —WATER SPORTS, SKIING, FISHING, HUNTING, GOLFING...AND DIGNIFIED GAMING.

ACTIVITIES FOR WIVES

CO-SPONSORS

NEVADA STATE MEDICAL AND DENTAL ASSOCIATIONS

INTERNATIONL COLLEGE OF SURGEONS, NEVADA CHAPTER

RENO CANCER CENTER

AMERICAN CANCER SOCIETY, NEVADA DIVISION, INC.

For further information, write American Cancer Society, Box 7432, Reno, Nevada.

★

Specialized Service
IN
PROFESSIONAL LIABILITY
INSURANCE

is a high mark
of distinction

THE
MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA
Professional Protection Exclusively since 1899

NORTHERN CALIFORNIA OFFICE:
Gordon C. Jones and John K. Galloway, Representatives
1518 Fifth Avenue, San Rafael, Telephone: 415-453-5140
Mailing Address: P.O. Box 1079, San Rafael 94902
SOUTHERN CALIFORNIA OFFICE:
Davis S. Spencer and Grady T. Dyer, Representatives
Suite 3, 33 East Huntington Drive, Arcadia, Tel. 213-681-5239
Mailing Address: P.O. Box 543, Arcadia 91008

Classsifying Youngsters For Physical Education

(Continued from Page 24)

be strengthened. Many communities need to increase the variety of their activities.

—Every student should have the kind of exposure to physical education which promotes understanding of the significance of physical activity in maintaining health and in motivating the individual to regular lifelong physical activity.

Patients Don't Always Follow Doctor's Orders

Physicians have long known that some patients don't follow orders for taking medicine, or refuse to take it at all.

A report in the January 16 *Journal of the American Medical Association* indicates how frequently this can occur. Among a group of Cleveland tuberculosis patients, 15 (30 per cent) of 50 patients were uncooperative about taking one drug, and 14 (42 per cent) of 33 failed to follow doctors' orders for taking another.

"In this era of potent and dangerous medications, patient reliability in taking drugs becomes a primary consideration," the article said. "Most physicians are aware that some patients fail to take prescribed medicines, but few realize the extent of the problem."

The study started with clinic and pharmacy records at the University Hospital of Cleveland, which indicated that 50 persons in the outpatient department were supposed to be taking the drugs isoniazid and/or aminosalicylic acid.

It became apparent large numbers of patients weren't following directions when urine tests failed to reveal any trace of the drugs.

There were 20 men and 30 women patients, ranging in age from 17 to 78, with an average and median age of 45. Forty-eight were known to have tuberculosis. Forty-five were Negro and five, white. All but one lived in a poor downtown section of east Cleveland. Forty-five (90 per cent) understood perfectly the instructions for taking their medicines, the authors said.

"Detection of the patient who is not taking his medicine is difficult," the report said. "Simply asking him seems unsatisfactory since patients tend to claim that they have taken medicine when direct evidence indicates that they have not.

An earlier study, for instance, indicated that 83 per cent of families claimed to have given a full 10-day course of penicillin to their children when evidence indicated that 82 per cent had stopped the medicine by the ninth day.

Some patients also tend to deny taking medicines when they have. One study found that 29 of 38 patients who had taken meprobamate, barbiturates, or phenothiazines denied taking these drugs.

dependability not dependence

ATARAX®
(hydroxyzine HCl) tablets, syrup

Product Information

Contraindications: Use in pregnancy: When administered to rats at doses substantially above the human therapeutic range, hydroxyzine induced fetal abnormalities. Until human clinical data are available adequate to establish safety in early pregnancy, hydroxyzine is contraindicated in early pregnancy. The drug is contraindicated for patients who have shown previous hypersensitivity to it.

Precautions: Hydroxyzine HCl may potentiate narcotics, barbiturates, meperidine and other CNS depressants. In conjunctive use, dosage of these drugs should be reduced by as much as 50 per cent. Atropine and other belladonna alkaloids are not affected by hydroxyzine. Because drowsiness may occur, patients given hydroxyzine should be cautioned against driving a car or operating dangerous machinery.

Adverse reactions: No serious side effects resulting from the oral administration of hydroxyzine have been reported and confirmed to date. Therapeutic doses seldom produce impairment of mental alertness. The transitory drowsiness that may occur usually disappears spontaneously in a few days of continued therapy or can be corrected by dosage reduction. Dryness of the mouth may be encountered at higher doses.
The absence of toxic effects on the liver and bone marrow has been demonstrated by extensive clinical use at recommended doses for more than four years of uninterrupted therapy and by experimental studies in which excessively high doses were given. Involuntary motor activity, including rare instances of tremor and convulsions, has been reported, usually with considerably higher than recommended doses.

Dosage: Ranges from 25 mg. t.i.d. to 100 mg. q.i.d.

References: 1. Fishbein, M.: J. Am. Geriatrics Soc. 10:911 (Nov.) 1962. 2. Dale, P. W.: New England J. Med. 265:185 (July 27) 1961. 3. Settel, E.: Am. Pract. 8:1584 (Oct.) 1957.

J.B. ROERIG DIVISION
CHAS. PFIZER & CO., INC.
NEW YORK, N.Y. 10017

What Areas Are Covered By the American Medical Association

What is the American Medical Association? How does it operate? What are some of its major areas of activities and accomplishments?

A 32-page pamphlet published by the Association readily answers such questions. The publication is equally suitable for distribution to medical society members, prospective members and the general public.

Requests for single copies or quantity orders should be directed to the Program Services Department, American Medical Association, 535 North Dearborn Street, Chicago, Illinois 60610. There's no charge.

AMA Announces Official Policy On Chiropractic

The American Medical Association has adopted an official policy on Chiropractic.

The statement, initiated by the Committee on Quackery and presented by the Board of Directors, was adopted unanimously by the House of Delegates at the Clinical Convention in November 1966.

The official policy of medicine on chiropractice states: "It is the position of the medical profession that chiropractic is an unscientific cult whose practitioners lack the necessary training and background to diagnose and treat human disease. Chiropractic constitutes a hazard to rational health care in the United States because of the substandard and unscientific education of its practitioners and their rigid adherence to an irrational, unscientific approach to disease causation.

"In 1965, a United States District Court, in upholding a state's constitutional right to refuse to license chiropractors, said that 'since chiropractic claims to be a complete and independent healing art capable of curing almost all kinds of disease, the state Legislature may have felt that the requirement of a foundation in materia medica and surgery . . . would be a protection to the public.' Without dissent, the United States Supreme Court affirmed the decision.

"The wisdom of these decisions by the nation's highest courts justifies the medical profession's educational program of alerting the nation to the public health threat posed by the cult of chiropractic.

"Patients should entrust their health care only to those who have a broad scientific knowledge of diseases and ailments of all kinds, and who are capable of diagnosing and treating them with all the resources of modern medicine. The delay of proper medical care caused by chiropractors and their opposition to the many scientific advances in modern medicine, such as life-saving vaccines, often ends with tragic results."

DIRECTORY

HOSPITALS • SANITARIUMS • REST HOMES

Exclusively for
ACUTE AND CHRONIC
ALCOHOLISM

Woodside Acres Hospital

MEMBER AMERICAN HOSPITAL ASSOCIATION
1600 Gordon Street Redwood City, California
EMerson 8-4134

Twin Pines
NEUROPSYCHIATRIC HOSPITAL

OPEN, VISITING AND CONSULTING STAFF

Eclectic Treatment of acute and chronic emotional and neuropsychiatric disorders

In-Patient and out-patient facilities

Visiting and consulting staff

Attending Staff
A. T. VORIS, M.D., Medical Director
DAVID S. WILDER, M.D. • ROBERT E. JAMES, M.D.
ALLEN GOLDWATER, M.D. • ROBERT L. MEIERS, M.D.

BELMONT, CALIFORNIA ESTABLISHED 1925 **Tel. (415) 591-8951** Located 22 miles south of San Francisco. Accessible to transportation.

Accredited by Joint Commission on Accreditation of Hospitals

Barlow Sanatorium For Tuberculosis

Partially endowed private eleemosynary hospital for Pulmonary Tuberculosis patients with hopeful prognosis, or diagnostic problems of diseases of the chest • Sliding scale of rates from $12.00 to $30.00 per day according to ability to pay. Includes medical supervision, routine nursing care, laboratory and x-ray services. • Accredited by Joint Commission on Accreditation of Hospitals

2000 STADIUM WAY, CHAVEZ RAVINE, LOS ANGELES, CALIFORNIA 90026 MAdison 8-4165

• Affiliated with University of Southern California Medical School • Member of Western Hospital Association, California Hospital Association and Hospital Council of Southern California • Apply by contacting Medical Director or by direct referral through morning clinic.

HOWARD W. BOSWORTH, M.D., ***Medical Director***

---

belmont hills neuropsychiatric center
ADULT TREATMENT SERVICE

A "therapeutic community" acute treatment service offering individual psychotherapy, community group therapy and small group therapy, somatic therapy, social casework and occupational-recreational therapy. A comprehensive diagnostic inventory comprised of psychodynamic evaluation, psychometric studies, family interviews, physical examination and appropriate consultation is provided for both treatment and diagnostic referrals.

Address inquiries:
HARVEY J. NEWTON, M.A., M.D., ***Center Director***
MARGARET H. SAINIO, Ph.D., M.D., ***Medical Director,***
Adult Treatment Service
P. O. Box 37
Belmont, California
(415) 593-2143

COMPTON FOUNDATION HOSPITAL
FORMERLY COMPTON SANITARIUM
820 West Compton Boulevard
COMPTON, CALIFORNIA 90220
636-1185 631-1148

G. CRESWELL BURNS, M.D.
Medical Director
HELEN RISLOW BURNS, M.D.
Assistant Medical Director

MEMBER OF
American Hospital Association and
National Association of Private Psychiatric Hospitals

High Standards of Psychiatric Treatment
. . . . . . Serving the Los Angeles Area

Fully Approved by Central Inspection Board of APA

Accredited by
Joint Commission on Accreditation of Hospitals

For people who eat more than one meal a day

A PLEGINE (Phendimetrazine bitartrate) tablet taken an hour before each meal affords peak activity at mealtime, when it is needed most. Reminds patients to eat according to your prescribed diet plan.

t.i.d./Plegine®

BRAND OF Phendimetrazine bitartrate

provides peak appetite suppression when it's needed most

Indications: Recommended in the management of excessive appetite leading to obesity.

Cautions and Contraindications: No adverse effects on blood pressure, heart rate, and respiration have been reported. However, as is true for all medications of this type, PLEGINE (Phendimetrazine bitartrate) is not recommended for patients with coronary disease, severe hypertension, or thyrotoxicosis, and should be used with caution in highly nervous or agitated individuals.

Side Effects: There have been occasional reports of insomnia and nervousness. Rare instances of mouth dryness, nausea, blurring of vision, dizziness, constipation, and stomach pain have been noted.

AYERST LABORATORIES New York, N.Y. 10017 · Montreal, Canada

6706

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

COMPLETE T-3
THYROID TESTING CAPABILITY
FROM ONE MANUFACTURER

Tri-IONEX®
CHEMICAL REAGENTS

SCINTIGRON®
INSTRUMENTATION

- T-3 THYROID FUNCTION TESTING
- SCHILLINGS VITAMIN B^{12} TOLERANCE IRON DEFICIENCY ANEMIA
- RED CELL SURVIVAL
- 24HR THYROID UPTAKE
- BLOOD VOLUMES

TO KNOW MORE ABOUT WHAT COMPLETE T-3 THYROID TESTING CAPABILITY FROM CURTIS WILL MEAN TO YOU . . . WRITE OR CALL COLLECT

16 YEARS SERVING THE MEDICAL FIELD

MANUFACTURER OF A COMPLETE INSTRUMENT LINE FOR YOUR RADIOISOTOPE PROGRAM.

CURTIS NUCLEAR CORPORATION
1948 E. 46th St., Los Angeles, Calif. 90058/(213) 232-3531

Manufacturers of fine medical equipment: Scintiscanner®, Scintigram®, Placentagron®, Probitron®, Dual Scintillation and special application systems

AMERICAN MEDICAL BUILDING GUILD

. . . *designs and constructs medical buildings from coast to coast*

WESTERN MEDICAL BUILDINGS, INC. is a division of the **AMERICAN MEDICAL BUILDING GUILD** formed to serve the medical profession in the Western United States. **WESTERN MEDICAL BUILDINGS, INC.** is fully staffed with specialists experienced in medical building design and construction in this area of the country.

For a 12-page, color brochure write —

WESTERN MEDICAL BUILDINGS, INC.
(Div. of AMERICAN MEDICAL BUILDING GUILD)
5135 Harbor Drive, Suite 'E'
San Diego, California 92106
Telephone collect (714) 224-2793

Air Pollution Conference To Meet July 21-28 in Denver

The 1968 Air Pollution Medical Research Conference will be held July 21-23, 1968, in Denver, Colorado. The AMA has been given a grant by the U.S. Public Health Service to partially cover conference expenses. James G. Telfer, M.D., Director of the AMA Environmental Health Department, has been appointed the principal investigator for the grant. The meeting, open to the public, will focus on physicians mainly.

New Snakebite Treatment

A Miami surgeon has developed a new technique for treating snakebite: Immediately cut away a half-dollar layer of tissue around the bite.

Getting rid of this venom-filled tissue as soon as possible is "the most effective treatment," according to Clifford C. Snyder, M.D. The technique marks a considerable departure from the traditional cross-cutting of the bite and drawing-out of the venom.

Dr. Snyder's successful treatment of 32 snakebite victims in the past two years is reported in the Medical News section of the December 26 *Journal of the American Medical Association.*

Immediate tissue removal could be done by a bitten person, Dr. Snyder noted. A relatively shallow cut the size of a large coin probably would get rid of most of the easily recognizable venom-killed tissue.

Not many persons would be able to perform this task, however, due to the shock of the bite and other factors, Dr. Snyder admits. A simple slash across the bite wound would be an alternate measure preceding the more radical treatment.

A tourniquet will prevent the venom's spread until a physician can perform the excision, Dr. Snyder said. The tourniquet should cut off only superficial circulation; it should be possible to insert a finger between the tightened bandage and the skin.

It's important that a tourniquet be applied immediately, Dr. Snyder said. Later, at the physician's office or hospital, additional tissue may be removed and antivenom administered through the artery serving the bitten area.

Despite the well-known tissue-killing effects of snake venom, no amputations have been necessary in Dr. Snyder's 32 patients.

Five of those treated were bitten by cottonmouth moccasins and 27 by rattlesnakes. About 87 per cent of snakebites in the U.S. are inflicted by the rattlesnake.

Dr. Snyder is associate professor of surgery at the University of Miami and is a plastic surgeon in private practice. Associated with him in continuing research on the technique are Robert P.

(Continued on Page 58)

California
Medicine

Blood Coagulation and the Coumarin Anticoagulant Drugs

PAUL M. AGGELER, M.D., *San Francisco*

Dr. Leslie L. Bennett*: Ladies and gentlemen, it gives me unusual pleasure to introduce Dr. Paul M. Aggeler as Faculty Research Lecturer from the San Francisco Division of the Academic Senate for 1966. There is no higher honor that the faculty can bestow upon one of its colleagues than to select him to represent publicly the faculty's common urge toward creativity which primarily manifests itself in our scientific setting as research.

Dr. Aggeler is uniquely a Californian. He is one of those rare individuals, a native son. His birth occurred in Ferndale among the redwoods of Humboldt County. After attending St. Ignatius High School in San Francisco, he enrolled as a premedical student at the University of California in Berkeley in 1929 and received the M.D. degree from the University of California in 1937. Dr. Aggeler interned at the University of California Hospital and was successively Assistant Resident, Research Fellow in Medicine and Resident in Medicine. Except for one year of training spent at the San Joaquin General Hospital, his professional and scientific training was entirely at the University of California; thus I say he is uniquely a California product.

As a medical student, Dr. Aggeler came under the influence of Dr. Salvatore Lucia and in Dr. Lucia's laboratory began a systematic study of factors affecting the prothrombin time as then determined by the method devised by Armand Quick. These studies resulted in his first publication dealing with clotting mechanisms, which appeared in 1938. Early recognizing the importance of the work of Dam and Almquist on the bleeding diathesis of vitamin K-deficient chicks and the probable significance for human disease of the observation of Greaves and Schmidt of the effectiveness of vitamin K in controlling the bleeding of rats with biliary fistulas, he began in the late 1930's his investigation of the role of vitamin K in controlling prothrombin levels in patients with jaundice and hepatic disease. This is the first of his classic works referred to in the citation of the Research Committee.

In this year, 1966, when there is increased awareness of the ethical responsibilities of physicians engaged in clinical research, it is important to recall that Dr. Aggeler was unusually adept at doing venipunctures on himself to obtain blood to set up the controls necessary for his investigation of the clotting mechanism in patients with jaundice. His critical observation and studies of patients with hemophilia led to his discovery of plasma thromboplastin component deficiency and resulted in an explosion of knowledge about hemophilia. Most recently, the same critical clinical acumen and inquiring mind resulted in his discovery of a genetic control of warfarin metabolism. His national and international reputation is thoroughly established and I shall not repeat the list of editorships he holds, advisory roles he has filled and the honors that have come his way. Let me simply say that I am proud to present my colleague for over a quarter of a century, the man who completely meets the criteria for scholar and gentleman, Dr. Paul Aggeler.

The Ninth Annual Faculty Research Lecture, University of California San Francisco Medical Center, 23 March 1966.

Submitted 20 January 1967.

Reprint requests to: Editorial Office, Room 4320, S.F. General Hospital, 1001 Potrero Avenue, San Francisco 94110.

*Morris Herzstein Professor of Physiology.

DR. PAUL M. AGGELER*[1]: Professor Bennett, members of the Academic Senate, ladies and gentlemen: I am sensible of the very great honor which the Academic Senate has bestowed on me, and I am most grateful. I am overwhelmed to be included in the illustrious company of those who have preceded me in this distinction. What I have done seems inconsequential in comparison with their accomplishments. I am pleased, however, that what I have done and what I have been has seemed meritorious to my colleagues.

At the outset I must acknowledge, with thanks, the great debt I owe to many associates, fellows and technical assistants for their help in my research endeavors. I am afraid that much of what I am given credit for is really their work. I am also grateful to the Board of Directors and Administration of the Children's Hospital of San Francisco for providing sponsorship and space for much of my activities during the past 10 years. Finally, I would like to express my thanks to my first mentor, Dr. Salvatore Lucia, for his stimulation, encouragement and support when I worked under him first as a research fellow and later as an Instructor in the Division of Preventive Medicine.

I have approached this lecture with more than the ordinary amount of apprehension. It would be appropriate perhaps to spend some time in philosophizing about medical research, but I do not feel qualified for that task. In fact, I cringe whenever I am referred to as a researcher. For the better part of my career I have held a title in the clinical professorial series. I have been a practicing internist specializing in hematology with special interest in the hemorrhagic disorders. I have been a member of that branch of the faculty whose principal concern has been with the care of patients. Such research as I have been able to accomplish has sprung directly from the bedside and has usually had immediate clinical relevance. With the increasing complexity of medical science it has become more difficult to pursue this kind of career, but I hope the opportunity will not be completely lost. For me it has been an exciting and rewarding way of life.

In attempting to shape my discussion this morning into a form which might have some interest for the many disciplines represented on this campus, I have decided to discuss two related topics. In the first part of the hour I will attempt to trace briefly the history of the development of our present concepts of the blood clotting mechanism as I have seen it unfold during the past 30 years. Blood clotting is somewhat unusual among physiologic processes in that a great deal of what we know about it has come from careful study of its derangement in sick patients. I will stress particularly the discoveries made through investigation of coagulation defects in such individuals. Because of the limitations of time this must, of necessity, be oversimplified and must ignore the enormous contributions of many investigators. During the second part of the hour I will outline some recent work we have done with a class of drugs which interferes with blood clotting.

I first became interested in blood clotting during my internship in this hospital in the year 1936-37. During that year I was involved in the care of several patients suffering from hemophilia. At that time the nature of the disease was poorly understood, and its treatment highly unsatisfactory. I had no special training for research and certainly no intention of becoming seriously involved in it, but I did have an urge to find out more about the curious inability of the hemophiliac's blood to clot. In partial explanation of why, almost 30 years later, I am still involved in blood clotting problems, I would like to quote a portion of the introduction to their book on blood coagulation by Drs. Rosemary Biggs and Roger Macfarlane: "To the uninitiated, the fascination that this work has for its devotees must seem difficult to understand. But a number of explicable factors operate to develop this attraction, so that once involved in it the new recruit is almost powerless to draw back. To begin with, the spontaneous transformation of fluid blood to solid clot has a stimulating effect on the curiosity like that produced by a well executed conjuring trick. One feels that so simple and striking a phenomenon must have a simple and striking explanation and the urge to discover it is immediate. Next there is the deceptive ease with which the work can be started. All that is needed is a few glass tubes and a watch, a supply of blood and ingenuity. Later the more mechanically-minded investigator will discover that almost equally good results can be obtained by apparatus of limitless complexity. . . . Thereafter experiments are easy to plan and carry out, and provide unlimited opportunity for being most satisfyingly clever."

Man's fascination with the phenomenon of blood clotting is very old. Hippocrates and Aristotle thought it was simply a matter of solidification on cooling. Later, a popular theory held that the blood contained an accumulation of minute particles of fibrin-like material which were kept widely dispersed by the circulation, but which would quickly aggregate with the cessation of blood flow. Andrew Buchanan, whose work was published in Glasgow in 1845, probably deserves credit as the founder of the modern theory of blood coagulation. He was the first to show that it took two substances to cause coagulation: "Liquid fibrin" or fibrinogen as we would now call it, which by itself was stable, and a substance capable of affecting the "liquid fibrin" and transforming it into an insoluble form. During the last half of the Nineteenth century a large number of investigations on blood clotting were undertaken. The conclusions of different investigators were divergent and often contradictory. However, a number of important discoveries were made: Fibrogen, the soluble precursor of fibrin, was isolated from the plasma by salt precipitation; thrombin, the agent which converted fibrogen to fibrin, was extracted from fresh serum by alcohol precipitation; tissue extracts were shown to hasten blood clotting but not to initiate it; and the need for ionized calcium in the clotting mechanism was demonstrated.

---

*[1] Professor of Medicine.

In a magnificent monograph on blood clotting published in 1905, containing some 490 references, Paul Morawitz summarizes the work done during the previous half century by Andrew Buchanan, Alexander Schmidt, E. Bruecke, P. Denis, L. Frédéricq, J. Bordet, L. Wooleridge, O. Hammarsten, M. A. Dastre, C. A. Pekelharing, M. Arthus and many others, and proposed what we now call the "classical" theory of blood clotting, shown in Figure 1.

This theory proposes that in the presence of ionized calcium and thromboplastin, prothrombin is converted to thrombin, which then converts fibrinogen to fibrin. (The Roman numerals, which I have inserted after the names of these substances, refer to the system of nomenclature of blood clotting factors recently adopted by the International Committee on Hemostasis and Thrombosis).

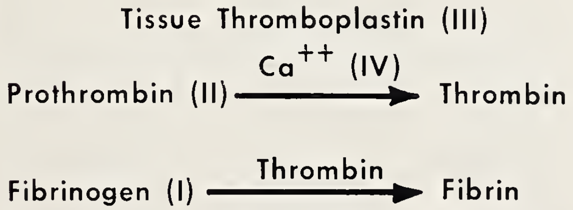
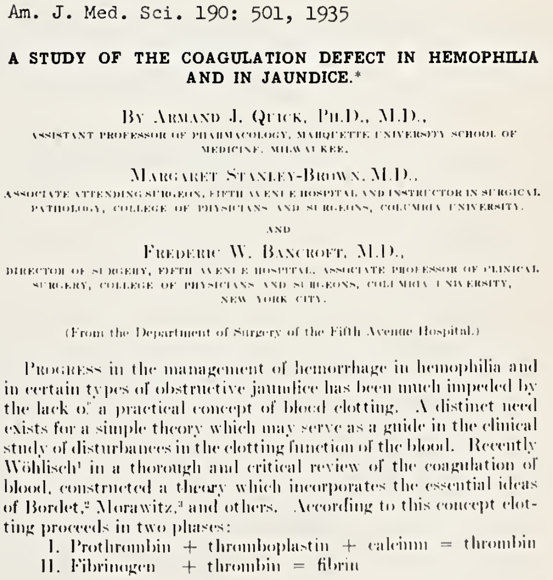


Figure 1.—Morawitz' "classical" theory of blood clotting, 1905.

Morawitz also recognized that the production of thrombin from its plasma precursors was in some way initiated or at least enhanced by contact with a foreign surface.

During the early part of the Twentieth century a number of investigators including C. H. Mills, P. Nolf, A. Fischer, J. J. Mellanby, W. H. Howell and others proposed conflicting theories. The "classical" theory of Morawitz was by no means universally accepted.

Dr. Armand Quick, now Professor Emeritus of Physiology at Marquette University, was one of the pioneers in the present era of blood coagulation research. The paper shown in Figure 2, published in 1935, did much to change the course of my own life. In it Dr. Quick reaffirmed the "classical" theory of blood clotting as proposed by Morawitz and introduced his now famous one-stage method for the measurement of prothrombin. The success of Quick's test depended in large part on the use of a stable, standardized tissue thromboplastin prepared from rabbit brain. In the test, plasma is recalcified in the presence of an optimum amount of this tissue extract. When normal plasma is used in the test it clots in 11 to 12 seconds. Hemophilic plasma clots in a normal time, but plasma from many patients with jaundice requires as long as 30 to 40 seconds or longer. Quick concluded that the prothrombin concentration is reduced in the plasma of many patients with jaundice but is normal in the plasma of patients with hemophilia.



Am. J. Med. Sci. 190: 501, 1935

A STUDY OF THE COAGULATION DEFECT IN HEMOPHILIA AND IN JAUNDICE.*

BY ARMAND J. QUICK, PH.D., M.D.,
ASSISTANT PROFESSOR OF PHARMACOLOGY, MARQUETTE UNIVERSITY SCHOOL OF MEDICINE, MILWAUKEE.

MARGARET STANLEY-BROWN, M.D.,
ASSOCIATE ATTENDING SURGEON, FIFTH AVENUE HOSPITAL AND INSTRUCTOR IN SURGICAL PATHOLOGY, COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY.

AND

FREDERIC W. BANCROFT, M.D.,
DIRECTOR OF SURGERY, FIFTH AVENUE HOSPITAL, ASSOCIATE PROFESSOR OF CLINICAL SURGERY, COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK CITY.

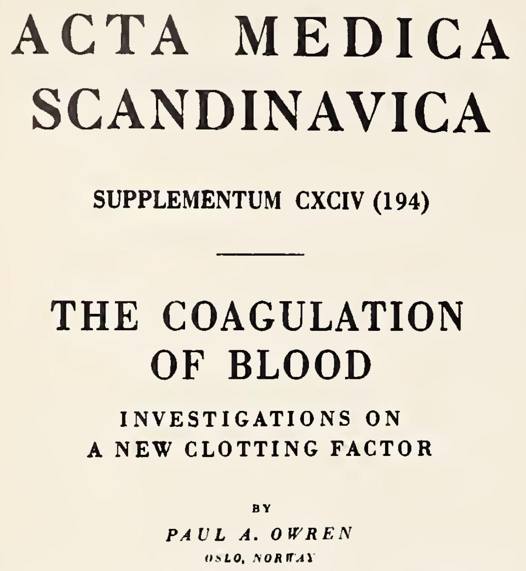
(From the Department of Surgery of the Fifth Avenue Hospital.)

PROGRESS in the management of hemorrhage in hemophilia and in certain types of obstructive jaundice has been much impeded by the lack of a practical concept of blood clotting. A distinct need exists for a simple theory which may serve as a guide in the clinical study of disturbances in the clotting function of the blood. Recently Wöhlisch[1] in a thorough and critical review of the coagulation of blood, constructed a theory which incorporates the essential ideas of Bordet,[2] Morawitz,[3] and others. According to this concept clotting proceeds in two phases:

I. Prothrombin + thromboplastin + calcium = thrombin
II. Fibrinogen + thrombin = fibrin

Figure 2.—Title page of article by Dr. Armand J. Quick which introduced the one-stage method for the measurement of plasma prothrombin.

Shortly after this test was introduced both vitamin K and dicumarol were synthesized and made available for general use. Quick's test proved to be ideal for measuring their effects. I learned to do this simple but very useful little test and thereupon became the local blood clotting expert.



ACTA MEDICA
SCANDINAVICA

SUPPLEMENTUM CXCIV (194)

THE COAGULATION
OF BLOOD

INVESTIGATIONS ON
A NEW CLOTTING FACTOR

BY

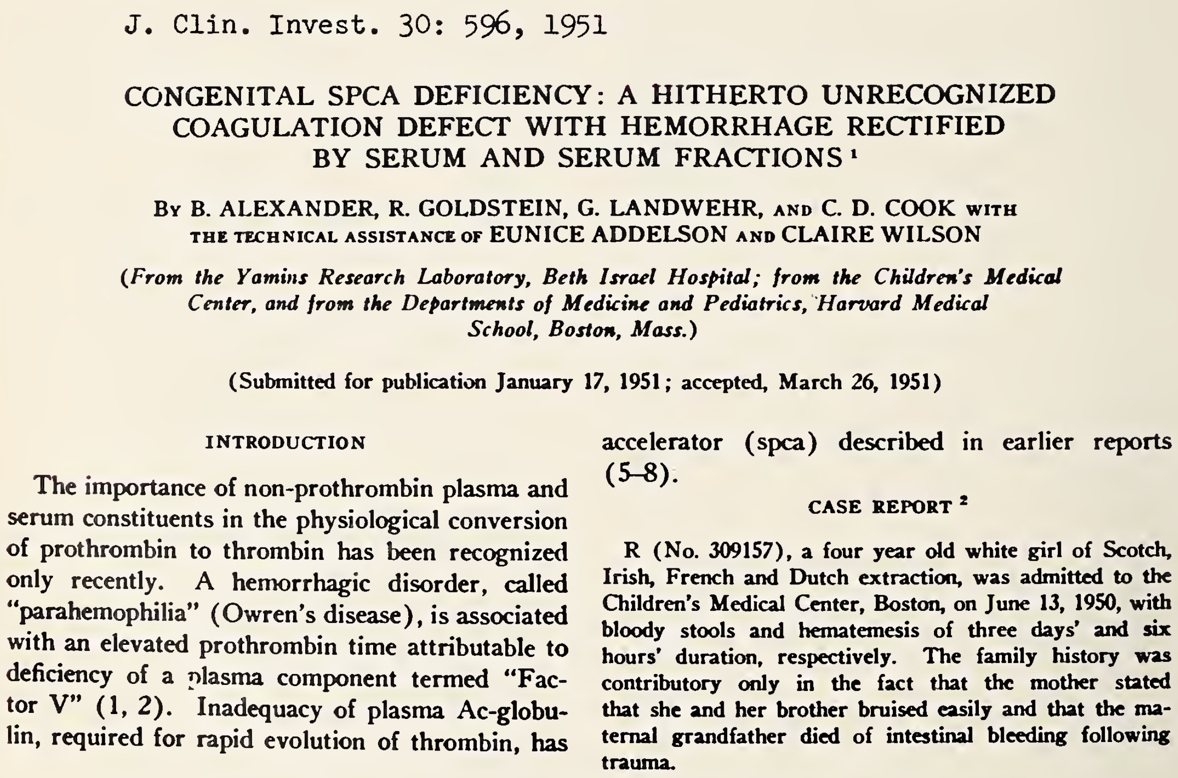
PAUL A. OWREN

OSLO, NORWAY

Figure 3.—Owren's article in which he described proaccelerin (now called Factor V). Source: Acta Medica Scandinavica, Suppl. 194:1-327, 1947.

In 1947, Dr. Paul Owren published his magnificent treatise concerning the investigations on a new blood clotting factor which he had conducted in seclusion in Nazi-occupied Norway during World War II (Figure 3). The subject of his studies was a 29-year-old woman who had suffered all her life from a bleeding tendency characterized by severe epistaxis, hematomata, bleeding from lacerations and menorrhagia. She had a prolonged "prothrombin" time by Quick's test but was shown by other methods to have a normal amount of prothrombin in her blood. Owren showed that the defect in this woman's blood clotting was not a deficiency of prothrombin but rather a delayed conversion of prothrombin to thrombin. Her prothrombin could be converted to thrombin if a small amount of normal plasma, from which all of the prothrombin had been removed by adsorption, was added to her plasma. This new clotting factor, which Owren called *proaccelerin,* was not removed from normal plasma by adsorption, and it disappeared rapidly from normal serum.

At about the same time Quick noted that the prothrombin time (as measured by his test) gradually lengthened as normal blood was stored; he attributed this to the disappearance of what he



J. Clin. Invest. 30: 596, 1951

CONGENITAL SPCA DEFICIENCY: A HITHERTO UNRECOGNIZED COAGULATION DEFECT WITH HEMORRHAGE RECTIFIED BY SERUM AND SERUM FRACTIONS[1]

By B. ALEXANDER, R. GOLDSTEIN, G. LANDWEHR, AND C. D. COOK WITH THE TECHNICAL ASSISTANCE OF EUNICE ADDELSON AND CLAIRE WILSON

(From the Yamins Research Laboratory, Beth Israel Hospital; from the Children's Medical Center, and from the Departments of Medicine and Pediatrics, Harvard Medical School, Boston, Mass.)

(Submitted for publication January 17, 1951; accepted, March 26, 1951)

INTRODUCTION

The importance of non-prothrombin plasma and serum constituents in the physiological conversion of prothrombin to thrombin has been recognized only recently. A hemorrhagic disorder, called "parahemophilia" (Owren's disease), is associated with an elevated prothrombin time attributable to deficiency of a plasma component termed "Factor V" (1, 2). Inadequacy of plasma Ac-globulin, required for rapid evolution of thrombin, has accelerator (spca) described in earlier reports (5–8).

CASE REPORT[2]

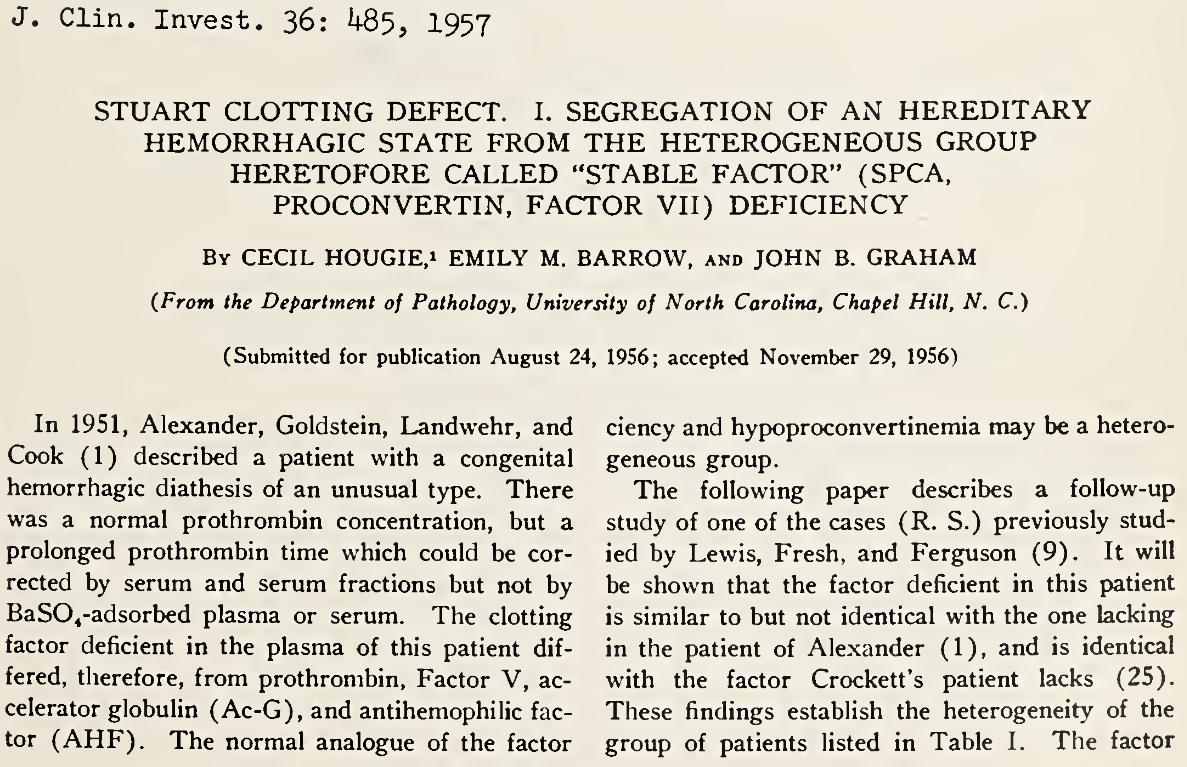
R (No. 309157), a four year old white girl of Scotch, Irish, French and Dutch extraction, was admitted to the Children's Medical Center, Boston, on June 13, 1950, with bloody stools and hematemesis of three days' and six hours' duration, respectively. The family history was contributory only in the fact that the mother stated that she and her brother bruised easily and that the maternal grandfather died of intestinal bleeding following trauma.

Figure 4.—Title page of paper by Goldstein et al. which first described SPCA (now called Factor VII).

called the "labile factor" of prothrombin. Walter Seegers and his coworkers also found that their preparation of purified prothrombin could be converted to thrombin much more rapidly in the presence of a factor they found in beef serum which they called *accelerator globulin,* or AcG. Within a short time it became apparent that the activities attributed to proaccelerin, labile factor and accelerator globulin all arose from the same factor. In the present system of nomenclature this is known as Factor V.

In 1951 Dr. Benjamin Alexander and his associates at the Beth Israel Hospital in Boston described the congenital deficiency of a new clotting factor which they called *serum prothrombin conversion accelerator* or SPCA (now known as proconvertin or Factor VII). This factor was found to be deficient in a four-year-old girl who suffered from easy bruising, spontaneous epistaxis and recurrent bouts of gastrointestinal bleeding (Figure 4). They showed that this factor, like Factor V was required for the conversion of prothrombin to thrombin. Unlike Factor V, it was quite stable on storage, was present in human serum and was readily adsorbed onto barium sulfate. Advantage was taken of its adsorbability to prepare an artificially depleted normal plasma as a substrate for testing purposes. During the next few years a number of cases were diagnosed as instances of congenital deficiency of Factor VII on the basis of the results of tests employing such plasma. After a time it became apparent, however, that one group of these patients differed from the remainder in certain essential details.

In 1957, Cecil Hougie, E. M. Barrow and John B. Graham in North Carolina segregated these two types of cases and clearly demonstrated that one type was identical to that observed in Alexander's original patient with Factor VII deficiency, while the other was caused by the absence of a previously unknown factor which they called the Stuart Factor, after the family name of their first patient in whom its absence was demonstrated (This is now known as Factor X) (Figure 5). Their first patient was a 36-year-old farmer who had had frequent epistaxis and hematomata, and occasional mild hemarthrosis. They showed that Factor X was required for coagulation of blood whether tissue thromboplastin was present or not. Factor VII, on the other hand, was found to be required only as a co-factor of tissue thromboplastin. It was not required for the intrinsic production of blood thromboplastin. It was apparent, then, that at least three factors (Factors V, VII



J. Clin. Invest. 36: 485, 1957

STUART CLOTTING DEFECT. I. SEGREGATION OF AN HEREDITARY HEMORRHAGIC STATE FROM THE HETEROGENEOUS GROUP HERETOFORE CALLED "STABLE FACTOR" (SPCA, PROCONVERTIN, FACTOR VII) DEFICIENCY

BY CECIL HOUGIE,[1] EMILY M. BARROW, AND JOHN B. GRAHAM

(From the Department of Pathology, University of North Carolina, Chapel Hill, N. C.)

(Submitted for publication August 24, 1956; accepted November 29, 1956)

In 1951, Alexander, Goldstein, Landwehr, and Cook (1) described a patient with a congenital hemorrhagic diathesis of an unusual type. There was a normal prothrombin concentration, but a prolonged prothrombin time which could be corrected by serum and serum fractions but not by $BaSO_4$-adsorbed plasma or serum. The clotting factor deficient in the plasma of this patient differed, therefore, from prothrombin, Factor V, accelerator globulin (Ac-G), and antihemophilic factor (AHF). The normal analogue of the factor ciency and hypoproconvertinemia may be a heterogeneous group.

The following paper describes a follow-up study of one of the cases (R. S.) previously studied by Lewis, Fresh, and Ferguson (9). It will be shown that the factor deficient in this patient is similar to but not identical with the one lacking in the patient of Alexander (1), and is identical with the factor Crockett's patient lacks (25). These findings establish the heterogeneity of the group of patients listed in Table I. The factor

Figure 5.—Title page of paper by Hougie et al. in 1957 which described the Stuart Factor (now called Factor X).

and X) were required for conversion of prothrombin to thrombin in the presence of an optimal amount of tissue thromboplastin and calcium, as shown in Figure 6.

It was clear also that extracts of tissues were capable of supplying optimum amounts of thromboplastin, but it remained a mystery how this thromboplastic activity developed in blood kept free of contact with the tissues and allowed to clot in the test tube. How the defect in hemophilia fitted into the classical scheme of clotting remained an enigma. In the early days it was thought that thromboplastin was released from the breakdown of platelets and that in hemophilia the platelets failed to disrupt in a normal manner.

However, in a series of papers from the Thorndike Memorial Laboratory in Boston between 1936 and 1939, A. J. Patek, R. P. Stetson, F. H. L. Taylor, F. J. Pohle and E. L. Lozner clearly demonstrated that an euglobulin fraction could be prepared from normal platelet-free plasma which would correct the defect when added to hemophilic blood (Figure 7). This substance was called antihemophilic globulin (AHG) (now known as Factor VIII), and it was thought that the platelets and AHG united in some way to produce blood thromboplastin.

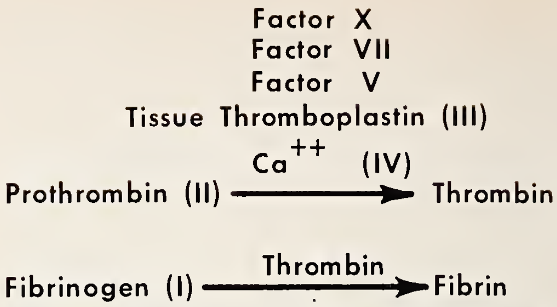
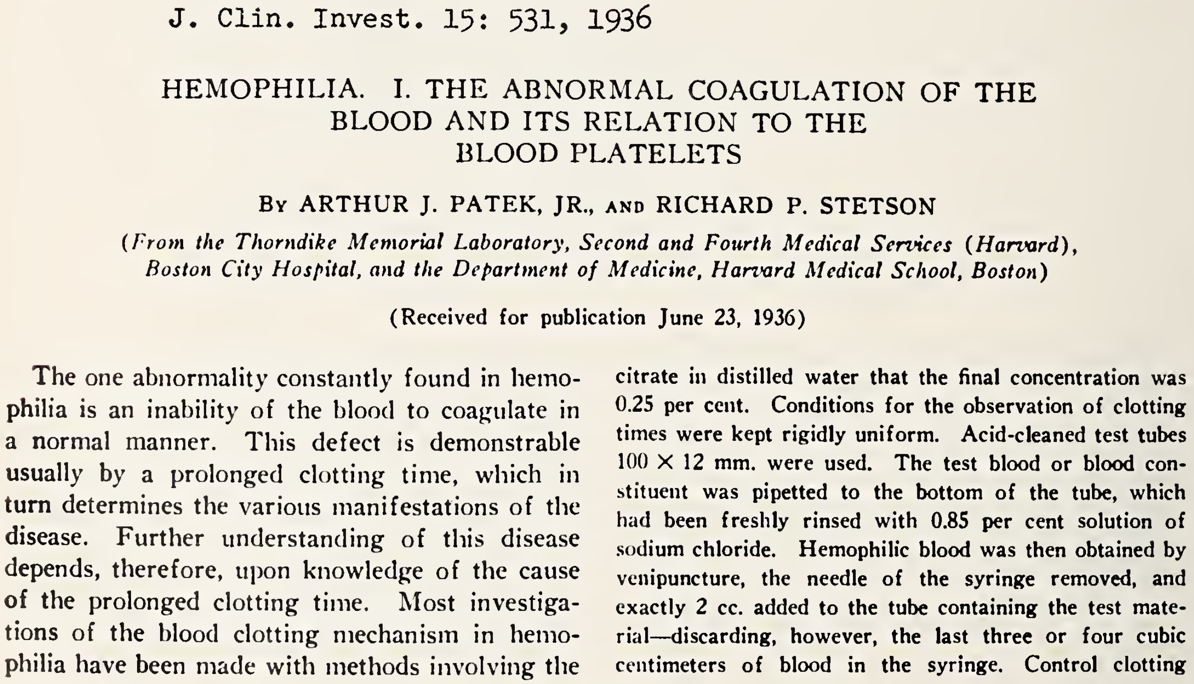


Figure 6.—Three factors, V, VII and X were found to be required to convert prothrombin to thrombin in the presence of an optimal amount of tissue thromboplastin and calcium.

In 1946, Professor E. J. Cohn and his coworkers at Harvard University published a method for the separation of plasma proteins by alcohol fractionation. It was soon found that AHG was present in Cohn's Fraction I.

In 1948, we were engaged in testing the AHG potency of Cohn Fraction I, which was supplied to us by the Cutter Laboratories in Berkeley. One of our hemophiliac patients was an 11-year-old boy named Kent Kincaid, an only child with no family history of bleeding, who had a decidedly prolonged clotting time and had been in hospital over a hundred times for major hemorrhagic episodes. He had impressed his pediatrician, Dr. George Bates, however, not only by the frequency



J. Clin. Invest. 15: 531, 1936

HEMOPHILIA. I. THE ABNORMAL COAGULATION OF THE BLOOD AND ITS RELATION TO THE BLOOD PLATELETS

By ARTHUR J. PATEK, JR., and RICHARD P. STETSON

(From the Thorndike Memorial Laboratory, Second and Fourth Medical Services (Harvard), Boston City Hospital, and the Department of Medicine, Harvard Medical School, Boston)

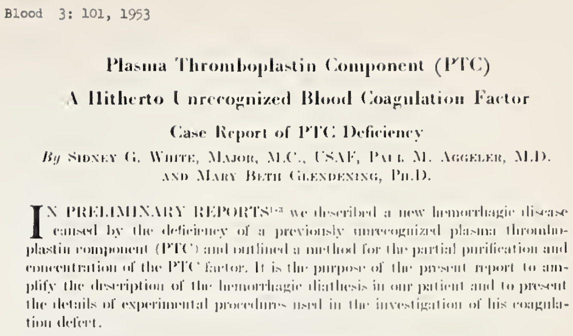
(Received for publication June 23, 1936)

The one abnormality constantly found in hemophilia is an inability of the blood to coagulate in a normal manner. This defect is demonstrable usually by a prolonged clotting time, which in turn determines the various manifestations of the disease. Further understanding of this disease depends, therefore, upon knowledge of the cause of the prolonged clotting time. Most investigations of the blood clotting mechanism in hemophilia have been made with methods involving the

citrate in distilled water that the final concentration was 0.25 per cent. Conditions for the observation of clotting times were kept rigidly uniform. Acid-cleaned test tubes 100 × 12 mm. were used. The test blood or blood constituent was pipetted to the bottom of the tube, which had been freshly rinsed with 0.85 per cent solution of sodium chloride. Hemophilic blood was then obtained by venipuncture, the needle of the syringe removed, and exactly 2 cc. added to the tube containing the test material—discarding, however, the last three or four cubic centimeters of blood in the syringe. Control clotting

Figure 7.—Title page from a paper by Patek which first described antihemophilic globulin (now called Factor VIII).

of his hemorrhages but also by the unusually good response he invariably showed to the transfusion of normal whole blood or plasma. Most of our Fraction I preparation showed good antihemophilic potency when tested on one or another of our hemophilic patients. However, an occasional one was impotent. We did not attach any particular significance to the observation when the first preparation we tried on Kent was incapable of correcting his defect. Some months later we tested a new lot of Fraction I on Kent; again we found that the preparation had no power to correct his coagulation defect, but this time we found that the same lot was potent when tested with the blood of other hemophiliac patients. It was then but a simple matter to show, by direct plasma-mixing tests, that Kent's blood and that of other hemophiliacs were mutually corrective. We knew then that Kent did not have hemophilia and subsequently we were able to show that he had no other defect in clotting time at that time.



Blood 3: 101, 1953

Plasma Thromboplastin Component (PTC)

A Hitherto Unrecognized Blood Coagulation Factor

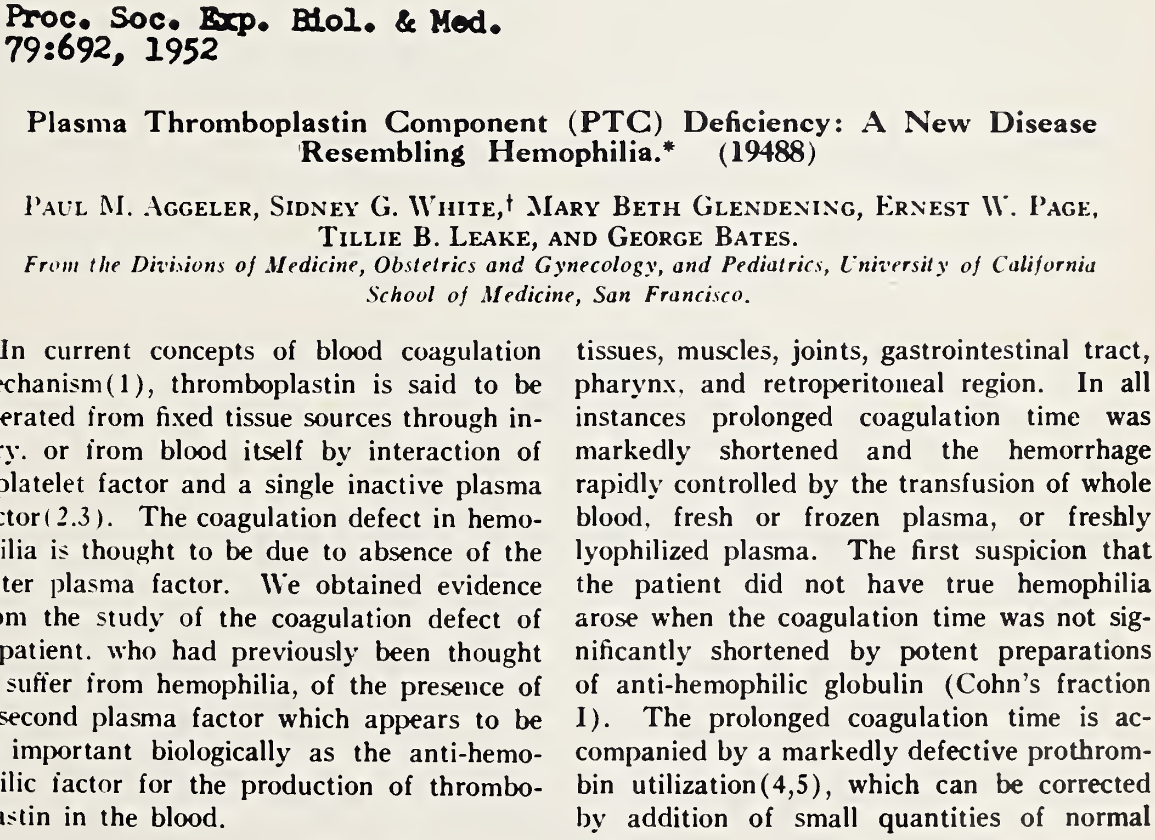
Case Report of PTC Deficiency

By Sidney G. White, Major, M.C., USAF, Paul M. Aggeler, M.D.
and Mary Beth Glendening, Ph.D.

In preliminary reports[1-3] we described a new hemorrhagic disease caused by the deficiency of a previously unrecognized plasma thromboplastin component (PTC) and outlined a method for the partial purification and concentration of the PTC factor. It is the purpose of the present report to amplify the description of the hemorrhagic diathesis in our patient and to present the details of experimental procedures used in the investigation of his coagulation defect.

Figure 9.—Further studies on PTC deficiency.

In 1951, Dr. Sidney White joined us as a research fellow, and in May of 1952 we published the first report showing the essential features of the clotting feature missing in Kent's case (Figure 8). You will note that this was a most cooperative venture. Dr. Bates, of the Department of Pediatrics, I have mentioned already. Drs. Ernest Page and Mary-Beth Glendening of the Department of Obstetrics and Gynecology were involved at that time in their pioneering studies on the phenomenon of disseminated intravascular coagulation in patients with placentae previa, and we were able to enlist their interest in our strange case. I would also like to express my particular appre-



Proc. Soc. Exp. Biol. & Med.
79:692, 1952

Plasma Thromboplastin Component (PTC) Deficiency: A New Disease Resembling Hemophilia.* (19488)

Paul M. Aggeler, Sidney G. White,† Mary Beth Glendening, Ernest W. Page, Tillie B. Leake, and George Bates.

From the Divisions of Medicine, Obstetrics and Gynecology, and Pediatrics, University of California School of Medicine, San Francisco.

In current concepts of blood coagulation mechanism(1), thromboplastin is said to be liberated from fixed tissue sources through injury, or from blood itself by interaction of a platelet factor and a single inactive plasma factor(2,3). The coagulation defect in hemophilia is thought to be due to absence of the latter plasma factor. We obtained evidence from the study of the coagulation defect of a patient, who had previously been thought to suffer from hemophilia, of the presence of a second plasma factor which appears to be as important biologically as the anti-hemophilic factor for the production of thromboplastin in the blood.

tissues, muscles, joints, gastrointestinal tract, pharynx, and retroperitoneal region. In all instances prolonged coagulation time was markedly shortened and the hemorrhage rapidly controlled by the transfusion of whole blood, fresh or frozen plasma, or freshly lyophilized plasma. The first suspicion that the patient did not have true hemophilia arose when the coagulation time was not significantly shortened by potent preparations of anti-hemophilic globulin (Cohn's fraction I). The prolonged coagulation time is accompanied by a markedly defective prothrombin utilization(4,5), which can be corrected by addition of small quantities of normal

Figure 8.—First article by the author and his coworkers on PTC, a new clotting factor (now called Factor IX).

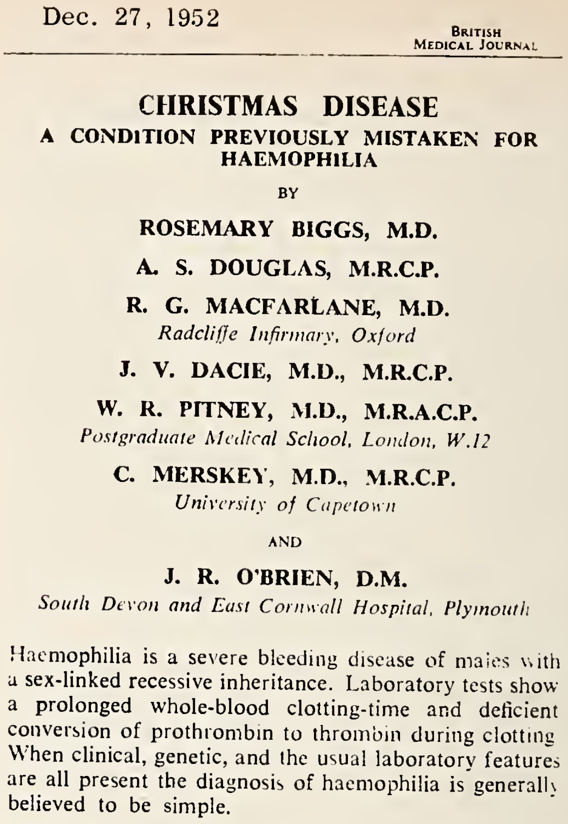
ciation of Mrs. Tillie Leake,* who was my very capable blood clotting research technician at that time.

In February of the following year we published a more detailed account of these investigations (Figure 9). We named the factor *plasma thromboplastin component,* or PTC. It is now known as Factor IX. In contrast to AHF it could be readily adsorbed onto barium sulfate, was present in the serum and was found in Cohn's Fraction IV rather than Fraction I, and in the fraction of plasma precipitated between 40 per cent and 50 per cent saturation with ammonium sulfate rather than the 0 to 25 per cent saturated fraction. Unlike AHG, this factor was quite stable on storage. Plasma from outdated blood bank blood could be used for treatment, rather than the very fresh or freshly frozen plasma required in hemophilia. The beneficial effect of transfusion on the clotting time persisted for a much longer time than in AHG deficiency.

In the 27 December 1952 issue of the *British Medical Journal,* there appeared a paper appropriately entitled "Christmas Disease: A Condition Previously Mistaken for Hemophilia," by Dr. Rosemary Biggs and her associates (Figure 10). This paper described seven patients with the same condition we had observed in Kent, and in addi-

---

*Deceased.



Dec. 27, 1952 BRITISH MEDICAL JOURNAL

CHRISTMAS DISEASE

A CONDITION PREVIOUSLY MISTAKEN FOR HAEMOPHILIA

BY

ROSEMARY BIGGS, M.D.

A. S. DOUGLAS, M.R.C.P.

R. G. MACFARLANE, M.D.

Radcliffe Infirmary, Oxford

J. V. DACIE, M.D., M.R.C.P.

W. R. PITNEY, M.D., M.R.A.C.P.

Postgraduate Medical School, London, W.12

C. MERSKEY, M.D., M.R.C.P.

University of Capetown

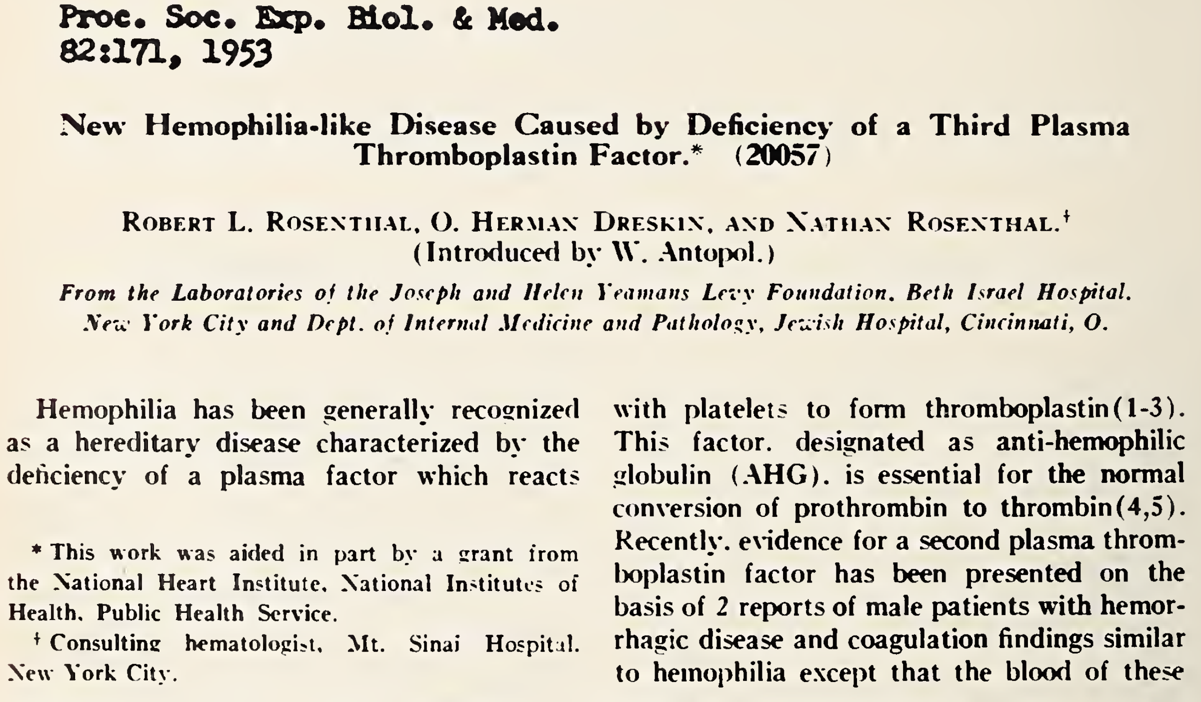
AND

J. R. O'BRIEN, D.M.

South Devon and East Cornwall Hospital, Plymouth

Haemophilia is a severe bleeding disease of males with a sex-linked recessive inheritance. Laboratory tests show a prolonged whole-blood clotting-time and deficient conversion of prothrombin to thrombin during clotting. When clinical, genetic, and the usual laboratory features are all present the diagnosis of haemophilia is generally believed to be simple.

Figure 10.—Title page of article by Dr. Biggs and her coworkers describing "Christmas" factor (later shown to be same as PTC).



Proc. Soc. Exp. Biol. & Med. 82:171, 1953

New Hemophilia-like Disease Caused by Deficiency of a Third Plasma Thromboplastin Factor.* (20057)

ROBERT L. ROSENTHAL, O. HERMAN DRESKIN, AND NATHAN ROSENTHAL.†
(Introduced by W. Antopol.)

From the Laboratories of the Joseph and Helen Yeamans Levy Foundation, Beth Israel Hospital, New York City and Dept. of Internal Medicine and Pathology, Jewish Hospital, Cincinnati, O.

Hemophilia has been generally recognized as a hereditary disease characterized by the deficiency of a plasma factor which reacts with platelets to form thromboplastin(1-3). This factor, designated as anti-hemophilic globulin (AHG), is essential for the normal conversion of prothrombin to thrombin(4,5). Recently, evidence for a second plasma thromboplastin factor has been presented on the basis of 2 reports of male patients with hemorrhagic disease and coagulation findings similar to hemophilia except that the blood of these

* This work was aided in part by a grant from the National Heart Institute, National Institutes of Health, Public Health Service.

† Consulting hematologist, Mt. Sinai Hospital, New York City.

Figure 11.—Title page of article by Rosenthal et al. describing plasma thromboplastin antecedent (now called Factor XI).

tion showed that it was transmitted as an X-linked recessive trait, exactly as in classical hemophilia. Their first patient was a five-year-old boy whose family name was Christmas.

A short time later, Dr. Robert Rosenthal of New York and his associates described another defect in clotting in three patients in one family, a 50-year-old man and his two nieces, who had shown since childhood moderate evidences of a bleeding tendency, marked chiefly by bleeding after tooth extractions (Figure 11). Like AHF and PTC, this factor seemed to be required for the production of blood thromboplastin but was not necessary for clotting with tissue extracts. They called it *plasma thromboplastin antecedent,* or PTA. It is now known as Factor XI. Like Factor IX this factor was present in serum but unlike Factor IX it was only partially adsorbed on barium sulfate. It also seemed to be involved in the activation of the clotting mechanism on exposure to glass and similar surfaces.

Finally in 1955 Oscar Ratnoff and Joan Colopy at Western Reserve University described a new defect in clotting in a 37-year-old railroad brakeman named Hageman and in two housewives aged 46 and 50 years (Figure 12). Curiously, although the clotting time was decidedly prolonged, none of these patients or the many that have been described since then with the same condition have suffered from abnormal bleeding. Why these people do not bleed is one of the remaining enigmas in the blood clotting field. This factor is now known as Factor XII. Its properties were very similar to those of Factor XI, and it could be distinguished clearly from Factor XI only by direct plasma-mixing tests.

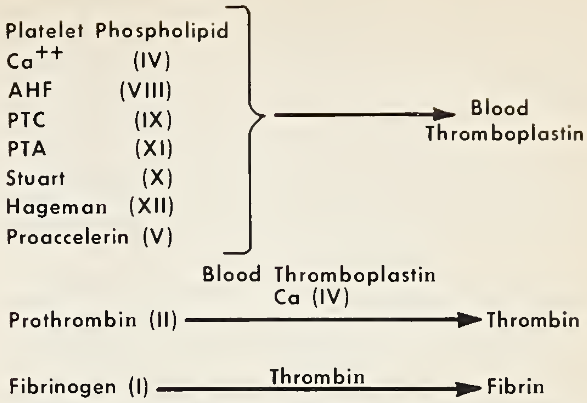
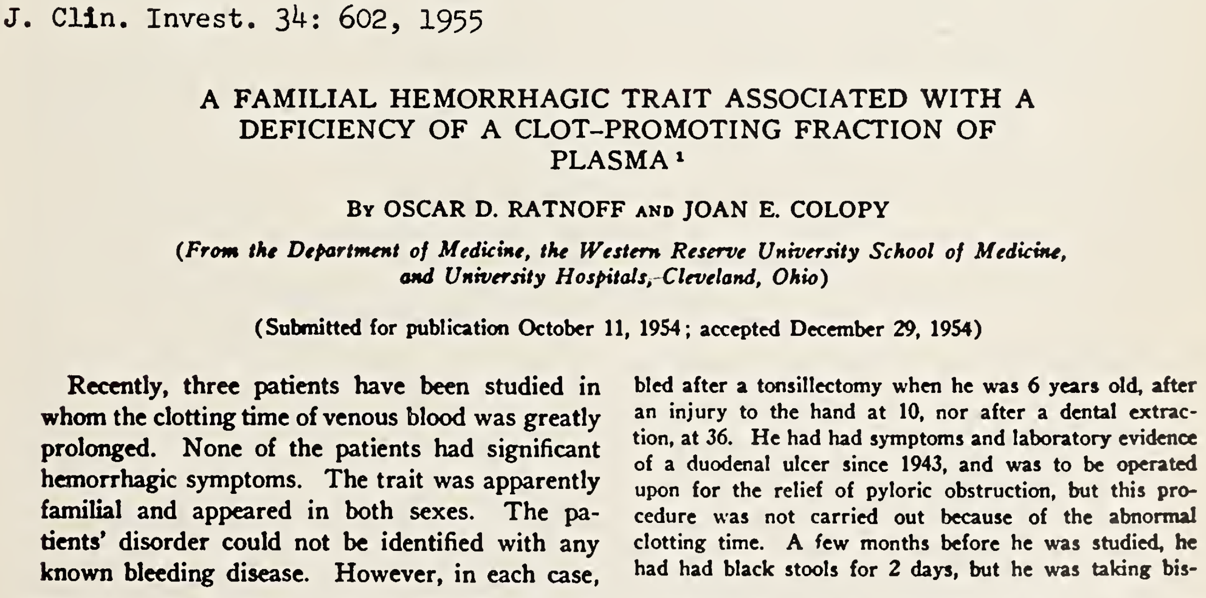


Figure 13.—By 1957 a revision of the classical theory of clotting included all the above.

By 1957 a revision of the classical theory of clotting would have to include all of the factors shown in Figure 13. However, no one ever suggested that all of these new factors united together at once to form the prothrombin converting principle. From the beginning, attempts had been made to study their manner of interaction. At first it was proposed that a series of products might be formed, as shown in Figure 14.

However, the concept of even three or four fac-



J. Clin. Invest. 34: 602, 1955

A FAMILIAL HEMORRHAGIC TRAIT ASSOCIATED WITH A DEFICIENCY OF A CLOT-PROMOTING FRACTION OF PLASMA[1]

By OSCAR D. RATNOFF and JOAN E. COLOPY

(From the Department of Medicine, the Western Reserve University School of Medicine, and University Hospitals, Cleveland, Ohio)

(Submitted for publication October 11, 1954; accepted December 29, 1954)

Recently, three patients have been studied in whom the clotting time of venous blood was greatly prolonged. None of the patients had significant hemorrhagic symptoms. The trait was apparently familial and appeared in both sexes. The patients' disorder could not be identified with any known bleeding disease. However, in each case,

bled after a tonsillectomy when he was 6 years old, after an injury to the hand at 10, nor after a dental extraction, at 36. He had had symptoms and laboratory evidence of a duodenal ulcer since 1943, and was to be operated upon for the relief of pyloric obstruction, but this procedure was not carried out because of the abnormal clotting time. A few months before he was studied, he had had black stools for 2 days, but he was taking bis-

Figure 12.—Title page of article by Ratnoff and Colopy describing a new factor in blood clotting (now called Factor XII).

tors uniting simultaneously in such a manner as this was repugnant to most physiologists, and work during the past several years has centered on attempts to reduce the blood clotting process into a series of two-factor reactions. Almost all of the experimental data now available are consistent with the theory expressed by Roger Macfarlane, and by E. W. Davie and Oscar Ratnoff in 1964, as shown in Figure 15.

These investigators have proposed a simple waterfall or cascade sequence to explain the function of the various factors in blood clotting. According to this theory, clotting is initiated by surface contact activation of Factor XII, possibly by unfolding the molecule. Then each factor in turn is converted to a form that has enzymatic activity. The activation occurs in a step-wise sequence with each newly formed enzyme reacting with its specific substrate, converting it to an active enzyme until the final substrate, fibrinogen, is reached. There is still some uncertainty regarding the steps involving the interaction of Factors X, V and phospholipid. Ratnoff believes that the final prothrombin converting factor is activated Factor V, as indicated in the diagram. Other investigators feel that activated Factor X, Factor V and phospholipid combine together to form the final prothrombin converting factor.

The key role of Factor X in the clotting mechanism has only recently become apparent. This factor can be activated either by activated Factor VIII in the intrinsic clotting system or by Factor VII and tissue thromboplastin in the extrinsic system. It can also be activated by Russell's viper venom, trypsin, papain and other enzymes.

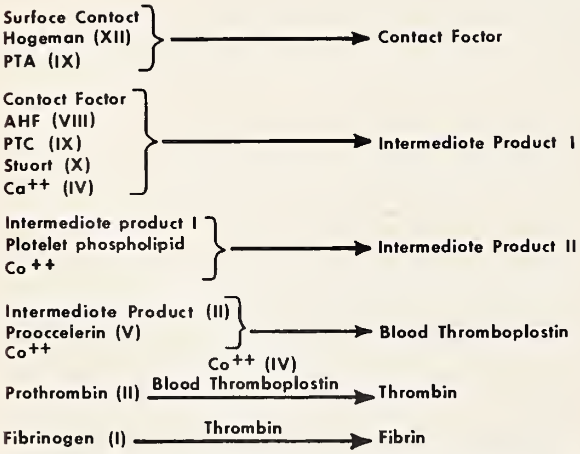
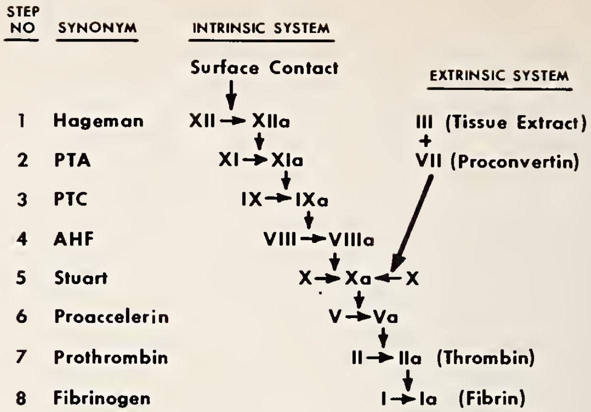

Surface Contact
Hageman (XII)
PTA (IX)
Contact Factor
Contact Factor
AHF (VIII)
PTC (IX)
Stuart (X)
Ca++ (IV)
Intermediate Product I
Intermediate product I
Platelet phospholipid
Ca ++
Intermediate Product II
Intermediate Product (II)
Proaccelerin (V)
Ca++
Blood Thromboplastin
Ca++ (IV)
Prothrombin (II)
Blood Thromboplastin
Thrombin
Fibrinogen (I)
Thrombin
Fibrin

Figure 14.—One of the theories of the mechanism of coagulation popular in the early 1960's.


STEP NO
SYNONYM
INTRINSIC SYSTEM
Surface Contact
EXTRINSIC SYSTEM
1 Hageman
XII → XIIa
III (Tissue Extract)
+
VII (Proconvertin)
2 PTA
XI → XIa
3 PTC
IX → IXa
4 AHF
VIII → VIIIa
5 Stuart
X → Xa ← X
6 Proaccelerin
V → Va
7 Prothrombin
II → IIa (Thrombin)
8 Fibrinogen
I → Ia (Fibrin)

Ca++ (Factor IV) required for steps 3-6.
Platelet phospholipid required for step 6 and possibly step 4.
Thrombin accelerates and perhaps is required for steps 4 and 6.

Figure 15.—The "cascade" or "false waterfall" theory of the mechanism of blood coagulation as described by Macfarlane and by Davie and Ratnoff in 1964.

Recent work also strongly suggests that a small amount of thrombin is required to activate Factors V and VIII. How this first tiny bit of thrombin is generated is not known.

The role of the natural inhibitors of blood clotting is not so clear. It is probable that there are two lines of defense against intravascular clotting. One consists of the reticuloendothelial system which appears capable of removing activated intermediates in the clotting mechanism. The other is a series of natural circulating inhibitors of the activated factors such as the well known antithrombin III.

Use of Drugs to Inhibit Clotting

There is much more one could say about other recent developments in the blood clotting field. However, I have chosen to use the remaining time to discuss some aspects of the use of drugs to inhibit blood clotting.

A list of the oral anticoagulant drugs most commonly used here and abroad is shown in Figure 16. Since the isolation of dicumarol from spoiled sweet clover by Karl Paul Link and his students in 1939, a large number of coumarin and indanedione anticoagulant compounds have been synthesized. Since 1942, many thousands of patients have received treatment with these compounds for a variety of thromboembolic disorders. The two drugs most commonly used in the United States are warfarin and dicumarol.

The pharmacodynamics of this group of drugs varies widely, but they all influence the clotting mechanism in the same manner; they all interfere with the production of clotting Factors II, VII, IX and X in the liver. Their effects can all be neutralized with large doses of vitamin K. The physiologic response to these drugs can be measured conveniently by Quick's original "prothrombin" test which reflects the combined activity of Factors II, V, VII and X. These latter factors are sometimes referred to as the "prothrombin complex."

There are many difficulties associated with the use of these drugs. Several years ago Dr. Robert O'Reilly and I embarked on a study of some of these problems. So far, we have limited our investigations to dicumarol and warfarin. We have attempted to differentiate between conditions which alter the clotting factor response by influencing the absorption, distribution or elimination of the drugs and those which alter the response by directly affecting the clotting synthesizing mechanism in the liver.

Theoretically, incomplete absorption of these drugs might occur in patients with vomiting, diarrhea or malabsorption syndromes. We have obtained some evidence that barbital compounds may also cause decreased absorption.

Previous investigators showed that the disappearance rate of dicumarol from the plasma is increased by the concomitant administration of barbital compounds and suggested that this resulted from "enzyme stimulation" in the liver. The ability of one drug to reduce the pharmacologic activity of another by stimulating its metabolic inactivation is well known. Drugs capable of causing this effect act by increasing the amount of drug metabolizing enzymes in liver microsomes.

The administration of large doses of barbitals to rats increases the amount of coumarin drug metabolizing enzymes in their liver homogenates. There is no direct proof, however, that a similar mechanism can be activated by barbitals in man.

We have performed in human volunteers a number of experiments which suggest to us that absorption of dicumarol is impaired by barbital administration. We first determined the pattern of response of an oral dose of 600 mg of dicumarol. The subjects were then given 400 mg of heptabarbital on each of three evenings; on the morning of the fourth day the same dose of heptabarbital was given, followed by 600 mg of dicumarol an hour later. The initial rate of absorption of dicu-

GENERIC NAME	CHEMICAL NAME	TRADE NAME
	COUMARIN ANTICOAGULANT DRUGS	
Sodium Warfarin	Sodium salt of 3-(α-acetonylbenzyl)-4-hydroxycoumarin	Coumadin (Endo) Panwarfin (Abbott)
Dicoumarol **Bishydroxycoumarin**	3,3'-methylenebis [4-hydroxycoumarin])	Dicumarol (Abbott) Dicumarol (Lilly)
Phenprocoumon	3-(1'-phenylpropyl)-4-hydroxycoumarin	Liquomar (Organon) Marcoumar (Roche)
Nicoumalone **Acenocoumarin**	3-(α-acetonyl-4-nitrobenzyl)-4-hydroxycoumarin	Sintrom (Geigy)
Cyclcoumarol	3,4-(2' methyl-2-methoxy-4'-phenyl)-dihydropyranocoumarin	Cumopyran (Abbott)
Ethyl Biscoumacetate	3,3'-carboxymethylene bis -(4-hydroxycoumarin) ethyl ester	Tromexan (Geigy)
	INDANEDIONE DERIVATIVES	
Phenindione	2-phenylindane-1:3-dione	Dindevan (Evans) Hedulin (Walker) Danilone (Schieffelin)
Diphenadione	2-(diphenylacetyl)-indate-1:3 dione	Dipaxin (Upjohn)
Anisindione	2-p-anisyl-indane-1:3 dione	Miradon (Schering)

Figure 16.—List of the most commonly used oral anticoagulant drugs.

marol appeared identical in the two experiments, but when heptabarbital was given the duration of absorption was much shorter and an increased amount of dicumarol was found in the stool.

A typical experiment is shown in Figure 17. The results without heptabarbital are shown by the open circles and cross-hatched bars; the peak drug level was 52 mg per liter of plasma and the biologic half-life of the drug was 62 hours; 24 per cent of the dose was excreted in the stool, 9 per cent within the first 24 hours. The results obtained when the subject was pretreated with heptabarbital are shown by the solid circles and bars; the peak blood level was only 40 mg per liter of plasma and the biologic half-life 30 hours; 50 per cent of the dose was excreted in the stool, 48 per cent of it within the first 24 hours. The prothrombin time response was significantly less when the subject was pretreated with heptabarbital.

Now it so happens that dicumarol is one of the few drugs known whose disappearance rate is directly proportional to the dose administered. The increased rate of metabolism of dicumarol when the barbital was administered could have been caused, therefore, by the smaller amount of dicumarol that was actually absorbed. It is not necessary to invoke the concept of enzyme stimulation to account for it.

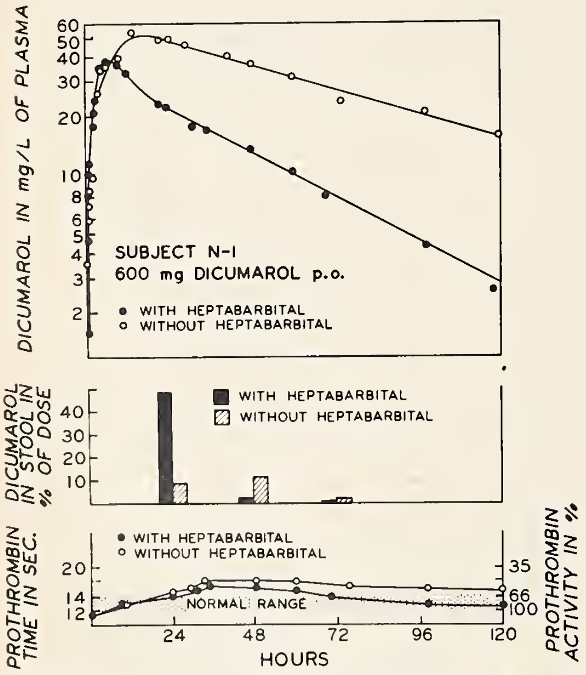


Figure 17.—An experiment indicating the effect of barbital on the metabolism of a single large dose of dicumarol. Source: Aggeler, P. M., and O'Reilly, R. A.: Pharmacologic basis of oral anticoagulant therapy, Thromb. Diath. Haemorrh., 21:227-256, 1966.

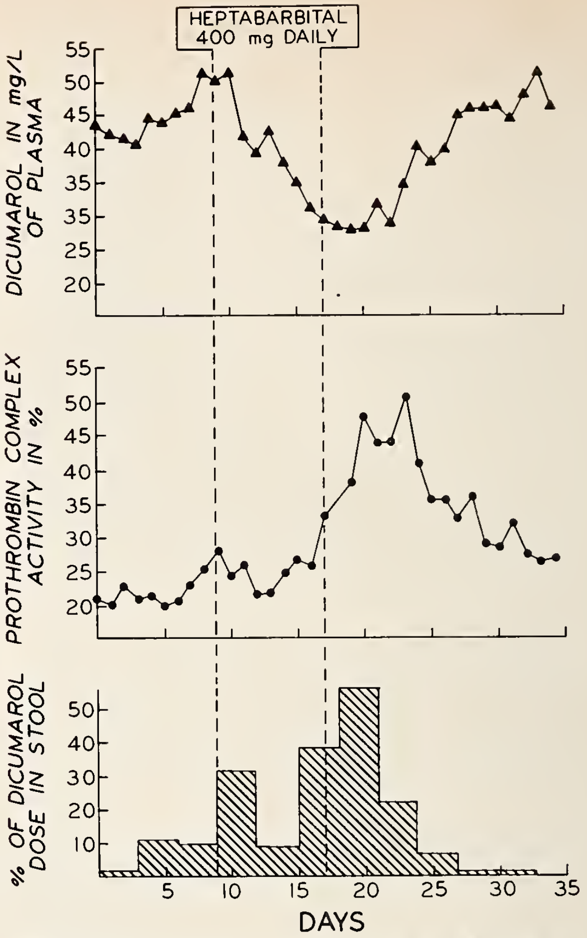


Figure 18.—An experiment indicating the effect of barbital on the metabolism of dicumarol in a chronically anticoagulated subject. Source: Aggeler, P. M., and O'Reilly, R. A.: Pharmacologic basis of oral anticoagulant therapy, Thromb. Diath. Haemorrh., 21:227-256, 1966.

The course of events when the same subject, now stabilized on a daily maintenance dose of 60 mg of dicumarol, was given heptabarbital is shown in Figure 18. Four hundred milligrams of heptabarbital was taken each evening, one hour before the daily dose of dicumarol, on eight successive days. The dicumarol concentration of blood and stool specimens collected each morning was measured, and the prothrombin complex activity of the plasma was determined. Allowing a period of 24 to 72 hours for passage of dicumarol through the gut, the sequence of events appears to be: (1) A decrease in absorption of dicumarol, (2) an increase in its excretion in the stool, (3) a lowered concentration of drug in the blood, and (4) a decreased prothrombinopenic response in the plasma. We do not yet have sufficient data to determine whether failure of absorption accounts for the entire effect of barbital compounds on dicumarol metabolism or whether the same effect can be demonstrated with other oral anticoagulant drugs.

The disposition of these drugs has been studied directly in a variety of animal species; in general, no unusual pattern has been found, and no organ has shown higher concentration than plasma. In most studies the concentration of anticoagulant drugs has been higher in liver and kidneys than in other organs. Anticoagulant drug levels in liver tissue and in plasma normally decline at approximately the same rates. We have found that the volume of distribution of warfarin and of dicumarol when given in small doses is 12.6 per cent of body weight—about the same as for serum albumin.

These drugs are extensively bound to serum albumin. They are not present in appreciable amounts in red blood cells or cerebrospinal fluid in man. Transplacental transfer and the passage of unchanged drug into breast milk occurs. Neither dicumarol nor warfarin is excreted unchanged in the urine. Normally up to 30 per cent of the dose of dicumarol may be found in the stool. We have never found unchanged warfarin in the stool.

We know of no condition which can alter distribution of these drugs in the body. However, we are investigating the possibility that the amount of plasma protein-binding of these drugs may be changed by the administration of other compounds, and that this may lead to an altered prothrombinopenic response.

There is a good correlation between the disappearance rate of warfarin and the maximum prothrombin response in normal subjects. In a group of 24 normal subjects the mean half-life of warfarin was 44 hours and the standard deviation 9.75 hours.

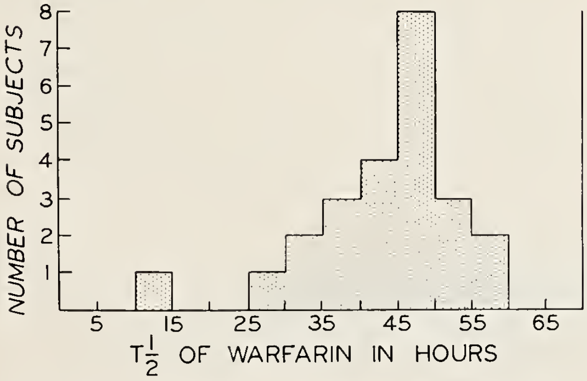


Figure 19.—Frequency distribution of biologic half-life of warfarin in normal subjects. Source: Aggeler, P. M., and O'Reilly, R. A.: Pharmacologic basis of oral anticoagulant therapy, Thromb. Diath. Haemorrh., 21:227-256, 1966.

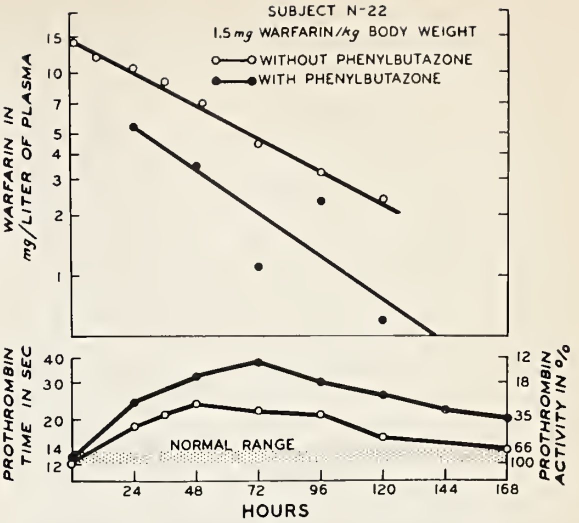


Figure 20.—Potentiation of the prothrombinopenic effect of warfarin by phenylbutazone.

The frequency distribution of the biologic half-life of warfarin in 24 normal subjects is shown in Figure 19. It is skewed somewhat toward the more rapid disappearance rates. We have recently investigated a patient who required 40 mg of warfarin per day compared with the average daily dose of 7.5 mg. The biologic half-life of the drug in his case was only five and a half hours. It seems likely that some instances of extraordinary sensitivity or resistance to the prothrombinopenic effects of these drugs may be caused by extreme variations in their rate of catabolism. Such variations could be on either a genetic or an environmental basis.

Few diseases are known to affect the elimination rate of these drugs. Although impaired liver function may profoundly increase the physiologic response, the disappearance rate of the drugs themselves is normal in patients with liver disease. The disappearance rate is also normal in patients with severe azotemia. We have been unable to detect unchanged warfarin in the urine even when massive proteinuria was present.

A pronounced increase in the response to coumarin anticoagulant drugs occurs when pyrazolone compounds (phenylbutazone and oxyphenylbutazone) are administered concomitantly. Murray Weiner and his associates recently stated that oxyphenylbutazone slowed the disappearance rate of dicumarol in some subjects and that this was responsible for the enhanced prothrombinopenic response. They suggested that this might result from inhibition by the pyrazolone drugs of enzymes in the liver which metabolize the coumarin anticoagulants.

We have regularly found decided potentiation of the prothrombinopenic effect of warfarin and dicumarol by phenylbutazone and oxyphenylbutazone, but, in contrast to Weiner, we have found an increase rather than a decrease in the rate of disappearance of the coumarin drugs from the plasma. The results of one such experiment are shown in Figure 20. A normal subject was given 200 mg of phenylbutazone thrice daily for 11 days before and during the entire course of observation after the oral administration of a standard dose of 1.5 mg of warfarin per kilogram of body weight. Although decided potentiation of the prothrombinopenic effect occurred in the presence of phenylbutazone, the rate of elimination of warfarin from the plasma was speeded rather than slowed. These results are shown in Figure 20 by the line drawn through the open circles. The control experiment without phenylbutazone is shown by the line drawn through the closed circles. We are investigating the possibility that phenylbutazone potentiates the action of warfarin by displacing warfarin from its binding to plasma albumin *in vivo,* making increased quantities of free drug available for excretion, biotransformation and biologic action at specific drug receptor sites in the liver.

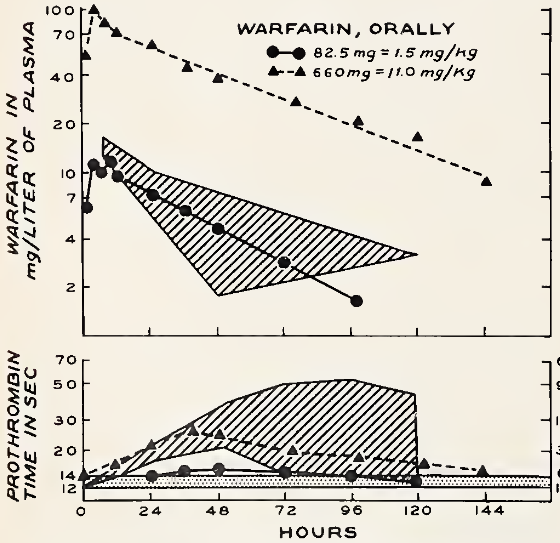


Figure 21.—An experiment demonstrating lack of response to a standard dose of warfarin, but the satisfactory response to a very large dose in a patient with genetically-determined resistance to the action of the drug. Source: O'Reilly, R. A., and Aggeler, P. M.: Coumarin anticoagulant drugs: hereditary resistance in man, Fed. Proc. 24:1266-1273, 1965.

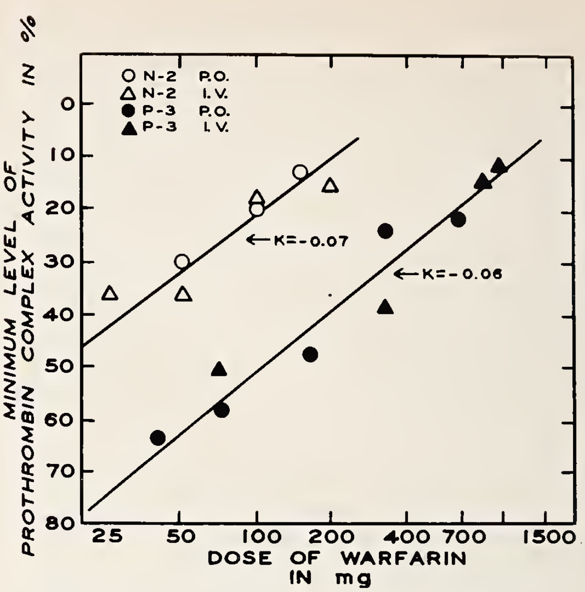


Figure 22.—A comparison of prothrombinopenic responses to increased doses of warfarin in a normal subject and a patient with hereditary resistance to the drug. P.O.=Oral. I.V.=Intravenous. Source: O'Reilly, R. A., Aggeler, P. M. Hoag, M. S., Leong, L. S., and Kropatkin, M. L.: Hereditary transmission of exceptional resistance to coumarin anticoagulant drugs: first reported Kindred, New Engl. J. Med., 271:809-815, 1964.

Other drugs have been reported to cause an increased prothrombinopenic response to the oral anticoagulant drugs and doubtless new ones will be discovered. Those mentioned in the literature include phenylramidol HCl, clofibrate, chlorpromazine and the C-17 alkylated anabolic steroids. In most instances the mechanism of this potentiation has not been studied, but in the case of the anabolic steroids Kalevi Pyörälä reported that it was not associated with any alteration in the metabolism of the coumarin drugs.

Alterations in the prothrombin response which are not dependent upon variations in the metabolism of the drugs are well known. These include the increased sensitivity of patients with impaired liver function or diseases causing vitamin K deficiency. In these conditions the increased response is caused by the already impaired production of clotting factors in the liver. Extreme variations in the dietary content of vitamin K can also alter the response to the oral anticoagulant drugs. The response to these drugs is also increased in febrile states and in hyperthyroidism and is decreased in hypothyroidism, presumably because of corresponding changes in the turnover rates of the relevant clotting factor proteins in these conditions.

The ability of the clotting factor synthesizing mechanism in the liver to respond to oral anticoagulant drugs is also under genetic control. Several years ago we discovered a family in whom extraordinary resistance to the coumarin drugs was on this basis. The propositus was a 73-year-old oil prospector named Harry Martin, who had sustained an acute myocardial infarction and was found to be resistant to ordinary doses of warfarin.

He was given a single standard oral dose of 1.5 mg of warfarin per kg of body weight (Figure 21). The concentration of warfarin in plasma and the prothrombin activity were determined serially and compared with the results obtained with the same dose of warfarin in a group of 23 normal subjects (shown in the cross-hatched area). The concentrations of drug in the plasma of the patient (shown by the line connecting the solid circles in the upper graph) were well within the normal range at all times tested. However, this amount of warfarin had only a slight effect on the prothrombin activity in the patient (shown in the lower figure). It was necessary to give the patient a dose of 12 mg of warfarin per kilogram of body weight before the prothrombin response (shown by the dotted line in the lower figure) could be brought within the range of values observed when normal subjects were given only 1.5 of the drug per kilogram of body weight. When this was done, the concentration of warfarin in the patient's plasma rose proportionately (shown by the dotted line in the upper figure).

The patient was tested with a series of orally and intravenously administered doses of warfarin ranging from 40 to 1,000 mg (Figure 22). The results in the patient are shown as open circles and triangles and those in a normal subject as solid circles and triangles. In both, the prothrombin responses were proportional to the size of the dose administered, but it required a much larger dose in the patient than in the normal subject to produce the same effect.

During long-term treatment with warfarin over a period of several years it was found that the patient required 145 mg daily to produce a satisfactory prolongation of the prothrombin time (Figure 23). The dose is shown in the lower graph. The plasma drug levels are in the middle graph

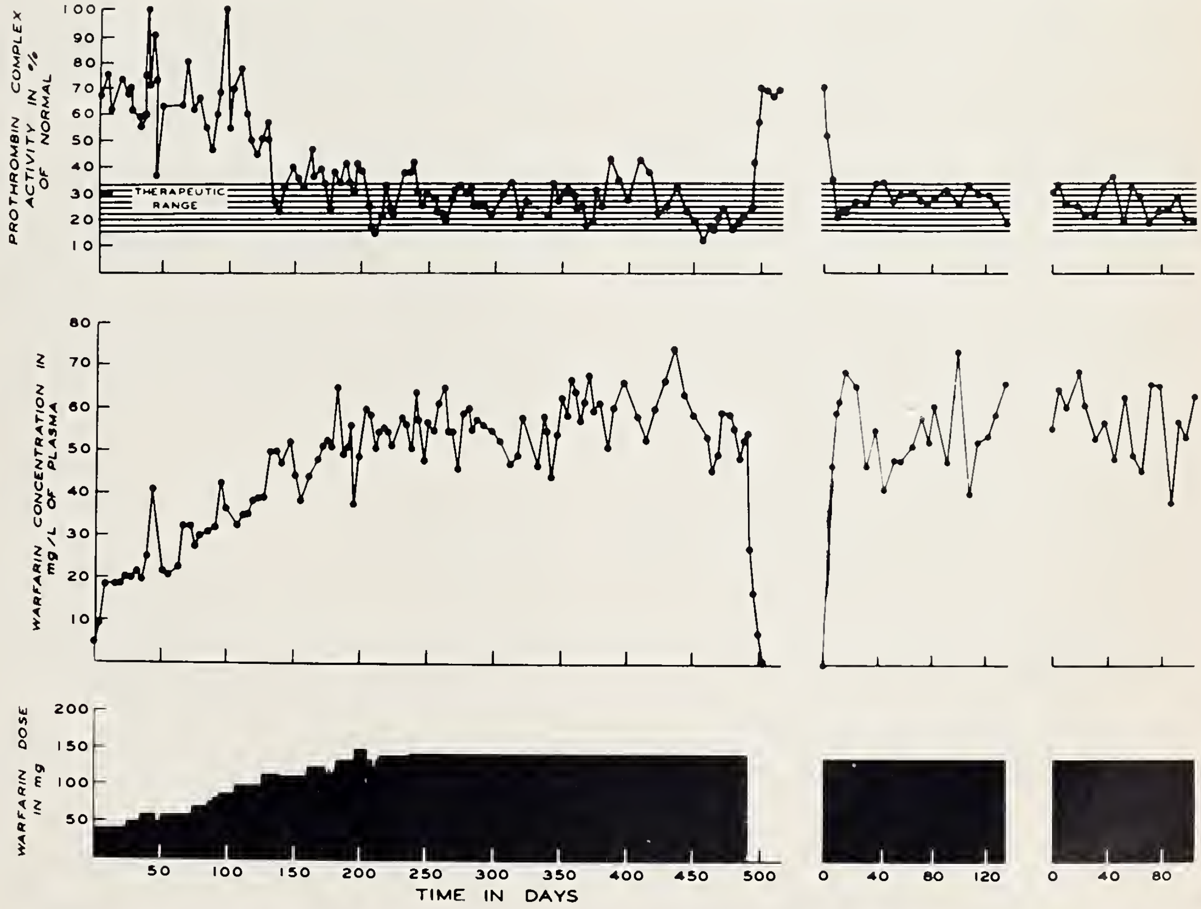


Figure 23.—Plasma concentration of drug and prothrombin response in a patient with hereditary resistance to coumarin drugs during long-term treatment with warfarin. Source: O'Reilly, R. A., and Aggeler, P. M.: Coumarin anticoagulant drugs: Hereditary resistance in man, Fed. Proc., 24:1266-1273, 1965.

and the prothrombin response in the upper graph. Both the dose of warfarin and the average plasma drug concentration (55 mg per liter of plasma) were about 20 times those of the average patient receiving long-term treatment with this drug.

The responsiveness of other members of the family was determined and seven of the eight members tested were found to be resistant. The pedigree of the family showing the incidence of resistance is given in Figure 24.

We have postulated that a pair of allelic genes control the structure or amount of the substances involved in the synthesis of the clotting factors dependent on vitamin K and that resistance to these drugs is transmitted by a dominant allele. Whether the gene is located on an X or autosomal chromosome cannot be discerned from the available data. In terms of the operon theory of F. Jacob and J. Monod, the most likely hypothesis is that in this family an abnormal regulator gene produced an altered repressor substance which has a decreased affinity for the coumarin anticoagulant drugs, or an increased affinity for vitamin K or both.

The findings in this family are of more than passing interest, as they document the first instance reported in the literature of genetically determined resistance not only to a coumarin but to any drug in man.

Finally, I would like to speak for a moment about a curious hemorrhagic disorder resulting from the surreptitious ingestion of these drugs. The data shown in Figure 25 is from the case of a 23-year-old nurse admitted to this hospital in 1962 because of recent onset of generalized purpura, epistaxis and gross hematuria. Prothrombin complex activity was reduced to 8 per cent. A detailed analysis of plasma clotting factors showed a pattern suggesting vitamin K deficiency or the ingestion of a coumarin drug. Repeated large doses of vitamin K_1 were required before the bleeding stopped and prothrombin complex activity returned to normal. At the time of admission the patient's plasma contained a very large amount of dicumarol (110 mg per liter). This and subsequent dicumarol levels are also shown in Figure 25. On a number of occasions during her hospital stay, the patient denied ingestion of the drug, but six months later she wrote that she was now under psychiatric care, that she had been in a severe depression and that she had taken 5 gm of dicumarol over a two-week period before entering the hospital.

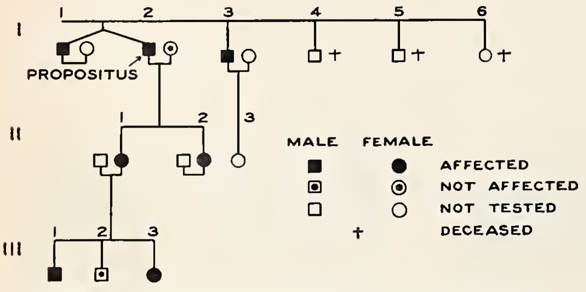


Figure 24.—Pedigree of a family showing incidence of resistance to coumarin drugs. Source: O'Reilly, R. A., and Aggeler, P. M.: Coumarin anticoagulant drugs: Hereditary resistance in man, Fed. Proc., 24:1266-1273, 1965.

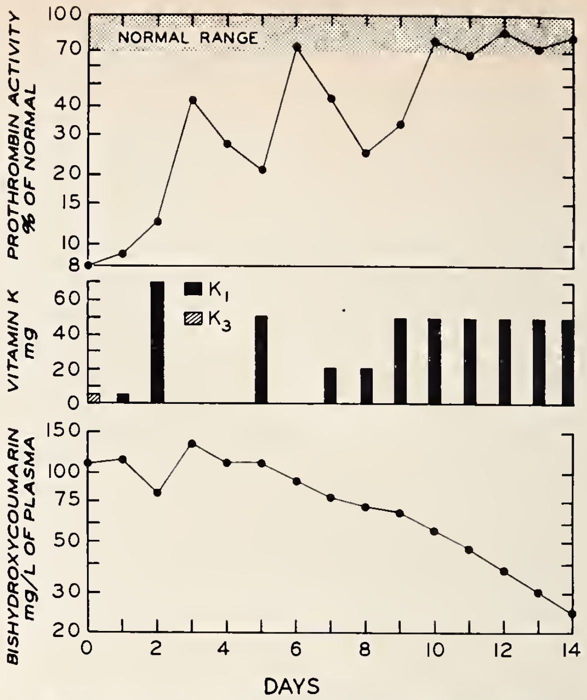


Figure 25.—Levels of anticoagulant and prothrombin activity in plasma and the response to vitamin K therapy in a patient suffering from surreptitious ingestion of bishydroxycoumarin. Source: O'Reilly, R. A., and Aggeler, P. M.: Surreptitious ingestion of coumarin anticoagulant drugs, Ann. Intern. Med., 64:1034-1041, 1966.

The production of factitious disease by a wide variety of means is well known. Individuals have been found to induce thyrotoxicosis with the self-administration of large doses of thyroid, diarrhea with phenolphthalein and hypokalemia with chlorothiazide; some have caused persistent skin ulcerations by excoriation with acids and others have produced factitious purpura by self-inflicted blunt injury. Within the past four years we have found warfarin or dicumarol in the plasma of 12 patients who had ingested the drug surreptitiously (Figures 26 and 27). All but one were women; almost all were young adults. One was a physician, seven were nurses and one was a pharmacy clerk.

Only three of the patients had no medical affiliation. Two of these, however, had been treated previously with anticoagulant drugs. Five of the patients had a previous history of bleeding episodes, which in all likelihood were also caused by surreptitious ingestion of anticoagulant drugs. Widespread ecchymosis was the most common hemorrhagic finding, although hematuria, metrorrhagia and hematochezia also occurred.

The prothrombin complex activity was less than 10 per cent in all cases; four of the patients had ingested warfarin and eight dicumarol. An admission of surreptitious ingestion could be obtained from only four patients. People frequently ask us why anyone would do such a stupid thing as this. We really don't know the motive, but in our cases it appeared to be either suicide or the production of a factitious hemorrhagic disease in order to attract attention, to gain sympathy or to obtain narcotic drugs. It has been suggested that patients of this type differ from most malingerers and many hysterics in that their disability is not directed to immediate material gain or advantage. It has been theorized that these patients, unlike hysterics, are capable only of limited repression and dissociation, and that when a certain threshold of mental conflict is exceeded, they are driven to a consciously determined symptomatic solution, whether the original motivating situation is consciously appreciated or not. Whatever the psychological basis may be, the proper diagnosis of surreptitious ingestion is important in order that attention may be focused on the psychiatric rather than the somatic disorder. I would also like to mention that with the aid of chemical assays we have found the opposite situation. In several patients we have been able to trace purported resistance to coumarin drugs to simple, willful, secret rejection of the prescribed medication. We call this "surreptitious non-ingestion" of the drugs.

CASE NO.	SEX	AGE	OCCUPATION	PREVIOUS ANTI-COAGULANT THERAPY	PREVIOUS HEMORRHAGIC EPISODE	HEMORRHAGIC MANIFESTATIONS
1	M	30	PHYSICIAN	NO	NO	ECCHYMOSES, HEMATURIA
2	F	23	NURSE	NO	NO	"
3	F	38	NURSE*	YES	NO	METRORRHAGIA,",HEMATOCHEZIA
4	F	26	NURSE	NO	YES	ECCHYMOSES, HEMATOCHEZIA
5	F	30	PHARMACY CLERK	NO	NO	" HEMATURIA
6	F	63	HOUSEWIFE	YES	YES	"
7	F	28	NURSE	NO	NO	"
8	F	39	HOUSEWIFE	NO	NO	"
9	F	44	BANK CLERK	YES	YES	"
10	F	38	NURSE	NO	YES	"
11	F	30	NURSE	NO	NO	" HEMATURIA
12	F	30	NURSE	NO	YES	METRORRHAGIA

*ALSO PHYSICIAN'S WIFE

Figure 26.—Findings in surreptitious ingestors of coumarin anticoagulant drugs.

CASE NO.	PROTHROMBIN COMPLEX ACTIVITY IN %	ANTI-COAGULANT INGESTED	PLASMA CONCENTRATION OF DRUG IN mg/L	APPARENT MOTIVE	ADMISSION OF INGESTION
1	< 5	WARFARIN	5	TO INDUCE PUPURA	NO
2	< 5	"	5	UNKNOWN	NO
3	< 5	"	8	SUICIDE	NO
4	< 5	"	45	TO INDUCE PUPURA	YES
5	< 10	DICUMAROL	22	" " "	NO
6	< 10	"	54	UNKNOWN	NO
7	< 10	"	80	TO INDUCE PUPURA	YES
8	< 5	"	82	UNKNOWN	NO
9	< 10	"	82	SUICIDE	NO
10	< 10	"	95	TO INDUCE PUPURA	NO
11	< 10	"	135	SUICIDE	YES
12	< 5	"	192	"	YES

Figure 27.—Findings in surreptitious ingestors of coumarin anticoagulant drugs.

In summary: I have attempted to show the importance of the careful study of individual cases in the development of knowledge regarding the blood clotting mechanism. I have also reviewed a few of the pharmacologic features of the oral anticoagulant drugs which may influence the response to therapy, and I have indicated the nature of the disorder which results from the surreptitious ingestion of these drugs. I hope that the intensive investigation of selected patients with blood coagulation anomalies will continue to yield information of some fundamental significance.

In closing, I would like to thank my two favorite patients, Kent Kincaid and Harry Martin, for their superb cooperation in our investigations which now have extended over a number of years. Mr. Martin is still in good health at the age of 78 years and is probably at this moment out tramping the hills in search of that ephemeral oil deposit he swears will some day make us all rich. Kent is now 30 years old and a successful primary school teacher. He also does part-time work in our laboratory. He still has plenty of bleeding problems but he is very active. Last year he left a picture of himself on skis on my desk, with the cryptic title, "K.K.—29-yr.-old Caucasian male with no siblings." However, last night he left me a note on our laboratory blackboard, which reads "Dear Dr. Aggeler, Remember—I don't race cars anymore!"

Medicine in 2032

An Impressionist's View

MALCOLM S. M. WATTS, M.D., *San Francisco*

A CHILD CONCEIVED in 1900 became eligible for "Medicare" (PL 89-97) in 1966. A child conceived in 1966 becomes eligible for "Medicare" in 2032. This gives some measure in terms of human life of the span of time we are to consider. There is not time here to review the past. The future is upon us already. The task of picturing what medicine will be like in 2032 staggers the imagination. One can only paint broadly with the suggestive strokes of the impressionist, and be thankful that human biology is such that this impressionist, at least, will not be on hand for the public exhibition and critique in 2032.

It has been estimated that since 1900 the total amount of scientific knowledge has approximately doubled during each decade. This exponential rate of acquisition of new knowledge shows no signs of slowing. If this rate actually continues the total body of scientific knowledge in 2032 will be about 96 times what it is today. The implications of this for medicine are fantastic. They will still be fantastic even if the expansion turns out to be only one-half or one-fourth of the figure anticipated. This knowledge explosion is the fundamental force which will determine the characteristics of medicine in 2032.

No one has prophetic vision, but perhaps one may sense some of what is sure to come. May I try a few impressionistic vignettes and then make a brave and perhaps foolhardy try with a broader canvas?

Vignettes

By 2032 infectious disease, even the common cold, will have been virtually eliminated. Chronic diseases, such as heart disease, cancer and stroke, will still be around, but much more preventable and much more treatable. Injuries, whether due to illness or accident, will continue to plague a faster moving and faster living technologic society. Prevention of injuries, and of illness as well, will have become a major preoccupation of a government still seeking to make good on its guarantee of the right of health to all, and having assumed responsibility for most, if not all, of the health bill. Surgical operation, which now is largely a matter of excision or repair may well have become dominated by the then relatively simple exchange or replacement of diseased or damaged parts, much as is done in the garages of today. Mental illness will, of course, have become identified as chemical derangement and psychiatry as we know it now will have ceased to exist. The aging process will be as inexorable and irreversible as ever. Although the average human life span will have been extended, perhaps even dragged out for many, death will be just as inevitable, and in many instances just as welcome in 2032 as it is today.

It is quite certain that by 2032 life itself will have long since been created in the scientific laboratory. There will be a growing body of knowledge of how living things can be shaped, moulded and modified at will. The genetically inherited diseases, diabetes for example, will probably have responded substantially to research into the chemical properties of the genes themselves, and to yet-to-be-

---

An address presented in connection with the "Community Time Capsule" sponsored by the Intercoast Life Insurance Company, Sacramento, 6 October 1966.

Reprint requests to: 909 Hyde Street, San Francisco 94109.

discovered techniques to alter these genes. This new capability for tampering with the fundamental life processes themselves will have far-reaching implications for medicine.

Yet the dream of the 1960's, that health for all is something that can be planned and provided equally for all, will elude realization, and by 2032 some of the reasons for this will have begun to enter the social consciousness. The process of enlightenment began in the last century when scientific progress finally established that man is a biologic species subject to biologic law. In similar fashion, by 2032, scientific progress in this century will have established that human society, being composed of human beings, is in fact biologic in nature and also subject to biologic law. As this reality permeates the social consciousness, many of the assumptions and theories upon which the health dream of the '60s was based will have to be discarded in recognition of biologic fact. Important changes will occur when human uniqueness, the fact that no two humans are identical, that each individual is chemically and biologically different from every other individual, fully permeates the social philosophy. When this finally occurs, the "proposition that all men are created equal" upon which so much of present-day social philosophy is based, must eventually become recognized as unscientific myth.

A scientifically oriented society, recognizing biologic law as its matrix or ground substance, will then place less emphasis upon the social ideal of a human equality which can actually never be achieved. Rather it will address itself more realistically to the encouragement of uniqueness and individual fulfillment, and to accomplishing this within the framework of a complex, highly specialized technologic society. Perhaps the "beatniks" of today (who will be about 85 years old in 2032) will actually turn out to have been the forerunners of a new renaissance. They may prove to have been among the first to reject the rigid socialization, based upon the erroneous concept that all men are equal, which will have been attempted by political theorists and their legislative supporters during the latter half of the Twentieth Century.

There are other portents of a growing importance for human biology and therefore for medicine. By 2032 the population crisis which we now foresee will have found some kind of solution. Whatever the solution, it will have made a mockery of present Twentieth Century efforts at population control. The biologic desire, even need, for reasonably unrestricted copulation will not be sufficiently curbed by any voluntary restraints. Human sterilization will become mandatory under circumstances which will be determined by society. It may be anticipated that there will be concern not only with the quantity but also with the quality of the population which is to be procreated. There will be some kind of selection for mental, physical and emotional fitness. Medicine and the health sciences cannot but play an important role in this effort of the human race to guide the further evolution of its own species.

By 2032 a biologically oriented society will have learned that a mad man in a position of power can be devastatingly destructive. The experience with Adolph Hitler was not enough. Nuclear power will make the point—short (we may hope) of some sort of cataclysm. It is likely that by then medicine will have been brought to bear to monitor the sanity of political leaders and any others who are in a position to unleash forces of enormous physical, biologic or social destruction. Similar attention will be given to the sanity of those who are led; and the equally dangerous aberrations of mass psychology will have become recognized as problems of medicine and human biology.

By 2032 medicine will be deeply concerned with the learning process and with improving the utilization of the human intellectual capacity. The enormous task of transmitting an exponentially growing body of knowledge from one generation to the next will have become recognized for the biologic problem that it is. Much more will have to be accomplished in the same or less time. Medicine will become an active participant in the educational process to an extent undreamed of at present.

A Broader Canvas

So much for vignettes. Now for an impressionist's try at a broader canvas.

The medicine of the future and the role of the physician will be the resultant of three major forces: First, scientific progress; second, acceptance of the World Health Organization conception of health as "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity"; and, third, recognition that not only the human being but society itself is biologic in nature and subject to biologic law. Let us for a moment examine each of these forces.

Scientific progress will bring about a staggering specialization of personnel and facilities. Much of the work now done by the physician will be done by other members of the health team—that is, by the allied health professions, by paramedical technicians and by physicians, many of whom will by then have become so technically specialized that they will in fact simply be highly trained technicians on the health team.

The new concept of health, "a state of complete physical, mental and social well-being," actually equates health with human welfare. Health, perhaps under the guise of "rehabilitation," will absorb all of what are now called social services and social welfare. This "rehabilitation" will come to concern itself with all the disadvantaged and deprived, whether the disadvantage be physical, mental, social, educational, economic, political or simply the harsh result of automation and technologic progress. The concept of human equality is intolerant of "welfare" as we know it, but it can accept "rehabilitation" as part of a governmental responsibility to guarantee health as a human right.

The recognition that not only the human being but society itself is biologic in nature and subject to biologic law will focus attention upon the biological characteristics of human institutions and the factors which govern their physical, emotional and social health. Like the various biologic species, human institutions in society have variable life spans and undergo birth, infancy, growth, maturity and senescence. They may be trim and fit, or they may be fat and flabby. Their death can and does occur from inability or failure to adapt for survival. Like any complex biologic species, organizations of human beings must adjust to internal and external stress, and their failure to do so results in illness, injury or emotional disturbance. The technology of biology and medicine will surely be applied to the health and diseases of these human organizations and institutions themselves, as well as to the human individuals who compose them and must interact with them.

The scope of medicine will be determined by each of these forces. But medicine will never be able to escape the basic one-to-one human relationship which is the heart of its role in society. It has been well stated that:

"In the last analysis, the ultimate situation in medical care is the one in which two human beings are working together upon the basis of a close interpersonal relationship. This relationship is the atmosphere in which scientific and technical procedures are applied."*

It would therefore appear that the true physician in 2032 will be a new kind of human biologist, a new kind of health advisor to patients, to people as individuals, to business, industry, communities, government, to virtually every segment of a society which recognizes its biological nature. The physician in 2032 will be the central figure of a complex health team with access to most complex facilities, the use of which he can tailor to meet the needs of an individual patient, an individual seeking self-expression and self-fulfillment in an interdependent society, or to the needs of some organizational segment of society. However, many of the present-day physician's medical colleagues will have foregone this broad yet personal responsibility to become specialized technicians within the health team and these doctors will work closely with many other technicians and professions in various technical aspects of health care. But the true physician of 2032 will be both scholar in the disciplines of human biology and professional health advisor to patients and others who seek a "state of complete physical, mental and social well-being," whether for themselves or for the human institutions with and within which they must work.

It would therefore appear that medicine will be quite different by the time a child born next year would become eligible for "Medicare"—under the present law, of course.

---

*A statement of Dr. Ward Darley at a Conference on "Medical Care of the Future," University of California Medical Center, San Francisco 18 and 19 June 1966.

Myasthenia Gravis: Brief Guide to Diagnosis and Management

CHRISTIAN HERRMANN, JR., M.D., *Los Angeles*

■ *Physicians should consider the possibility of myasthenia gravis in patients complaining of fluctuating muscle weakness and easy fatigability. The disorder may occur in either sex at any age. Repeated measurements of multiple muscle groups and functions to include baseline strength and response to a control as well as anticholinesterase drugs are desirable in reaching a diagnosis. Appropriate doses of anticholinesterase medication reduce weakness and fatigability in most patients. Selected patients may benefit from thymectomy. Patients in crisis failing to respond to anticholinesterase drugs, having difficulty maintaining a patent airway or adequate respiratory exchange are best managed by prompt tracheostomy using a cuffed tube, with adequate tracheobronchial toilet and mechanical respiratory assistance.*

MYASTHENIA GRAVIS IS a disorder manifested by varying weakness and excessive fatigability of the voluntary muscles of the body. Following a brief period of rest, there is at least a partial recovery of strength.[13] The exact cause of myasthenia gravis is unknown. The possibilities under consideration include a circulating neuromuscular blocking agent similar to curare,[23,24] an autoimmune process in which the body makes antibodies against its own muscle,[20,21] a defect in the production or liberation of acetylcholine at the neuromuscular junction,[2,22] and a slow virus infection with long latency and low virulence.[4] The disorder is not new. Early descriptions are credited to Thomas Willis in 1672.[12] Erb and Goldflam gave more extensive descriptions of myasthenia gravis toward the end of the 19th century. It is sometimes called the Erb-Goldflam disease or myasthenia gravis pseudoparalytica.

Pathology

There are no specific pathological findings; however, voluntary muscle biopsy may show collections of lymphocytes, called lymphorrhages. Muscle atrophy and necrosis are less frequently seen.[3,17] Studies of the neuromuscular junction by special techniques may show irregular elongation of the motor end-plates as well as preterminal branching.[25] Thymic enlargement with lymphocytic germinal centers is common. Thymomas also are frequent.

Incidence and Forms

The exact incidence of the disorder is unknown since it is not reportable to health authorities. Estimates range between one to 40,000 and one to 10,000 of the general population.[10]

From the Myasthenia Gravis Clinic, Division of Neurology, Department of Medicine, University of California School of Medicine, Los Angeles.

This work was supported in part by the California Chapter of the Myasthenia Gravis Foundation.

Submitted 24 October 1966.

Reprint requests to: Division of Neurology, UCLA School of Medicine, Los Angeles 90024.

The disorder occurs in both sexes at any age and in any race. It is more common in women in their late teens and before the age of 40 and in men after the age of 40.[16] It is not directly inherited, but there is a greater familial incidence than might be expected by sheer chance.[10] Two forms of the disorder occur in infancy. One is a transient, neonatal myasthenia occurring in some newborns of myasthenic mothers. This seldom lasts more than a few days or weeks, following which the infant is normal. The other form is congenital myasthenia gravis occurring in infants of non-myasthenic mothers. This is a relatively permanent form of the disease. It is often more symmetrical in distribution than the adult form, but similar in other respects.

Symptoms and Signs

Symptoms of the disorder are characteristically fluctuating and evanescent early in its onset. They may consist of varying amounts of any of the following, alone or in various combinations: drooping of one or both eyelids, double vision, facial weakness and difficulty smiling, trouble chewing, trouble swallowing, trouble talking, weakness of the neck, difficulty raising the arms above the head, weakness of the arms, forearms and hands, with difficulty extending the fingers, difficulty flexing the thighs at the hips and weakness in the lower limbs, particularly in attempting to walk on the heels. These symptoms are summarized in Table 1. The weakness and fatigability are often worse in the presence of intercurrent infection, emotional stress, menstrual periods, sleep loss and increased physical activity.

The signs of muscle weakness and fatigability are similarly evanescent early, are variable and are asymmetric in amount and distribution. Some patients have a relatively restricted form of the disease, involving, for example, the extraocular muscles. Others may have a patchy distribution and in others the distribution may be more generalized. Ptosis of the eyelids is usually asymmetric and fluctuating. Upward, medial and lateral movements of the eyes are more frequently limited than downward movement. Again, the involvement of the extraocular muscles is asymmetrical. Pupillary responses are generally normal. The muscles closing the jaw are more frequently involved than those opening it, so that the patient may support his chin with his hand or use his hand to close his jaw in order to chew. Among the facial muscles, the orbicularis oculi is often more extensively involved than the orbicularis oris and buccinator. The face is relatively smooth and often expressionless because of weakness. The risorius is frequently spared, the phenomenon resulting in a sneering expression where a smile is intended. Weakness of the muscles of the soft palate and pharynx often accompany dysarthria and dysphagia. The patient may find protruding the tongue into either cheek difficult or impossible.

TABLE 1.—*Common Initial Symptoms of Patients with Myasthenia Gravis**

	Incidence (Per Cent) in		
Symptom	*Males*	*Females*	*All Patients*
Drooping eyelids	24	26	25
Double vision	55	31	41
Difficulty talking	7	23	16
Difficulty swallowing and regurgitation	5	14	10
Difficulty chewing	11	5	7
Weakness of hands and fingers	4	10	7
Weakness of legs	9	17	13
General weakness	5	15	11

*Findings from 177 patients with myasthenia gravis (102 females and 75 males) seen by the author in the myasthenia gravis clinic and Division of Neurology at UCLA School of Medicine, Los Angeles.

The neck flexors are more commonly involved than the extensors, and where that imbalance occurs the patient has difficulty raising his head from the supine position. Often the deltoids are involved and the patient has difficulty abducting and extending the arms at the shoulders or raising them above the head. The biceps or the triceps muscles, or both, may be involved, with varying degrees of weakness in flexing and extending the forearms at the elbow. Wrist extensors and, in particular, the finger extensors are more frequently and severely involved than finger flexors. Weakness and fatigability among the intrinsic muscles of the hand are common within the thenar and hypothenar groups, in particular the abductor pollicis brevis and abductor digiti quinti. Weakness of the infraspinati at the shoulders is manifested by difficulty in externally rotating the arms at the shoulders with elbows at the side.

Varying degrees of weakness and fatigability of the respiratory muscles, including intercostals, diaphragm, and accessory muscles of respiration, may be manifest by shallow breathing, little movement of the rib cage, muffled cough and reduced vital capacity. Prompt reduction of respiratory excursions and vital capacity may occur following repeated deep breathing or a rapid series of coughs.

About the hips the iliopsoas group is most frequently involved. This may be detected on flexion of the thigh at the hip and by having the patient repeatedly cross and uncross the knees in the sitting position. The abductors of the thighs at the hips are more frequently involved than the adductors, and the knee flexors more frequently than the extensors. The dorsiflexors and everters of the foot at the ankle as well as the extensors of the toes are more commonly involved than the plantar flexors and the inverters of the feet. For this reason the patient is often able to walk on the toes but is unable to walk on the heels or raise the forefoot off the floor in the standing position with the heel still touching the floor. (See Table 2.)

Diagnostic Measures

The diagnosis of myasthenia gravis should be suspected in patients complaining of muscle weakness and easy fatigability of muscle strength.[7,18] Tests may include having the patient sustain upward gaze to note any fatigue of the levators of the lids or the extraocular muscles. Continuous counting may detect fatigue of speech. Holding the arms extended or over the head may show fatigability of these muscles. Other means of demonstrating increased muscle weakness are repeated crossing and uncrossing of the legs, squatting exercises and repeated compression of a rolled-up and inflated blood pressure cuff or dynamometer with the hand. Fatigability should be searched for in these various muscle groups.

TABLE 2.—*Frequently Involved Muscles of Patients with Myasthenia Gravis**

Muscle or Muscle Groups	*Incidence (Per Cent) in*		
	Males	*Females*	*All Patients*
Lid levator	58	59	58
Extrocular muscles	63	47	54
Jaw	31	32	32
Orbicularis oculi	76	66	71
Face	64	62	63
Deglutition	20	26	24
Voice	28	37	33
Tongue	36	31	33
Neck	57	63	61
Shoulder and arm	73	71	72
Hand and finger	70	66	68
Leg	74	76	75
General	10	12	11

*Findings from 177 patients with myasthenia gravis (102 females and 75 males) seen by the author in the myasthenia gravis clinic and Division of Neurology at UCLA School of Medicine, Los Angeles.

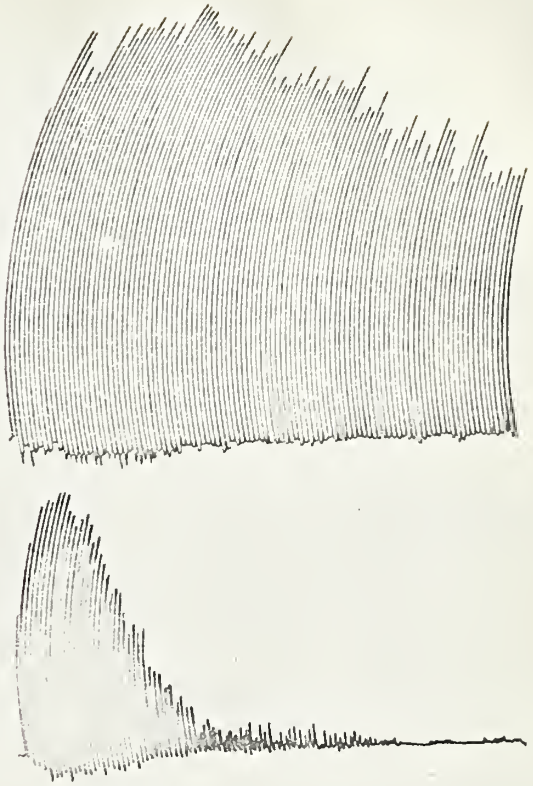


Figure 1.—Bulb ergograph tracings of the hand grips in normal person (above) and myasthenic patient (below) showing pronounced reduction in effective strength in the patient after approximately 30 squeezes in 30 seconds.

In the bulb ergograph test of the hand grip, the patient squeezes a rubber bulb similar to that on a sphygmomanometer at one-second intervals signaled by a bell. The bulb is attached to an ink-writing kymograph (Figure 1).

Another feature of myasthenia gravis is generally improved strength on administration of anticholinesterase drugs. The response to anticholinesterase therapy should be preceded by baseline observations of strength in several muscle groups as well as observations after a protective placebo injection of atropine to evaluate the psychologic aspects and suggestibility of the patient. The atropine also serves to protect the patient from the sometimes unpleasant or toxic cholinergic side effects of the anticholinesterase medication to follow. After a second period of rest for five to 10 minutes, he is given an injection of 1 to 2 mg of edrophonium (Tensilon®) intravenously and then is tested again.[15] If there is no favorable response to this dosage, a larger dose, 4 to 8 mg, may be given after another five to 10 minutes of rest. The patient is then retested for evidence of improvement or weakening as well as toxic side effects such as fasciculation of the muscles about the eyes, face and elsewhere, and increased salivation, lacrimation and sweating. Muscles detected to be weak before the administration of drugs should be retested systematically following each dose to note the presence or absence of any significant improvement or increased weakness.

Measurement of vital capacity as well as bulb ergograph tests may be used along with other observations for confirmatory help. Intramuscular neostigmine U.S.P. (Prostigmin®) or pyridostigmine (Mestinon®) may also be used for diagnostic testing, but results are slower and at times less well defined.

Electrical stimulation of the nerve to the thenar or hypothenar muscle groups, with recording of the resulting action potential in one of these areas, is also useful, but the procedure requires special equipment and experience. Tests with provocatives such as quinine and curare are best carried out by physicians experienced in the diagnosis and management of patients with myasthenia gravis in a hospital setting where facilities for a mechanical airway and artificial respiration are readily available.

TABLE 3.—*Other Disorders Entering the Differential Diagnosis of Myasthenia Gravis*

- Adynamia episodica hereditaria
- Amyotrophic lateral sclerosis (Progressive spinal muscular atrophy and progressive bulbar palsy)
- Botulism
- Catalepsy
- Chlorothiazide or hydrochlorothiazide electrolyte depletion
- Cholinergic crisis
- Dermatomyositis
- Familial periodic paralysis
- Hyperparathyroidism
- Hyperthyroidism
- Multiple sclerosis
- Myasthenic syndrome associated with anaplastic malignant disease
- Myasthenic component in other myopathic states
- Myotonia atrophica
- Narcolepsy
- Phosphorylase deficiency myopathy (McArdle's syndrome)
- Polymyositis
- Polyneuropathy
- Primary aldosteronism
- Progressive external ophthalmoplegia
- Progressive muscular dystrophy
- Psychogenic asthenic reaction

Routine x-ray films of the chest should include posteroanterior and lateral views. Lateral laminograms of the anterosuperior mediastinum may also be helpful in detecting the presence or absence of thymic hyperplasia or thymoma, but distinguishing between thymic hyperplasia and thymoma on radiographic findings alone is difficult or impossible.

There are at present no specific laboratory tests for myasthenia gravis. A partial list of other disorders entering into the differential diagnosis of myasthenia gravis is given in Table 3.

Therapy

Treatment may be started with oral administration of Prostigmin® 7.5 to 15 mg or Mestinon 30 to 60 mg two or three times daily, preferably one-half to three-quarters of an hour before meals.[11,19] The drugs may be taken with milk and crackers or bread, but should not be taken with coffee, fruit or tomato juice since they may enhance the parasympathomimetic side effects on the bowel and bladder. Dosage should be increased cautiously and only if followed by significant objective improvement in symptoms. Data on common anticholinesterase drugs are summarized in Table 4. Seldom does use of any of them bring about more than 80 per cent restoration of the normal muscle strength and performance. Even with optimal therapy a few patients do not respond significantly to anticholinesterase medication. It must be kept in mind that there is no evidence for a lack of anticholinesterase in the patient with myasthenia gravis and that these drugs are not normally present or lacking in the patient with myasthenia gravis. They are mainly pharmacological crutches used to facilitate what remains of neuromuscular transmission which is already defective in the myasthenic patient. An excess of anticholinesterase drug can produce increased weakness beyond that already caused by myasthenia gravis. Also a host of unpleasant, and at times serious, side reactions are produced.

Crisis

Myasthenia gravis may be aggravated by infections elsewhere in the body, particularly those involving the respiratory tract. It may also be aggravated by emotional stress and menses. The myasthenic process may increase to such proportions that the patient is said to have a myasthenic crisis. [5,8,14] In this circumstance the patient is

TABLE 4.—*Common Anticholinesterase Drugs Used in Diagnosis and Management of Myasthenia Gravis*

Drug	*Form*	*Adult Single Dose and Route*	*Usual Effective Duration and Range*
Tensilon® (Edrophonium chloride)	10 mg per ml	2-10 mg IV	10 minutes (2 min.-2 hours)
Prostigmin® (Neostigmin methyl sulfate)	0.25, 0.5 and 1.0 mg per ml	1 mg IM	2 hours (2-4)
Prostigmin® (Neostigmin bromide)	15 mg tablet	15 mg oral	3 hours (2-5 hours)
Mestinon® (Pyridostigmin bromide)	60 mg tablet	60 mg oral	4 hours (3-7 hours)
Mestinon Timespan® (Pyridostigmin bromide)	180 mg tablet (slow release)	180 mg oral	8 hours (6-12 hours)
Mestinon syrup® (Pyridostigmin bromide)	60 mg per 5 ml syrup	60 mg per 5 ml (1 tsp oral)	4 hours (3-7 hours)
Mytelase® (Ambenomium chloride)	10 mg and 25 mg tablets	10-25 mg oral	8 hours (6-10)

IV=Intravenous; IM=Intramuscular.

unable to maintain a patent airway or adequate respiratory exchange. In some instances this may result from inadequate anticholinesterase treatment; if so, an increase in the anticholinesterase dosage may help. Other patients may be unresponsive or insensitive to increased doses of anticholinesterase. In still others, weakness—called "cholinergic crisis" — may be provoked by an excess amount of anticholinesterase. The symptoms and signs of myasthenic and cholinergic crisis are listed in Table 5.

At times Tensilon may be used successfully to determine the need for more longer-acting anticholinesterase.[15] If the patient responds favorably to Tensilon, he may benefit from increased dosage of longer-acting anticholinesterase medication. However, in some patients there may be a positive response to Tensilon but no improvement, even deterioration of strength, following additional dosage of longer-acting anticholinesterase. Under these circumstances bedrest, tracheostomy, controlled or assisted mechanical respiration and withdrawal of anticholinesterase may be the treatment of choice.[5,8,14] This may be best handled in an intensive care unit facility for patients with chronic respiratory insufficiency or in a center dealing frequently with myasthenia gravis patients. At times it is difficult if not impossible to determine with certainty what kind of crisis is occurring —whether myasthenic, cholinergic, a combination of these or the so-called "insensitive state." Under these circumstances prompt control of the airway

TABLE 5.—*Symptoms and Signs of Myasthenic and Cholinergic Crisis*

Myasthenic Crisis	*Cholinergic Crisis*	
	Muscarinic Symptoms and Signs:	Nicotinic Symptoms and Signs:
Ocular ptosis	Sweating	Muscle fasciculations
Dysarthria or anarthria	Lacrimation	"Thick tongue"
Dysphagia or aphagia	Salivation	Dysarthric speech
Dyspnea or apnea	Anorexia	Dysphagia
Facial weakness	Abdominal cramping	Trismus
Masticatory weakness	Diarrhea	Muscle cramps and spasms
Difficulty handling secretions	Nausea	General weakness
General weakness	Vomiting	
	Urinary frequency	CNS Symptoms and Signs:
	Incontinence of bowel and bladder	Restlessness
	Miosis	Anxiety
	Blurred vision	Giddiness
	Bronchorrhea	Vertigo
	Dyspnea and wheezing	Headache
	Substernal pressure	Confusion and stupor
	Bronchospasm	Coma
	Pulmonary edema	Convulsions

and respirations should be obtained along with adequate tracheobronchial toilet and control of infection if present. After several days the patient's vital capacity may again improve along with strength elsewhere. Ultimately he may be able to manage for several days or weeks without anticholinesterase medication.

When administration of anticholinesterase is resumed, it should be started in low dosage and increased slowly.

Atropine to counteract parasympathomimetic or muscarinic side effects is fraught with hazards and it should be used sparingly if at all.[18] As this tends to mask the early signs of cholinergic intoxication, the patient may pass directly into the nicotinic or paralytic phase of cholinergic intoxication without other warnings. Also oropharyngeal and bronchopulmonary secretions are suppressed and they become tenacious and exceedingly difficult to remove. The patient seldom gains much improvement in voluntary muscular strength from additional anticholinesterase dosage once symptoms and signs of parasympathomimetic or muscarinic toxicity are present or suppressed with atropine or a similar parasympathetic blocking agent.

It is important for both the patient and his physician to understand that if one dosage level of anticholinesterase produces favorable effects, twice the dosage will not necessarily double the improvement, and it may increase weakness. Common side effects from anticholinesterase medication include increased sweating, salivation, abdominal cramps, diarrhea and urinary urgency and frequency.

Indications for Thymectomy

Thymectomy appears helpful in younger patients who have had myasthenia gravis less than five to seven years and who do not have a thymoma.[1,6,9,16] However, thymectomy is also indicated in patients suspected of having thymoma, since these tumors may increase in size and thus embarrass other intrathoracic structures. At times they also undergo malignant change. Patients with thymoma do less well with thymectomy than those with thymic hyperplasia. There is no evidence, however, that thymectomy in patients with thymoma unfavorably affects the course of myasthenia. Thymectomy should not be undertaken as an emergency procedure in a patient who is going rapidly down hill with myasthenia gravis or in one who is pregnant or who has active pulmonary inflammatory process.

While myasthenia gravis is a disorder often marked by remissions and exacerbations, a certain number of patients also achieve complete remission and freedom from symptoms, and no longer need anticholinesterase medication. With appropriate patient education, physician understanding, drug therapy and selective thymectomy, the majority of patients with myasthenia gravis may get along well.

Generic and Trade Names of Drugs

Endrophonium—*Tensilon.*®
Neostigmine U.S.P.—*Prostigmin.*®
Pyridostigmine—*Mestinon.*®

REFERENCES

1. Buckberg, G. D., Herrmann, Jr., C., Dillon, J. B., and Mulder, D. G.: A further evaluation of thymectomy for myasthenia gravis, J. Thorac. & Cardiovasc. Surg. (In press).

2. Desmedt, J. E.: Presynaptic mechanisms in myasthenia gravis, Ann. N.Y. Acad. Sci., 135:209-246, 1966.

3. Fenichel, G. M.: Muscle lesions in myasthenia gravis, Ann. N.Y. Acad. Sci., 135:60-78, 1966.

4. Gajdusek, D. C.: Slow, latent and temperate viral infections of the central nervous system, In: Proc. Assoc. Research in Nervous and Mental Disease, 44, 1964 (In press).

5. Glaser, G. H.: Crisis, precrisis and drug resistance in myasthenia gravis, Ann. N.Y. Acad. Sci., 135:335-349, 1966.

6. Henson, R. A., Stern, G. M., and Thompson, V. C.: Thymectomy for myasthenia gravis, Brain, 88:11-28, 1965.

7. Herrmann, Jr., C., and Rose, A. S.: Myasthenia gravis problems in diagnosis, Calif. Med., 87:373-379, 1957.

8. Herrmann, Jr. C.: Crisis in myasthenia gravis, In: Myasthenia Gravis, The Second International Symposium, edited by H. R. Viets, Springfield, Ill., Charles C Thomas, 1960, pp. 637-652.

9. Herrmann, Jr., C., Mulder, D. G., and Fonkalsrud, E. W.: Thymectomy for myasthenia gravis, Ann. Surg., 158:85-92, 1963.

10. Herrmann, Jr., C.: Myasthenia gravis occurring in families, Neurology, 16:75-85, 1966.

11. Johns, R. J.: Drug therapy of myasthenia gravis, Mod. Med., 33:286-306, 1965.

12. Keynes, G.: The history of myasthenia gravis, Med. Hist. (Lond.), 5:313-326, 1961.

13. Osserman, K. E.: Myasthenia Gravis, New York, Grune and Stratton, 1958.

14. Osserman, K. E., and Genkins, G.: Studies in myasthenia gravis: Reduction in mortality rate after crisis, J.A.M.A., 183:97-101, 1963.

15. Osserman, K. E., and Genkins, G.: Critical reappraisal of the use of edrophonium (tensilon) chloride tests in myasthenia gravis and significance of clinical classification, Ann. N.Y. Acad Sci., 135:312-334, 1966.

16. Perlo, V. P., Poskanzer, D. C., Schwab, R. S., Viets, H. R., Osserman, K. E., and Genkins, G.: Myasthenia gravis: Evaluation of treatment in 1,355 patients, Neurology, 16:431-439, 1966.

17. Russell, D. S.: Histological changes in the striped muscles in myasthenia gravis, J. Path. Bact., 65:279,289, 1953.

18. Schwab, R. S.: Problems in the diagnosis and

treatment of myasthenia gravis, Med. Clin. of North Amer., 47:1511-1524, 1963.

19. Schwab, R. S.: Management of myasthenia gravis, New Engl. J. Med., 268:596-597, 717-719, 1963.

20. Simpson, J. A.: Myasthenia gravis as an autoimmune disease: Clinical aspects, Ann. N.Y. Acad. Sci., 135:506-516, 1966.

21. Strauss, A. J. L., Smith, C. W., Cage, G. W., van der Geld, H. W. R., McFarlin, D. E., and Barlow, M.: Further studies on the specificity of presumed immune associations of myasthenia gravis and consideration of possible pathogenic implications, Ann. N.Y. Acad. Sci., 135:557-579, 1966.

22. Thesleff, S.: Acetylcholine utilization in myasthenia gravis, Ann. N.Y. Acad. Sci., 135:195-208, 1966.

23. Wilson, A., and Stoner, H. B.: Myasthenia gravis: A consideration of its causation in a study of 14 cases, Quart. J. Med., 13:1-18, 1944.

24. Windsor, C. E.: Preliminary report on the effect of the serum of myasthenia gravis patients on the neuromuscular transmission of the intact frog. In: Myasthenia Gravis, The Second International Symposium, edited by H. R. Viets, Springfield, Ill., Charles C Thomas, 1960, pp. 217-228.

25. Woolf, A. L.: Morphology of the myasthenic neuromuscular junction, Ann. N.Y. Acad Aci., 135: 35-59, 1966.

The Role of Radiation Therapy In Myasthenia Gravis

THEODORE L. PHILLIPS, M.D., AND FRANZ BUSCHKE, M.D., *San Francisco*

■ *Myasthenia gravis is a serious and debilitating disease associated with conduction defects occurring at the myoneural junction. About 15 per cent of the patients have associated tumors of the thymus and 80 per cent of the remaining patients show abnormalities of the thymus. Although a definite relationship between cause and effect has not been proved, thymectomy or radiotherapy of the thymus does seem to influence the disease.*

Seven cases of myasthenia gravis in which radiation therapy was used at the University of California Medical Center are reported and compared with those described in the literature. It is concluded that patients whose disease is progressing and not well managed medically, and who have no evidence of thymoma, should be treated by irradiation—whether preoperatively or as definitive treatment, depending on the result of irradiation. Those patients with evidence of thymoma should be irradiated before surgical procedure. Small tumors, or patients in whom surgical risk is increased, may be managed by radiation therapy alone.

MYASTHENIA GRAVIS is a serious disease which involves from 10,000 to 20,000 persons in the United States and is fatal in at least 10 per cent of cases.[20] Since 1901, when Weigert[25] reported a tumor of the thymus associated with myasthenia gravis, the relation of this organ to the cause and treatment of myasthenia has been of great interest. Between 8.5 and 15 per cent of patients with myasthenia gravis reported in the literature had associated thymomas. In 31 to 45 per cent of cases of thymoma, associated myasthenia gravis has been mentioned.[5,14] Recent investigations have demonstrated a possible relationship between myasthenia gravis and autoimmune diseases[22-24] and the importance of the thymus in the development of the immune mechanism.[15]

In patients without demonstrable thymoma at the time of operation or at necropsy, the thymus appears normal in 20 per cent and abnormal in 80 per cent.[4] These abnormalities consist of hyperplasia of the medulla and the presence of germinal centers in the medullary tissue. Thus, although its direct involvement has not yet been proved, the thymus definitely is closely associated with myasthenia gravis.

The discovery that cholinesterase inhibitors greatly improved the symptoms of myasthenia gravis led to the development of useful clinical agents. With these, most patients have attained a

---

From the Section of Therapeutic Radiology, Department of Radiology, University of California School of Medicine, San Francisco.

Presented before the Section on Radiology at the 95th Annual Session of the California Medical Association, Los Angeles, 19 to 23 March 1966.

Reprint requests to: Department of Radiology, U.C. Medical Center, San Francisco 94122 (Dr. Phillips).

fairly active life. According to Schwab,[20] in about one-third of cases the disease can be kept in check with drug therapy, in about one-third it is sufficiently alleviated that the patients have 60 to 75 per cent of normal strength and in the remaining one-third, patients are only partially helped by drug therapy. Thus, myasthenia gravis is not always well managed by drug therapy alone. Indeed, many patients continue to have severe symptoms even at dose levels at which pronounced side effects occur. For this reason, other means of treatment were sought, chiefly directed at the thymus because of the previously described association.

Surgical excision of the thymus in patients with myasthenia gravis but without evidence of thymic tumor was begun in a systematic manner by Blalock and coworkers.[3] This method has been applied in many treatment centers. Significant benefit has been reported in females in whom the disease had developed before the age of 30 years and had been present for less than two years.[11] In 100 surgically and 100 medically treated patients with myasthenia gravis, Schwab[19] noted complete remissions in 27 and 10 per cent, respectively.

This response to surgical therapy was not observed in patients with myasthenia gravis and thymic tumor. Keynes[12] reported only one survival in his first 11 myasthenic patients with tumor treated surgically. The high incidence of death resulted chiefly from fulminating myasthenia gravis in the immediate postoperative period. Perlo, Schwab and Castleman[17] reported that the average survival of patients with myasthenia gravis associated with thymomas and treated by operation alone was only 1.4 years.

The apparent relief of myasthenia gravis accomplished by surgical removal of the thymus led to early attempts at improvement by radiation therapy. Aring[1] reported three patients in 1942 in whom results were encouraging. After observing a poor survival rate from surgical treatment alone in thymoma, Keynes[12] gave these patients preoperative irradiation before excising the tumor, which led to improved results. Perlo, Schwab and Castleman[17] reported similar findings and, in addition, noted a 9 per cent higher remission rate[19] in patients without tumors who had irradiation preoperatively. These results encouraged certain large centers to adopt irradiation for the treatment of some cases of myasthenia gravis.

Since controversy exists regarding this mode of treatment, the records of seven patients with myasthenia gravis who were treated by radiation therapy at the University of California Medical Center were reviewed. The results were analyzed and then compared with those reported in the literature to better establish the role of radiation therapy in this disease. Following the brief case reports, the results of treatment will be presented. In the accompanying Table 1 the severity of the initial disease is graded from 1+ to 4+ and the response to treatment indicated by the letters *A* through *E*. This system is a slight modification of that used by Keynes.[11]

Reports of Cases

CASE 1.—Myasthenia gravis was diagnosed in a 44-year-old woman in February 1955. At the time of admission a tumor of the thymus was noted, which, after exploration and biopsy, proved

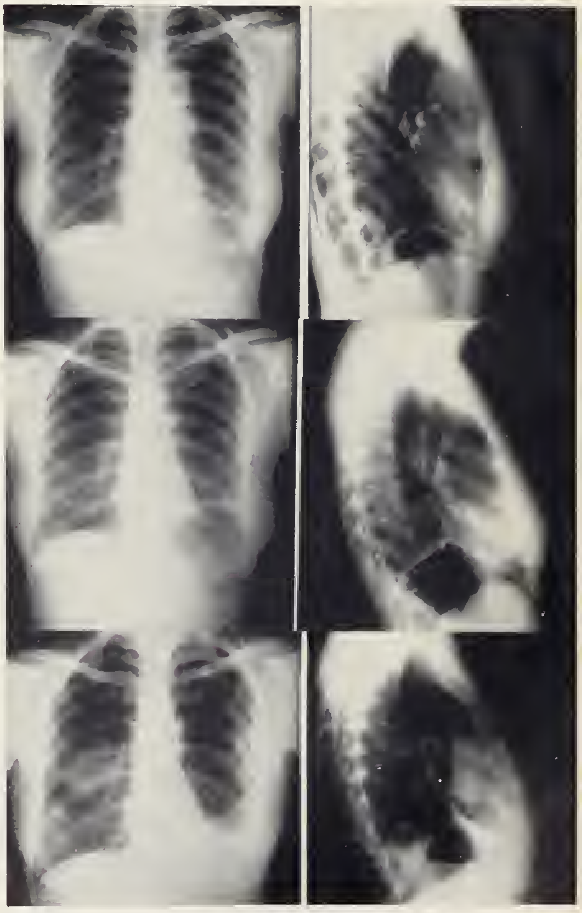


Figure 1.—(Case 1) Thymoma with invasion of great vessels and pleural metastasis. *Above*, roentgenogram before surgical procedure shows large thymoma. *Center*, regression of mass after partial resection and 4.000 r grid therapy. *Below*, one year later, enlargement of mediastinal mass and pleural nodules.

to be a lymphoepithelial thymoma with local invasion which precluded resection. Evidence of an anterior superior mediastinal mass was seen on a roentgenogram (Figure 1A) and, in addition, at thoracotomy, multiple pleural implants were found. Because of moderately severe myasthenia gravis, neostigmine was given, 30 mg every four hours. Between 10 March and 15 April 1955, a single anterior grid field was irradiated with 400 KvP to a maximum tumor dose of 4,000 r and a minimum tumor dose of 1,000 r at a 9 cm depth. After the irradiation the patient's symptoms improved markedly, the tumor regressed and the dose of neostigmine was cut in half for the next few months (Figure 1B). One year later myasthenic symptoms as well as cough had recurred. By September 1956, a decided increase in the size of the tumor as well as of the pleural nodules was evident (Figure 1C). As the main growth had occurred in the nodules, these were treated with small doses of radiation, but without significant result. The patient died on 3 October 1956 and necropsy revealed thymoma with multiple pleural implants. No evidence of spread below the diaphragm was seen.

Case 2.—In May 1957, symptoms of myasthenia had developed in a 53-year-old woman. One month later a tumor of the thymus and a widened upper mediastinum were revealed by roentgenogram (Figure 2). At operation in June 1957 a tumor of the thymus was observed which proved to be a lymphoepithelial thymoma. The lesion was large, adhering to the major vessels and showing evidence of invading the right lung. In addition, there were separate nodular implants on the lung and pericardium from which only a biopsy specimen was taken. Before the operation the patient required neostigmine 0.75 mg intramuscularly four times a day. After the operation neostigmine was discontinued and pyridostigmin, 120 mg, was given three times a day. Irradiation with 250 KvP was delivered to anterior and posterior fields of 10×15 cm. A midplane dose of 3,500 r was delivered in 47 days in August and September 1957. The symptoms improved and the dose of the drug was reduced. Symptoms recurred in October 1958, requiring an increase in the drugs. The tumor was enlarging and was retreated at this time. In February 1959 the patient died, presumably from liver metastasis, but necropsy was not performed.

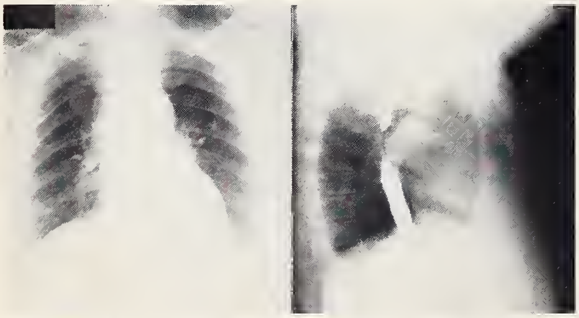


Figure 2.—(Case 2) Preoperative roentgenogram showing huge anterior mediastinal tumor which proved to be an invasive unresectable thymoma.

Case 3.—The diagnosis of myasthenia in a 54-year-old woman was made in August 1958. By March 1959 she was requiring 33 tablets (60 mg each) of pyridostigmin a day and was experiencing some side effects. The symptoms were not completely relieved by this drug. Roentgenograms of the chest gave no evidence of thymic tumor. Because of failure of complete response to medical management, the thymus was irradiated. In April and May 1959, a total skin dose of 4,600 r, 3,600 r at 4 cm depth, was delivered in 31 days by means of a 1 MeV generator. No significant change was noted during or after therapy. By January 1966, the patient's condition remained unaltered and she was still requiring pyridostigmin, 34 tablets daily. An attack of acute thyroiditis had occurred eight months previously.

Case 4.—The patient, a 50-year-old woman, was seen at the University of California Medical Center in December 1961. Myasthenia gravis had been diagnosed in 1952 because of symptoms of ptosis and of weakness in the arms and legs. Medication (kind unknown) was given for a brief period, but was stopped when symptoms improved. In July 1958 the symptoms had recurred, and ambenomium, 25 mg four times a day, was prescribed. In November 1961, a mass was seen in the anterior mediastinum on roentgen examination (Figure 3A). Mild symptoms of myasthenia gravis were present at that time and the patient was taking ambenomium, 10 mg every four hours. The symptoms increased in December 1961, and pyridostigmin, 300 mg a day, was given as a trial dose for one week. Then the prescription was changed back to ambenomium, 10 mg every three hours. The symptoms were relieved but, because of emotional overlay, evaluation of relief was difficult. The patient received irradiation between December 1961 and February 1962. A skin dose of 5,200 r, 3,900 r at 6 cm depth, was given in 53 days with 1 MeV radiation. The tumor regressed completely. By April 1962 the amount of medication had been decreased and in August 1964, when it was stopped completely, there was no evidence of neurologic defects and roentgenogram had remained essentially normal (Figure 3B). In November 1965 the patient's condition was excellent and a neurologist expressed the opinion that remission was complete. No evidence of recurrence of the tumor was noted on roentgen examination at that time.

CASE 5.—Myasthenia gravis was diagnosed in the patient, a 27-year-old Filipina, at the Massachusetts General Hospital by Dr. V. P. Perlo in January 1961. The disease was fairly severe. Because of the patient's age and the short duration of disease, radiation therapy and subsequent excision of the thymus had been advised and the patient was referred to us for preoperative irradiation. Just before radiation therapy the patient was taking 20 to 25 tablets (60 mg each) of pyridostigmin daily, as well as ambenomium, 15 mg three times a day, and a long-acting pyridostigmin tablet of 180 mg at bedtime. Roentgenograms of the chest revealed no evidence of a thymic mass.

We treated this patient by radiation through a single anterior field in March and April 1962. She was given 3,900 r to the skin, 3,000 r at 5 cm depth, in 36 days with cobalt-60. On reexamination in May 1962, Dr. Perlo expressed the opinion that symptoms had definitely improved, although medication had not been changed. In October 1963 the results of neurologic examination were within normal limits. Except for discontinuation of ambenomium, medication was not changed. Because of this continued improvement, Dr. Perlo, who observed the patient regularly, decided against the initially anticipated operation. The patient's condition had remained the same by January 1964, but by October 1964 the amount of pyridostigmin had been reduced to 14 tablets per day. The patient was active and was working full time. In October 1965 she was still in good health and had just married.

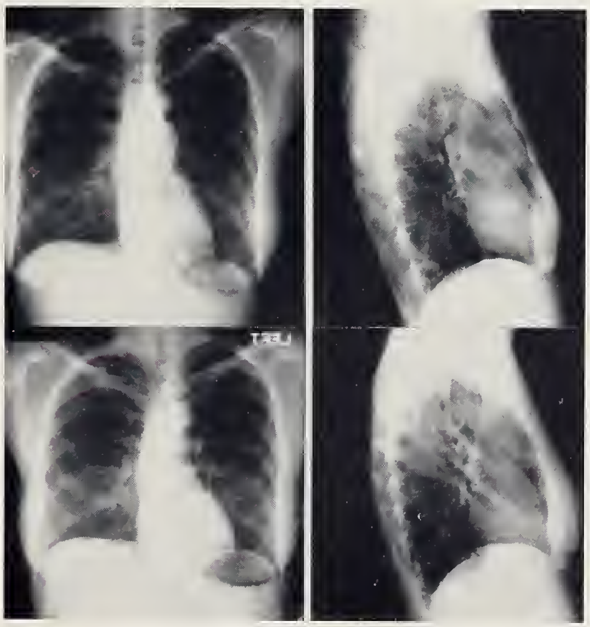


Figure 3.—(Case 4) *Above*, pretreatment roentgenogram showing anterior mediastinal mass interpreted as thymoma. *Below*, asymptomatic, two and one-half years after radiation therapy. Roentgenogram of chest shows regression of mass and appears essentially normal.

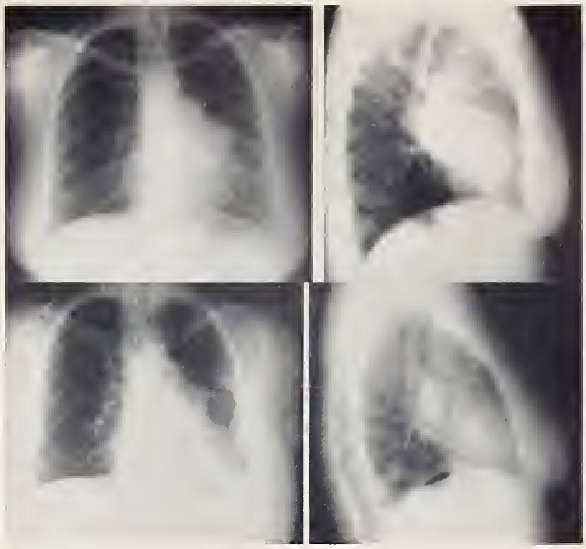


Figure 4.—(Case 6) *Above*, before thoracotomy roentgenogram of the chest shows large left hilar and anterior mediastinal mass which on biopsy proved to be a pulmonary small-cell carcinoma. *Below*, after thoracotomy and 5,500 r to mediastinum and left side of chest in 107 days.

CASE 6.—The patient, a woman, was first seen in June 1965 at the age of 33 years, at which time myasthenia gravis had developed. A huge mass in the left hilar area, as well as in the mediastinum, was revealed by a roentgenogram (Figure 4A). The patient said that easy fatigability and weakness of the legs, particularly of the thighs, had begun in September 1964. She had congenital ptosis. A muscle biopsy, done in January 1965 because of atrophy of thigh muscles, was reported as showing no abnormality. In May 1965 a test with edrophonium was equivocal. Because of the mediastinal mass, a thoracotomy was done after negative results on bronchoscopy and scalene biopsy. A huge mass involved the left hilum and the mediastinum, and a peripheral nodule was present in the left upper lobe. A biopsy of the mass showed an undifferentiated malignant lesion, probably a pulmonary small-cell carcinoma. Subsequent to the thoracotomy, decided weakness of the shoulder and hip muscles was noted, but an electromyelogram was not diagnostic. Although the symptoms of weakness improved after intravenous administration of edrophonium the myopathy was considered as secondary to pulmonary tumor. Neostigmine, 15 mg every two hours, was administered, resulting in a definite increase in strength. This medication was changed to long-acting pyridostigmin, 180 mg twice a day, which, during the initial phase of irradiation, was combined with neostigmine, 7.5 mg every two hours. In 107 days from June to September 1965 the midplane of the mediastinum and the entire left chest received 5,500 r. After the delivery of the first 2,500 r, all medication was discontinued because of pronounced improvement. The mass decreased (Figure 4B) and symptoms of muscle weakness lessened considerably. By February 1966 the mass had decreased further, but some fullness remained in the left hilar area. Strength at that time appeared normal. At the end of March 1966, evidence of regrowth of the tumor was noted and weakness was increasing.

CASE 7.—The patient, a man, was 80 years old when myasthenia was first diagnosed in October 1964. At first the disease was managed by small doses of ambenomium, but by March 1965 the doses had been increased to 10 mg seven times a day. In October 1965 a roentgenogram of the chest showed evidence of a poorly defined mass in the anterior mediastinum, which was interpreted as probable thymoma. (Reproduction of this roentgenogram was unsatisfactory and it is not included in this report.) At this time the patient was taking ambenomium, 10 mg four times a day and prostigmine 15 mg three times a day. Between October and November 1965, the mass was irradiated to a total of 6,000 r to the skin, 4,000 r at 7 cm depth, with the 1 MeV generator. By February 1966 the patient was receiving the same amount of medication as before, but the neurologist reported that symptoms were "fantastically better."

CASE 8.—In this case the patient was treated by one of us (F.B.) at the Swedish Hospital, Seattle.[2] It is included because it suggests the risk of initial doses that are too high in the treatment of this disease.

In December 1939, myasthenia gravis was diagnosed in a 56-year-old woman. Symptoms could not be relieved despite large doses of drugs. The disease progressed rapidly and in February 1940 the patient was admitted in very poor condition and treated immediately with a skin dose of 300 r over a 10×10 cm anterior mediastinal field with 250 KvP. The patient died suddenly 20 hours later from acute cardiocirculatory failure. At necropsy a thymoma was found. The sudden death may well have been attributable to the initial large dose of radiation.

Discussion

Table 1 summarizes the pertinent factors in the seven cases presented. Six of the seven patients were women, a somewhat abnormal distribution according to previously recorded series.[17] In addition, all but one were of advanced age. Four had a thymic neoplasm, proved microscopically in two; in two, no neoplasm could be recognized, and one had probably a small-cell carcinoma of the lung with pseudomyasthenia. Myasthenia of the generalized form was moderate to severe in all. Except in Case 4, the disease had been present for less

TABLE 1.—*Data on Seven Patients with Myasthenia Gravis Treated by Irradiation of Thymus*

Case	*Sex*	*Age at Onset*	*Severity*	*Year Treated*	*Thymic Tumor*	*Type of Operation*	*Radiotherapy Dose to Thymus (r)*	*Initial Response†*	*Present State*
1	F	45	2+	1955	Yes	Biopsy	4,000/ 1,000	B	E tumor
2	F	53	1+	1957	Yes	Biopsy	3,500	B	E tumor
3	F	54	3+	1959	No	None	3,600	C	C
4	F	50	1+	1961	Yes	None	3,900	A	A
5	F	26	3+	1962	No	None	3,000	B	B
6*	F	33	1+	1965	Yes	Biopsy	5,500	A	C
7	M	80	2+	1965	Yes	None	4,000	B	B

*Probable pseudomyasthenia.

†Response: A=Complete remission; no medication required. B=Marked decrease in requirements for medication and/or great improvement in symptoms on same medication. C=No change in medication or symptoms. D=More severe myasthenia gravis. E=Death due either to tumor or myasthenia gravis.

than two years. Of importance is the definite improvement in symptoms and reduction in drug dose after radiation therapy in six of the seven patients—with and without thymoma. Remission in one was complete and continued so at last report. One patient received no benefit from the therapy and the two with thymomas and evidence of extensive invasion and pleural seeding died of the tumor. Radiation therapy in this small group, therefore, appeared to affect the course of the disease favorably. In the two patients with large mediastinal masses and pleural metastatic growth, the tumor was uncontrollable, however. In Case 4, in which biopsy of the tumor was not carried out, the lesion has been controlled for more than four years.

The results in these patients were compared with those of the larger series in the literature:

Schwab,[19] reporting on a large group of patients treated by drugs only, stated that complete remissions can be expected in about 10 per cent during observation. An additional 11 per cent will show pronounced improvement. Schwab further stated that surgical resection of the thymus results in an even greater incidence of these two responses and is of particular benefit in females younger than 30 years with short duration of disease. The use of preoperative radiation therapy in patients with myasthenia gravis without evidence of tumor has been advocated by Schwab.[19] He expressed the belief that a higher incidence of permanent remissions and good responses is obtained when roentgen therapy precedes the surgical procedure. On the other hand, Henson, Stern and Thompson[8] stated that preoperative radiation therapy in patients without tumor is not indicated, since it is not logical to remove a thymus made atrophic by radiation therapy. They further stated that combinations of radiation and surgical treatment may cause a lower rate of complete remission than surgical treatment alone.

Radiation therapy alone in patients without associated tumors appears to be of some benefit. Of 25 patients reported by Robbins,[18] who were treated by radiation alone, one had complete remission, 17 had improvement and seven were not benefited. Robbins said that after radiation therapy the gland will be found to be normal or involutional if operation is performed. Osserman[16] reported that radiation therapy consisting of doses of 600 to 2,000 r in 34 patients without thymoma resulted in moderate to pronounced improvement in 37 per cent. Remission in one of these was complete and six died. Osserman recommended that in patients without thymoma whose course was downward and who were unsuitable for surgical procedure, irradiation was definitely indicated. Glaser[6] observed a remarkable change in drug responsiveness and in general symptoms after radiotherapy in three of six patients who did not have thymomas and in whom myasthenia was drug-resistant. A fourth had transitory improvement and the other two were not benefited.

Grob[7] reported improvement in 40 per cent of 40 patients without thymoma who had irradiation to a dose of 2,000 r. These results were not considered statistically better than for patients treated medically only. However, the death rate was lower than for the medically treated patients even though the disease in the ones selected for radiation therapy was, in many instances, rapidly progressing. Thus, it appears that in patients with myasthenia not well controlled by drug therapy and not meeting the criteria for operation (sex, age and duration of disease), radiation therapy to the thymus should be attempted. Schwab[20,21] now advises that the nontumor patients should be given about 3,000 r to the anterior mediastinum, after which about 50 per cent will probably show remission. The favorable response of one of two patients reported in this series agrees with this concept.

The place of radiation therapy in the management of myasthenia gravis associated with tumor is more definite. The results reported by Keynes[12] indicated that the incidence of survival was considerably higher if the tumor was irradiated preoperatively. The technique used in these patients has been described by Williams.[26] A minimum tumor dose of 4,000 r was given in four weeks with 1 MeV irradiation. Williams[26] and Jones and coworkers[10] stated that therapy should be started slowly, at 50 to 100 r per day, with careful observation of the patient to avoid precipitation of a crisis. The validity of this precaution seems to be demonstrated in one of the cases reported herein (Case 8), in which the patient died less than 24 hours after an initial dose of 300 r. All of the other patients in the present series received initial skin doses of 100 r or less, usually 50 r. The first week's tumor dose was 170 to 500 r in these patients. Keynes[12] stated that 15 of 20 patients who had radiotherapy before excision of the thymus and tumor were alive, four with complete remission. The fact that radiation therapy and complete careful excision of the thymus and tumor bring about complete remission in only 25 per cent of patients indicates that the thymus is not the only source of the disease.

Williams also reported on three cases in which irradiation alone was used in the treatment of patients with thymomas. One of these patients had discontinued all medication after six months and was still improving three years later. A second patient did well for one year, then the symptoms recurred. After retreatment with 1,000 r it was again possible to reduce the drug dose. A third patient with a large tumor and metastatic lesions responded to the extent that reduction of drug dose was possible. Schwab[21] stated that approximately 25 per cent of the patients treated by radiation alone will respond well when a thymoma is present. Perlo, Schwab and Castleman[17] noted that preoperative or postoperative irradiation in patients treated surgically increased the average postoperative survival from 1.4 to 4.6 years. The overall results for patients with excision of thymomas were much poorer than for those without thymoma who were operated on. Only 37 per cent of the thymoma patients were living, compared with 87 per cent of those without thymoma.

The results of surgical treatment alone in myasthenia gravis associated with thymoma are poor in other series. Survival of only one of five was reported by Hermann and coworkers[9] and five of 15 reported by Grob. The results of Lattes[14] were somewhat better, although only five of 18 patients improved after operation, and eight died. When these results are compared with those of Keynes[12] and of Perlo, Schwab and Castleman, certain indications become evident. These are that all patients with thymoma and myasthenia gravis should receive preoperative radiation therapy and that the elderly and other patients responding well can be managed by radiation therapy alone. It is stressed that these tumors consist always of a mixture of lymphoid and epithelial elements, as described by Castleman, Viets and Schwab.[4] The initial rapid decrease in the tumor secondary to lymphocyte death should, therefore, not be interpreted as great radiosensitivity of the tumor. To control the epithelial elements, doses to the limit of tolerance will most likely be required. Thymomas, therefore, should be treated with doses of 5,000 r or more, and when invasion and extension are present, a minimum of 6,000 r tumor dose is desirable.

In the present series the patient whose condition was considered to represent pseudomyasthenia (Case 6) was included because of the great similarity in response to drug medication and the relief of symptoms after radiation therapy. Most likely this case was one of myasthenic syndrome associated with bronchial neoplasm, as described by Lambert and coworkers,[13] since the characteristics of muscle wasting and of small-cell carcinoma were similar to those in their patients. This case is unusual, however, in that none of their patients was a female this young.

Once a patient with myasthenia gravis is selected for radiation therapy, the following procedures should be followed. Since the thymus can extend as high as the isthmus of the thyroid and as low as the fourth intercostal space, the area must be included in the treatment field. Therapy must begin slowly with daily doses for the first week of less than 100 r to the skin and then be increased slowly. Patients without evidence of tumor should be given about 3,000 r to the thymus. Patients with tumors will require doses of between 5,000 r and 6,000 r and the same care in initiating treatment. Treatment in patients without tumor is possible through a single anterior mediastinal field; however, in patients with tumor, multiple fields must be irradiated.

Radiation therapy, therefore, is an important part of the management of myasthenia gravis. Patients with tumor should certainly be given radiation either preoperatively or as definitive treatment. Patients without tumor, who are not suitable for primary surgical procedure, should also be considered for raditaion therapy if not managed well on drug therapy. The evidence in the literature and the reports of our seven patients support these views.

REFERENCES

1. Aring, C. D.: Treatment of myasthenia gravis with the Roentgen ray, Ohio Med. J., 39:241-243, March 1943.

2. Aronson, S. F.: Myasthenia gravis; discussion, with presentation of case associated with thymoma, Ann. Int. Med., 15:137-145, July 1941.

3. Blalock, A., Harvey, A. M., Ford, F. R., and Lilienthal, J. L., Jr.: Treatment of myasthenia gravis by removal of thymus gland; preliminary report, J.A.M.A., 117:1529-1533, 1 November 1941.

4. Castleman, B.: Pathology of the thymus gland in myasthenia gravis, In Viets, H. R., and Schwab, R. S.: Thymectomy of Myasthenia Gravis, Charles C Thomas, Springfield, 1960, pp. 51-66.

5. Effler, D. B., and McCormack, L. J.: Thymic neoplasms, J. Thor. Surg., 31:60-82, January 1956.

6. Glaser, G. H.: Crisis, precrisis and drug resistance in myasthenia gravis, Ann. N.Y. Acad. Sci., 135:335-345, 26 January 1966.

7. Grob, D.: Course and management of myasthenia gravis, J.A.M.A., 153:529-532, 10 October 1953.

8. Henson, R. A., Stern, G. M., and Thompson, V. C.: Thymectomy for myasthenia gravis, Brain, 88:11-28, 1965.

Hypercholesteremia

Treatment with Cholestyramine, a Bile Acid Sequestering Resin

H. R. CASDORPH, M.D., PH.D., *Long Beach*

■ *The results of administration of cholestyramine to 19 patients for periods of two to 21 months are reported.*

All patients consistently taking the drug obtained a significant reduction in the serum cholesterol. The average reduction was 26 per cent and the range was from 16 to 52 per cent.

Cholestyramine acts as a bile acid sequestering resin, causing an increased loss of bile salts (cholates) in the stool. The drug acts by increasing the normal excretory pathway for cholesterol.

AN EFFECTIVE AGENT with which to treat hypercholesteremia has been sought after for a number of years. In recent years, as the undesirable effect of hypercholesteremia has become an accepted fact, renewed efforts have been made to find an agent which would effectively lower the plasma cholesterol without producing significant undesirable effects.

During the past three years we have accumulated experience with the administration of cholestyramine (Questran®) to patients with hypercholesteremia. Practically all of these patients have also had some manifestation of vascular disease, and in most instances this was arteriosclerotic heart disease.

Cholestyramine is a quaternary ammonium anion exchange resin with a polystyrene polymer skeleton. As the chloride salt, it binds bile acids, exchanging chlorides for bile acids.[4] With the increased loss of bile acids in the stool, there is a reduction in the serum cholesterol.[3] Cholesterol of the blood has been shown to be converted to bile acids appearing in the bile.[1] In studies on rats, Carbon-14 labeled cholestyramine was not absorbed from the gastrointestinal tract.[2]

Present Study

The present report deals with a review of 19 patients who took anhydrous cholestyramine at a dose of 8 to 12 gm per day for one month or more.

The average or usual adult dose recommended by the manufacturer is 12 gm of cholestyramine daily. (Some of the patients in the present series, either because of the occurrence of constipation as a side effect, or because of the taste of the medication, did not wish to increase the dose beyond 4 gm a day. For the purposes of this report these patients are not included, since the dosage did not approach the usual adult dose. It should be noted, however, that all of the patients, even those taking only 4 gm per day, did have a significant reduction in serum cholesterol.)

Results

Clinical information and results of this study are shown in Table 1.

The duration of therapy ranged from two to 21 months, and the range of reduction of the serum cholesterol was 16 to 52 per cent with a mean reduction of 26 per cent. The mode of administration was as follows: Each patient initially was given 4 gm a day, and the amount was gradually increased to a dose that would return the cholesterol

---

Submitted 5 May 1966.

Reprint requests to: 1703 Termino Avenue, Suite 6, Long Beach 90803.

TABLE 1.—*Clinical Data and Results in 19 Patients Taking Cholestyramine for Hypercholesteremia*

			Serum Cholesterol (mg per 100 ml)*					
Case	*Age*	*Sex*	*Control Average*	*Treatment Average*	*Per Cent Reduction*	*Dose Grams Per Day*	*Duration of Therapy (Months)*	*Side Effects*
1	37	M	461	337	26.9	12	20	None
2	58	M	312	264	16.0	8-12	21	None
3	67	F	284	232	20.1	8	18	Moderate constipation
4	63	F	293	230	21.5	8	18	None
5	73	M	274	223	18.8	8	13	None
6	69	F	300	212	26.0	8	6	None
7	71	F	543	261	51.8	8	13	Mild constipation
8	74	F	281	206	26.7	8	15	None
9	66	F	424	307	27.6	8-12	15	None
10	71	M	323	252	22.0	8	3	? heartburn
11	71	F	308	193	49.5	12	5	Mild constipation
12	75	F	426	357	16.2	8	5	None
13	76	M	286	208	27.2	8-12	14	None
14	72	F	325	249	23.3	8	12	Mild upset stomach (nausea)
15	78	M	293	226	22.9	8	11	Mild constipation
16	65	M	306	250	18.2	12	10	None
17	77	F	355	250	29.7	8	4	None
18	72	F	380	286	24.7	8	4	None
19	63	F	266	206	22.4	12	2	None

Reduction Range: 16 to 52 per cent. Average Reduction: 26 per cent (25.8 per cent).

*Determined by modified Lieberman Burchard Method (by HYCEL, INC.).

Error of Method: Average error on duplicate cholesterol determinations in this laboratory less than 1 per cent (Range 0-3 per cent).

Classical t-test applied to results here reported indicates a significant reduction in cholesterol ($P<.001$).

to within the normal limits of 150 to 250 mg per 100 ml of serum or until the maximal dosage was achieved which could be tolerated by the patient. Approximately one-third to one-half of the patients taking the drug experienced some mild constipation when administration was started. This was usually easily controlled and frequently the constipation disappeared with continued administration of the drug. Patients reporting no side effects at the time of reaching the full dosage are recorded as having no side effects.

Other possible side effects, such as interference with the absorption of calcium or other administered drugs, are under observation and will not be considered in this presentation.

Experimental Design

Table 2 gives greater detail regarding the individual cholesterol determinations, before and after treatment, that were used to calculate the average cholesterol level.

Cholesterol determinations before treatment

TABLE 2.—*Serum Cholesterol Determinations (mg per 100 ml)*

	Before Treatment		*After Treatment*	
Case		*Average*	*Average Per Cent*	
1	510, 426, 446	460	337 (26.9)	328, 310, 317, 340, 350, 286, 298, 392
2	292, 332	312	264 (16.0)	233, 240, 286, 266, 397, 266, 259, 252
3	273, 292, 286	284	232 (20.1)	249, 246, 200, 233
4	281, 306, 292	293	230 (21.5)	240, 212, 240
5	304, 286, 262	274	223 (18.8)	246, 179, 233, 233
6	306, 293	300	212 (26.0)	212
7	585, 500	543	261 (51.8)	252, 273, 259
8	306, 256	281	206 (26.7)	186, 200, 233, 206
9	412, 433, 426	424	307 (27.6)	292, 306, 313, 320, 306
10	320, 326	323	252 (22.0)	252
11	323, 292	308	193 (49.5)	193
12	419, 452, 406	426	357 (16.2)	320, 372, 372, 367
13	291, 280	286	208 (27.2)	219, 200, 186, 240, 219, 186
14	296, 320, 360	325	249 (23.3)	246, 252
15	280, 306	293	226 (24.9)	226, 226
16	292, 320	306	250 (18.2)	252, 259, 240
17	356, 354, 419, 292	355	250 (29.7)	259, 240
18	379, 400, 360	380	286 (24.7)	286
19	280, 259, 266, 273, 252	66	206 (22.4)	206

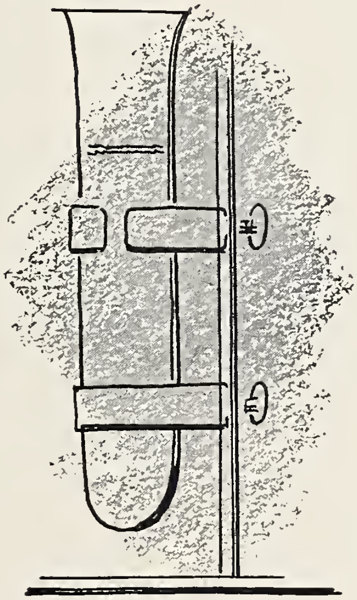
were generally obtained over a period of several months. In most cases past data on cholesterol levels as determined at roughly annual intervals for two or three years before treatment, were also available. However, generally only the cholesterol data obtained during the 12 months before therapy was begun were included and considered significant.

After the beginning of therapy, serum cholesterol determinations were generally obtained at one- to two-month intervals. In some instances only one cholesterol determination is shown, it being the only value obtained at a given level of dosage of the drug. In all cases, additional cholesterol determinations had been obtained at a lower level of drug dosage.

Cholestryamine, supplied as Questran® by Mead Johnson Co., Evansville, Indiana.

REFERENCES

1. Black, K., Berg, B. N., and Rittenberg, D.: The biologic conversion of cholesterol to cholic acid, J. Biol. Chem., 149: 511-517, 1943.
2. Gallo, D. G., and Sheffner, A. L.: The disposition of orally administered cholestyramine-C^{11}, Proc. Soc. Exp. Biol. Med., 120:91-93, 1965.
3. Hashim, S. A., and Van Itallie, T. B.: Resin therapy for hypercholesteremia, J.A.M.A., 192:289-293, 26 April 1965.
4. Tennent, D. M., Siegel, H., Zanetti, M. E., Kuron, G. W., Ott, W. H., Wolf, F. J.: Plasma cholesterol lowering action of bile acid binding polymers in experimental animals, J. Lipid Res., I:469-473, October 1960.



Histamine—The View Today

GILDON N. BEALL, M.D., *Los Angeles*

THE DISCOVERY OF THE widespread distribution of histamine in animals and its intense pharmacologic activities antedated by some time the current scientific explosion, and yet documentation of histamine's functions (if any) in the body is still lacking. Progress has been made in a number of areas, however. It has seemed clear for some time that histamine is involved in many hypersensitivity processes, and recent work has served both to clarify and to make more complex this knowledge. The possible importance of histamine as a mediator of inflammation and vascular permeability has been the subject of new research work. New information about histamine metabolism has been acquired and there now appears to be, for the first time, the possibility that measurement of an excreted histamine metabolite will provide an index of histamine release in the body.

Substances that will inhibit the formation and release of histamine have also been studied. The results so far have been somewhat disappointing but future developments may be more interesting.

These matters will be discussed with a background of the earlier knowledge about histamine. Several excellent monographs and reviews are available* for those who want to read further in the subject.

Histamine Formation

Histamine, betaimidazolethylamine, was first discovered in extracts of decaying organic material. In these circumstances histamine is formed from the amino acid *histidine* by bacterial decarboxylation. This same phenomenon probably accounts for the rather large amounts of histamine that can be found in decaying fish and in the guts of mammals, including man. There has been some controversy in the past about the possible physiologic role of the histamine formed by bacteria in the gut, since many investigators felt that histamine could be absorbed from the gastrointestinal tract and contribute to bodily physiologic processes. There is now little support for this view. It appears that even if histamine were absorbed from the gastrointestinal tract it would be cleared by the kidney with exceeding rapidity.

It has been shown that there are at least two different types of mammalian histidine decarboxylating (HD) enzymes.[75] A specific HD enzyme has been found in mast cell tumors and in a transplantable rat hepatoma.[46] One human mast cell tumor has been studied and found also to possess this specific histidine decarboxylase activity.[7] The specific enzyme uses only histidine as a substrate and catalyzes its conversion to histamine. The second enzyme is a non-specific aromatic amino acid decarboxylase. It will catalyze the decarboxylation of a number of other aromatic amines including dihydroxyphenylalanine (DOPA) to dopamine and 5-hydroxytryptamine to serotonin. The specific histidine decarboxylase is not much inhibited by such compounds as alphamethyldopa, while the aromatic amino acid decarboxylase is very sensitive to this agent. The non-specific enzyme may have little to do with histidine formation *in vivo*.

Specific HD may be present either in mast cells or several other tissues. In the mast cells, the histidine that is formed is bound and stored so the distribution of mast cell histidine decarboxylase in tissues parallels the distribution of histamine. This is not true of non-mast cell histidine decarboxylase. There are also considerable differences between different animals in the distribution and relative importance of mast cell and non-mast cell

---

Reprint requests to: Department of Medicine, UCLA School of Medicine, Los Angeles 90024.

*Reference Nos. 12, 53, 59, 69, 79, 82.

enzymes. In the rat, the stomach is rich in non-mast cell specific HD enzyme. Fetal rats appear to have more of this activity. Kahlson and coworkers[34] have used this type of finding to support their hypothesis that histamine forming capacity parallels tissue growth and repair. This will be considered further later.

The amount of histidine actually converted to histamine in humans is unknown. Rats convert about 1 per cent of their dietary histidine to histamine. Histamine formation seems to be increased at certain times in fetal development of some animals. Schayer has found that "stress" will increase the formation of histamine from histidine.[62] Most of these studies have been performed in rodents and the stresses that have been used included such things as cold, burns and treatment with histamine-liberating drugs and bacterial products. Such induced histamine formation can be inhibited by corticosteroid administraton. These findings led Schayer to a comprehensive hypothesis of the role of histamine in blood vessel regulation which will be discussed later.

Increased histamine formation has also been found in exercising or oxygen-deprived rodents[23] and after injection of adrenalin into kittens—perhaps further indication of the role of "stress" in inducing histamine formation.

A curious report that quartz particles would decarboxylate a certain amount of histidine *in vitro,* suggests that this may have some bearing on the pathogenesis of pneumoconiosis.[47] These interesting studies should be confirmed with some of the more specific methods for measurement of histamine formation that are now available.[40]

A number of recent studies have appeared of compounds which have inhibitory activity for HD.[57,58] It has been difficult to determine whether or not such inhibition interferes with hypersensitivity or inflammatory processes in intact animals, partly because of the small supplies and toxicity of some of the potential HD inhibitors. Those that were used did not inhibit or prevent anaphylaxis in guinea pigs.[6,48]

Corticosteroids will decrease the amount of histamine in some rodent tissues. This may be due to inhibition of histidine decarboxylation, explaining Humphrey's observation that corticosteroids would decrease the fatality rate from anaphylaxis in guinea pigs.[29]

Some clinical trials of HD inhibitors have been made in patients with systemic mastocytosis. The results are as yet uncertain.

Histamine Binding

Following the formation of histamine in the body, some of it is bound and stored in an inert form in basophil or mast cell granules. The proportion of histamine so bound is not known. It seems likely that histamine formed in or near the mast cells would be more likely to be stored. Histamine is probably bound to heparin in the granules.[39] Furano and Green[22] demonstrated that rat mast cells *in vitro* will take up reasonably large amounts of histamine from solution. This suggests that mast cells may not synthesize all their own histamine but some may be manufactured elsewhere.

A different type of histamine binding has been proposed to explain the inhibitory activity that serum proteins exert on histamine-induced smooth muscle contraction. The extent of this inhibition varies with different sera. A deficiency of this histamine-inhibiting or "histaminopexic" effect of serum has been proposed as a concomitant of the allergic state or "terrain."[54] Deficiency of the histaminopexic action of serum, however, is extremely non-specific.[15] Sera from patients with a number of other diseases besides those that are known to have immunologic mechanisms have deficient "histaminopexy." It also seems fairly clear that the inhibition of the smooth muscle contraction is not due to binding of histamine to serum proteins, since such binding cannot be demonstrated by equilibrium dialysis or gel filtration.[4]

Histamine Measurements and Distribution

A number of different procedures have been used to assay histamine in tissues, blood and urine. There has been some concern about specificity with most of the methods. Most laboratories are now using some modification of a fluorometric method.[63] This method seems to be adequate for most tissues but it has been suspected that brain tissue may contain non-specific interfering materials. In humans relatively large amounts of histamine, 20 to 30 mcg per gram of tissue, are found in lungs, mucous membranes and facial skin.[83] Intermediate amounts, 15 mcg per gram of tissue, are found in the upper gastrointestinal tract. Small amounts, 2 to 7 mcg per gram of tissue, are found in most of the rest of the viscera. The amounts of histamine found in the blood are quite variable, ranging from 20 to 90 mcg per liter, depending upon the number of basophils present. Plasma content of histamine, on the other hand, is low and relatively constant at around 5 mcg per liter. Measurements of plasma histamine are at the limits of sensitivity of the fluorometric method, and it may be that no free histamine is actually present in the plasma.

There has been continued interest in the possible role of histamine as a neurohumor. Histamine can be formed in brain tissue, and small amounts are found in many areas of the brain.[78] Histamine remains a suitable candidate for a role as a neurohumor.

It is interesting and probably significant that in humans the nose and lungs, prime shock organs in allergic disorders, contain large amounts of histamine. Guinea pigs also have large quantities of histamine in their anaphylactic shock organ, the lung. The severity of anaphylaxis has been shown to be related to the lung histamine content in inbred strains of guinea pigs.[67] Guinea pig tissue histamine content can be reduced by adrenal corticosteroids.[41] It is possible that the effects of these drugs in humans, particularly the effects on asthma, may be related to reduction of tissue histamine content presumably by inhibiting HD activity.

Histamine Release

Mast cells and basophils either synthesize or accumulate histamine and store it in an inert form for subsequent release. A number of drugs and chemical substances will release histamine from mast cells. The amount that is released and the sensitivity of mast cells to various releasers depends to a large extent on the tissue and the species that is studied.[55] In general, the materials that are most effective as histamine releasers are polyamines or strongly basic compounds with similar structure. Morphine and other opium alkaloids, d-tubocurarine and stilbamide, are the most potent of a large number of drugs which can act as histamine releasers in man.

Chemically mediated histamine release in humans has provided some useful information about human allergic diseases. The administration of histamine-releasing drugs to patients with urticaria tends to reproduce the disease.[31] This finding adds support to the concept that histamine is the important mediator of human urticaria. Some tissue extracts, such as extract of lobster muscle, will cause histamine to be released from animal preparations in amounts that might be associated with profound anaphylactoid symptoms in man.[56] This suggests that some of the reactions seen after the ingestion of shellfish and possibly other foods may be caused by an abnormal release of histamine and not by an antigen-antibody reaction.

How these chemicals act to release histamine is not clear. In a study of the release of histamine from rat mast cells, Bray and VanArsdel[9] found so much variation in the effects of various histamine liberating agents that they concluded that more than one mechanism of action was involved. Some evidence has been obtained that the susceptibility of mast cells to the effects of one histamine liberator, dextran, may be genetically determined in the rat.[76]

The release of histamine in anaphylaxis is by now a well known phenomenon. The chemical process of this reaction is still being explored in a number of *in vitro* anaphylactic systems.[2] It now appears that a number of paths may lead to the final common act of releasing histamine from sensitized cells but activation of a proteolytic enzyme seems to be a portion of all these processes. The concept of anaphylotoxin has been revived to explain some of the newer findings in this field. Anaphylotoxin as originally described was an unknown component of prepared rat serum which would produce histamine release. Anaphylotoxin is now felt to be an immune complex consisting of the first five components of the complement system.

Histamine can be released during *in vitro* anaphylactic reactions with rabbit platelets, guinea pig lung, rat peritoneal mast cells, human leukocytes, human lung and human skin. Some of these systems require participation of complement and others do not. The release of histamine is apparently usually mediated by an esterase related to but different from the esterase (C'_{1a}) which initiates the participation of complement in immune hemolysis.

Histamine-releasing techniques with human leukocytes have been used in an attempt to assay human allergic antibody (skin-sensitizing antibody, P-K antibody or reagin) in a manner more flexible and accurate than the usual skin test. Such methods have also shown some promise in evaluating the results of hyposensitization therapy.[42,72]

The hypothesis that anaphylactic histamine release from rat mast cells is induced by a lecithinase A, has not been widely accepted.[28] Since a number of different types of histamine release systems have been demonstrated, this possibility still exists. The product of the action of lecithinase A on lecithin, lysolecithin, will release histamine in human skin.[49]

Histamine Metabolism

Much of the original work on *in vivo* histamine catabolism was done by Schayer in the 1950's and reviewed by him in 1959.[60] He pointed out that there were two main pathways of histamine catabolism *in vivo*. In the first, histamine can be oxidized to imidazoleacetic acid (ImAA) and then coupled with ribose to form imidazoleacetic acid riboside. The second pathway consists of a preliminary methylation of a ring nitrogen to form 1,4-methylhistamine. This compound is oxidized at the side chain to form 1,4-methylimidazoleacetic acid (1,4-meImAA). This methylation pathway seems to be of primary importance in man, since most of the radioactive label of injected histamine-C^{14} is excreted as 1,4-meImAA or 1,4-methylhistamine. All these catabolic products are relatively inert pharmacologically.[3,26] Alivisatos[1] showed that histamine can interact with pyridine coenzymes to form histamine adenine dinucleotide. The significance of this reaction is not known.[1]

The methylation of the ring imino-nitrogen of histamine is accomplished by a histamine methylating enzyme which has been found in most tissues.[10,45] *In vivo* inhibition of the enzyme in mice has resulted from administration of chlorpromazine, serotonin and bufotenine.[65] Any consequences of this inhibition are not known. Oxidation of the side chain of histamine to ImAA or 1,4-methylhistamine to 1,4-meImAA can be catalyzed by amineoxidases. There is a good deal of controversy about the exact nature of this amineoxidase activity. It is uncertain whether both oxidations can be catalyzed by the same enzyme or whether there are specific enzymes for each. Inhibitor studies suggest the latter. Another controversy involves a discussion as to whether there is a specific amineoxidase, histaminase, or whether this enzyme is a non-specific diamine oxidase.[32,84]

Histamine-C^{14} is very rapidly removed from the blood by the kidney.[27] There is also a fair degree of extraction of histamine by the liver.

Measurement of the excretion of histamine or its catabolites might give information about the release of histamine in various diseases. Histamine has been measured in human urine with this intent. Since only about 1 per cent of injected histamine is excreted in the urine normally, urine histamine is not likely to represent a significant amount of the histamine released in the body. Added to this have been the methodological problems of many studies. Ion exchange methods are necessary to separate histamine from other materials in the urine so that appropriate assays can be done. The capacity of the ion exchange materials for histamine varies depending upon the salt content of the urine. Consequently, internal histamine standards added to each urine sample are necessary.[51] This has not always been done.[18] Most investigators are in agreement that healthy men excrete from 15 to 90 mcg of histamine in the urine per day. The mean is around 40 mcg per day. Some conflicting results have been obtained, possibly because of the methodological problems mentioned.

Histamine in the urine increases after a meat meal, indicating that bacterial decarboxylation of histidine in the gut results in absorption of histamine, which is then excreted.[30] I found a few patients who had an increase in urinary histamine after an asthmatic attack induced by inhaling pollen, but the results of this challenge were not consistent.[5]

Histamine in the urine has been found to be very greatly elevated in patients with systemic mastocytosis and in an unusual type of carcinoid syndrome associated with metastatic gastric carcinoid.[17,52] In no other situation has the determination of histamine in the urine been of any particular value.

Measurement of the specific metabolites of histamine in the urine should give more information about histamine release in the body. ImAA and its riboside are normal products of histidine metabolism, so they cannot be expected to give useful information about histamine release and catabolism. Methylhistamine assay should provide some information about *in vivo* histamine metabolism. An assay method has been reported with normal excretions of 140 to 480 mcg per 24 hours.[21] Sensitive techniques for quantitative determination of 1,4-meImAA, using thin layer and gas chromotography, have recently been reported.[24,70] Tham found that humans excrete in the urine 1 to 2.8 mcg of 1,4-meImAA per milligram of creatinine.[71] In addition, a variable excretion of 1,5-meImAA was also found. The latter disappeared from the urine when patients were on parenteral feeding, while 1,4-meImAA was not altered. The 1,5-meImAA is apparently of dietary origin, while the 1,4-meImAA was not altered by diet and may be almost entirely derived from the endogenous liberation of histamine. The excretion of 1,4-meImAA is increased in situations thought to be associated with liberation of histamine, such as burns, urticaria pigmentosa, carcinoid and several allergic conditions. These fascinating observations deserve immediate extension to a number of human diseases to see what part the histamine has to play in these disorders. It may be possible with this sort of diagnostic technique to determine if symptoms are produced by allergens. Challenge tests might be followed by measurement of 1,4-meImAA in the urine as an index of the response to the challenge. It will then be possible to tell if previous findings, such as those of Kerr[35,36] of increased urocanic acid and depression of 1,4-meImAA in the urine of patients with status asthmaticus, can be confirmed.

Histaminase in Pregnancy

Increased histamine catabolizing activity has been found in the plasma of pregnant women.[44,66] This has generally been attributed to increased histaminase or diamine oxidase activity, and its source is most likely the placenta, which contains such an enzyme.[33] Failure to find any significant difference in histamine catabolism between the pregnant and the non-pregnant despite this remarkable activity has cast some doubt upon its importance *in vivo.*

Histamine Distribution and Metabolism as Related to Sex

Differences in the handling of histamine by the two sexes have been observed for some time. Female rats metabolize less and excrete more histamine than males.[77] Castration of male rats increases their free histamine output, but ovariectomy and hysterectomy have no effect on the output of histamine by females. Testosterone given to castrated male rats suppressed the output of histamine but did not affect the normal female rats unless given in large amounts. Maleness or testosterone may produce these effects by increasing histamine methylation. The function of histamine in sexual tissues has been explored by Szego and Gitin,[68] who described histamine depletion in ovaries during the hyperemic response to luteinizing hormone, suggesting that histamine is of fundamental importance in mediating the action of the hormone.

The only work connecting sexual hormones and histamine in humans has demonstrated an increased methylation of histamine in females who were having increased estrogen secretion due to follicle stimulating hormone or chorionic gonadotrophin.[25] The apparent conflicts between these findings and those in rats cannot be resolved at present since different studies suggest that histamine methylation may be stimulated by both androgens and estrogens.

Pharmacologic and Physiologic Effects of Histamine

Despite the widespread occurrence of histamine in mammals, no physiologic function has yet been proved. There is no lack of theories, however. Most of these are based on the known pharmacologic effects of histamine. Histamine, of course, produces widespread contraction of smooth muscle and increases vascular permeability. When injected into the skin it produces the complete "triple response" as originally described by Lewis and Grant[43]—localized erythema, edema and an erythematous flare.

In humans, intravenous infusion of small amounts of histamine results in facial flushing and tachycardia.[74] Larger amounts will produce hypotension due to a decrease in peripheral arterial resistance. Headache is easily produced, apparently as a result of increased pulsations of cerebral arteries, in the recovery phase after the hypotension. Inhalation of histamine will produce slight bronchial obstruction in normal lungs.[8] It has been known for a long time, of course, that patients with asthma are very susceptible to histamine, so that injection or inhalation of small amounts produces severe, albeit reversible, bronchial obstruction.[16] This reaction is not entirely specific, since patients with chronic bronchitis and emphysema and chronic allergic rhinitis without pulmonary disease may have an intermediate sensitivity to histamine lying between that of patients with asthma and normal individuals.

Injection of histamine into man also produces decided gastric acid secretion. Oral administration produces few effects unless truly massive amounts are ingested. A number of other effects have been seen in various animals, but not proved in man. The interested reader can find reference to these in the reviews cited.

Alteration of the pharmacologic effects of histamine by disease have just been mentioned. Kind[38] discovered that immunization of mice with B pertussis resulted in hypersensitivity to both histamine and anaphylaxis. The explanation for this phenomenon has apparently been discovered by Fishel, Szentivanyi and Talmadge,[20] who showed that histamine metabolism in pertussis-immunized mice is apparently normal and the hypersensitivity to histamine could also be produced by a beta adrenergic blockade. It has been found that beta adrenergic blockade in humans with asthma will decidedly sensitize them to the effects of histamine. Patients with hay fever who are immunized with influenza vaccine have an increased bronchial sensitivity to histamine. This phenomenon may be caused by a pharmacologic effect similar to that of the B pertussis immunization in rodents.

Physiologic Effect of Histamine

Proceeding from the known effects of histamine, a number of theories have been proposed as to the actual physiologic function of the amine. The pronounced ability of histamine to stimulate gastric secretion and the large histidine decarboxylating potential of the stomach in most animals has supported the hypothesis that histamine is an important physiologic stimulator of gastric acid secretion. Now that gastrin has been isolated and purified, the possible role of histamine in gastric secretion seems to be somewhat restricted, yet Code[13] still feels that histamine may be a final mediator of acid secretion.

Histamine has long been proposed as a possible mediator of reactive hyperemia and other changes in the microcirculation, yet little direct evidence for this has been found. Schayer[61] theorized that a balance between the induced synthesis of histamine and adrenal corticosteroid secretion regulates flow in the microcirculation. He feels that tissue histidine decarboxylase stimulated by "stress" increases the formation of histamine, that the latter is immediately active in increasing blood vessel caliber, and that the resulting increased blood flow would then serve to wash away the histamine in the tissue. In experimental preparations in animals, the activity of histamine decarboxylase could be stimulated by a number of "stress" mechanisms and could be antagonized by corticosteroids. In Schayer's view, this induced histidine decarboxylase is the regulator of both normal vascular permeability and the pathologic vascular permeability of inflammation. This theory has some attractive features, not the least of which is the apparent explanation of the action of adrenal corticosteroids. Unfortunately, most of Schayer's experiments have been done in rodents. The catabolism and pharmacologic effects of histamine in rodents are not the same as in humans, so there is a distinct need for confirmatory experiments, particularly in man or other primates. The widespread distribution of other potent mediators of vascular permeability makes it unlikely that induced histamine formation is the only mediator of such permeability, particularly in the process of inflammation.

Another possible physiologic role of histamine has been mentioned previously: The possible growth-stimulating effect of histamine. This theory of Kahlson and coworkers[34] has, again, been largely developed from experiments in rodents. Rapidly growing tissues, fetal tissues and healing wounds have been found to contain more "histamine-forming capacity" than inactive tissues. Hence it is argued that the increased formation of histamine found is intimately connected with these processes of growth and repair. A few contradictory results by other workers and some questions about methods prevent immediate acceptance of this line of reasoning.[11]

Histamine in Human Disease

Although the possible roles of histamine in normal physiology are disputed, there is some agreement about the participation of endogenous histamine in a number of disease entities.

Asthma

In susceptible subjects asthma can be induced by small amounts of histamine. The lungs of asthmatic patients have been shown to release histamine when challenged with specific antigen. The metabolism of histamine in asthma is normal and no elevation of histamine concentration in blood plasma or lung tissue from asthmatics can be found. Some older experiments suggested that histamine might be sequestered in the tissues during asthmatic attacks because urinary excretion of histamine decreased. This has not been confirmed, yet the hypothesis has been revived by Kerr who found an absence of 1,4-meImAA, the principal histamine metabolite, from the urine of patients with status asthmaticus. Since there now are quantitative methods available for the estimation of 1,4-meImAA, it would be interesting to see if this can be confirmed. It seems clear that histamine is an important factor in producing the bronchospasm of asthma.

Anaphylaxis

There are great species differences in the mediation and course of anaphylaxis. The various types of *in vitro* anaphylactic reactions discussed earlier are effective in liberating histamine. This certainly suggests that the histamine liberated must play a role in mediating local or systemic anaphylaxis. The phenomena seen in human anaphylaxis could be produced by liberation of histamine but no direct evidence of this has ever been produced.

Urticaria

A decrease in circulating basophils is frequently seen in patients with chronic urticaria, providing some indirect evidence that histamine has been liberated. These patients usually have evidence of increased susceptibility to histamine and to histamine-liberating drugs. Antihistaminic agents are sometimes effective in relieving the urticaria. For all these reasons it seems likely that histamine is of some importance in urticaria, although it is unlikely that it is the only important mediator. One specific type of urticaria, that due to exposure to cold, has been shown to be associated with very pronounced release of histamine into plasma draining from an area exposed to cold at the time that the wheal is developing. Here the cause and effect relationships seems reasonably clear. It has been held that dermographism is somehow related to mechanical release of histamine in the skin. Some recent studies, however, have failed to demonstrate histamine in perfusates of urticarial areas.[81] These areas were perfused after the wheals had been formed, however, and it is possible that any histamine released was already washed away.

Other Conditions

Histamine has been proposed as a mediator of a number of other pathologic processes. Nadel, Colebatch and Olsen[50] demonstrated that microembolism of the lungs of cats produced terminal respiratory airway constriction which they felt was dependent upon the release of histamine. In cardiopulmonary bypass in man it has been shown that plasma histamine was increased.[14] It is uncertain whether this was contributing to any of the problems seen in these patients. Histamine has been proposed as a mediator of the increased gastric acid secretion resulting in ulcer formation that sometimes follows portacaval shunt. There is disagreement in the literature as to whether plasma or blood histamine in dogs with such a circulatory shunt is increased or whether this increase is in any way correlated with the secretion of acid.[64,80] Another aspect of this problem was brought up by Fischer and Snyder.[19] Using a different animal, the rat, they showed that there is increased histamine formation in all the tissues after portacaval shunt, but the increase is greatest in the stomach.[19] Studies in man might be useful to help solve this problem. Methodological problems and species differences probably contribute to the present contradictions.

Therapeutic Uses and Toxicology of Histamine

Attempts have been made occasionally to use histamine as a vasodilator to improve circulation in threatened areas. These effects are so short lived that they seem to be of little use. Even if one were able to continue infusion of histamine into a compromised vascular bed, it is doubtful that this would greatly increase the perfusion of the ischemic areas. It might well divert blood flow to areas where blood vessels could be easily dilated.

"Desensitization" with histamine has been used in an attempt to treat several diseases, particularly chronic urticaria, cold urticaria and so-called histaminic cephalalgia. It is now generally agreed that histamine headache is a variant type of vascular headache and not a specific disease. Vascular headache of almost any type may be triggered by the intense cerebral artery pulsations which can be induced by histamine injection. Hence triggering of headache with histamine cannot be regarded as a specific effect and there is no evidence that repeatedly doing this is of any benefit to the patient. Injection of histamine into patients with chronic urticaria often results in a flare of the urticaria, as these patients seem to be somewhat susceptible to its effects, but there is no evidence that treatment of this type produces a real change in this disorder which has so variable a pattern. Still another therapeutic effort in the past was to bind histamine to a protein. This material was then used to "desensitize" to histamine. There is no evidence that this treatment had any virtue in the care of allergic diseases.

From the studies on "histaminopexy" the concept has been developed that a deficiency of "histaminopexic" globulin in allergic persons might be remedied by giving globulin or a histamine-globulin complex to these patients. The questionable rationale for this procedure makes it unlikely that it will be an effective therapeutic device. No critical study of this form of treatment has appeared.

Histamine has been used as a test for familial dysautonomia. Intradermal injection of a 1:1,000 solution into the skin of normal persons produced intense pain and a large wheal with surrounding erythema. Patients with dysautonomia had a very limited area of pain and wheal formation and had no flare around the wheal. Histamine has long been used to produce maximal gastric stimulation where it seems to be of some value.

Toxicology

Patients with asthma, of course, may have an untoward reaction to an injection of histamine. Otherwise an overdose of histamine is not likely to occur. One possible exception to this is fish poisoning. Truly huge amounts of histamine may be present in some spoiled fish. Some peculiar reactions are occasionally seen following the ingestion of fish. These usually consist of erythema and shock, manifestations which suggest histamine toxicity. However, the ingestion of huge amounts of histamine by animals has not produced much in the way of symptoms and it is not at all clear that so-called "fish poisoning" in humans is due to histamine which is undoubtedly present in some spoiled fish.[37]

Antihistaminic Agents

Most antihistaminic drugs are structurally related to histamine, consisting of an ethylamine group linked through either nitrogen, carbon or oxygen to some kind of aromatic or heterocyclic nucleus. Antihistaminic drugs will effectively antagonize the effects of infused histamine. They will prevent bronchospasm from inhalation of histamine or histamine-liberating antigens in patients with asthma. On the other hand, they are not very effective in the treatment of asthma once it has developed, although they are of course reasonably effective in urticaria and allergic rhinitis. Thus, the therapeutic potential of these agents has been somewhat disappointing. Antihistaminics also do not have much effect on the vascular permeability of inflammation, a finding that has been used to argue against the role of histamine in inflammation. Schayer showed that histamine formed by stress-induced HD activity is not antagonized by antihistaminic drugs. This seems to fit with the observation that these agents are not effective in asthma after it has begun. Apparently the histamine can be released or formed close to the area in which it acts and is thus insulated from the antihistaminic.

Because It's There

Some years ago, VanArsdel and I reviewed the subject of histamine and completed the review with the question, "Why histamine?"[73] There is still no adequate answer for this. Histamine requires attention and continued study ". . . because it's there."

REFERENCES

1. Alivisatos, S. G. A.: Enzymatic interactions of histamine with pyridine coenzymes, Fed. Proc., 24:769-773, 1965.

2. Austen, K. F., and Humphrey, J. H.: In vitro studies of anaphylaxis, Advances in Immunology, 3:3-86, 1963.

3. Beall, G. N., and VanArsdel, P. P., Jr.: Histamine metabolism in human disease, J. Clin. Invest., 39:676-683, 1960.

4. Beall, Gildon: Is histamine bound by serum proteins?, Int. Arch. Allergy, 25:249-256, 1964.

5. Beall, Gildon: Histamine in human urine, Int. Arch. Allergy, 26:1-17, 1965.

6. Beall, Gildon: Decarboxylase inhibitors and histamine in guinea pigs, Biochem. Pharmacol., 14:1907-1911, 1966.

7. Birt, A. R., Hagen, P., and Zebrowski, E.: Amino acid decarboxylases of urticaria pigmentosa mast cells, J. Invest. Derm., 37:273-276, 1961.

8. Bouhuys, A., Jonsson, R., Lichtneckert, S., Lindell, S-E., Lundgren, C., Lundin, G., and Ringquist, T. R.: Effects of histamine on pulmonary ventilation in man, Clin. Sci., 19:79-94, 1960.

9. Bray, R. E., and VanArsdel, P. P., Jr.: In vitro histamine release from rat mast cells by chemical and physical agents, Proc. Soc. Exp. Biol. Med., 106:255-259, 1961.

10. Brown, D. D., Tomchick, R., and Axelrod, J.: The distribution and properties of a histamine-methylating enzyme, J. Biol. Chem., 234:2948-2950, 1959.

11. Burkhalter, A.: Histamine and growth, Fed. Proc., 24:1341-1342, 1965.

12. Code, C. F., Hurn, M. M., and Mitchell,, R. G.: Histamine in human disease, Mayo Clin. Proc., 39:715-737, 1964.

13. Code, Charles: Histamine and gastric secretion: a later look, 1955-1965, Fed. Proc., 24:1311-1321, 1965.

14. Cooper, T., Jellinek, M., Willman, V. L., Schweiss, J. F., and Hanlon, C. R.: Plasma histamine during cardiopulmonary bypass in man, Arch. Surg., 86:138-142, 1963.

15. Cruchaud, P. A., Girard, J. P., Domine, E., and Micheli, H.: Etude critique du pouvoir de fixation de l'histamine et de la serotonine par le serum sanguin, Int. Arch. Allergy, 19:65-85, 1961.

16. Curry, J. J., and Lowell, F. C.: Measurement of vital capacity in asthmatic subjects receiving histamine and acetyl-beta-methylcholine, J. Allergy, 19:9-18, 1948.

17. Demis, D. J., and Zimmer, J. G.: Histaminuria in mastocytosis, Arch. Int. Med., 111:309-314, 1963.

18. Duner, H., and Pernow, B.: Histamine in man under physiological and pathological conditions, Acta Med. Scand., 168:307-323, 1960.

19. Fischer, J. E., and Snyder, S. H.: Histamine synthesis and gastric secretion after portacaval shunt, Science, 150:1034-1035, 1965.

20. Fishel, C. W., Szentivanyi, A., and Talmage, D. W.: Sensitization and desensitization of mice to histamine and serotonin by neurohumors, J. Immunol., 89:8-18, 1962.

21. Fram, D. H., and Green, J. P.: The presence and measurement of methylhistamine in urine, J. Biol. Chem., 240:2036-2042, 1964.

22. Furano, A. V., and Green, J. P.: The uptake of biogenic amines by mast cells of the rat, J. Physiol., 170: 263-271, 1964.

23. Graham, P., Kahlson, G., and Rosengren, E.: Histamine formation in physical exercise, anoxia and under the influence of adrenaline and related substances, J. Physiol., 172:174-188, 1964.

24. Granerus, G., and Magnusson, R.: A method for

semiquantitative determination of 1-methyl-4-imidazoleacetic acid in human urine, Scand. J. Clin. and Lab. Invest., 17:483-490, 1965.

25. Green, J. P., Fram, D. H., and Kase, N.: Methylhistamine and histamine in the urine of women during the elaboration of estrogen, Nature, 204:1165-1168, 1964.

26. Helander, E., Lindell, S-E., Nilsson, K., and Westling, H.: Catabolism of C^{14}-labeled histamine in patients with allergic diseases, Acta Allergol., 17:86-97, 1962.

27. Helander, C. G., Lindell, S-E., and Westling, H.: The renal removal of C^{14}-labeled histamine from the blood in man, Scand. J. Clin. and Lab. Invest.. 17:524-528, 1965.

28. Hogberg, B., and Uvnas, B.: Further observations on the disruption of rat mesentery mast cells caused by compound 48/80, antigen-antibody reaction, lecithinase A and decylamine, Acta physiol. scand., 48:133-145, 1960.

29. Humphrey, John: The effect of cortisone upon some experimental hypersensitivity reactions, Brit. J. Exp. Path., 32:274-283, 1951.

30. Irvine, W. T., Duthie, H. L., and Waton, N. G.: Urinary output of free histamine after a meat meal, Lancet, i:1061-1064, 1959.

31. Lennart, J., and Rune, C.: Skin reactions to compound 48/80 in normal subjects and patients with urticaria and eczema, Acta Derm. Venereol., 42:11-16, 1962.

32. Kapeller-Adler, R.: Histamine catabolism in vitro and in vivo, Fed. Proc., 24:757-765, 1965.

33. Kapeller-Adler, R.: Recent attempts at the purification and identification of human placental "histaminase," Clin. Chim. Acta, 2:191-193, 1965.

34. Kahlson, G., and Rosengren, E.: Histamine, Ann. Rev. Pharmacol., 5:305-320, 1965.

35. Kerr, James: Identification of urocanic acid in the urine in status asthmaticus, Lancet, ii:709-710, 1963.

36. Kerr, James: Identification of histamine metabolite 1-methyl, 4-imidazoleacetic acid in human urine and its absence in status asthmaticus, Brit. Med. J., ii:606-608, 1964.

37. Kimato, Masao: The histamine problem; from Fish as Food, Vol. 1, Edited by G. Borgstrom, Academic Press, New York, 1961, pp. 329-392.

38. Kind, Leon: The altered reactivity of mice after inoculation with Bordetella Pertussis vaccine, Bact. Rev., 22:173-182, 1958.

39. Kobayashi, Yutaka: Histamine binding by heparin, Arch. Biochem. Biophys., 96:20-27, 1962.

40. Kobayashi, Yutaka: Determination of histidine decarboxylase activity by liquid scintillation counting of $C^{14}O_2$, Anal. Biochem., 5:284-290, 1963.

41. Kovacs, Eva: Changes in tissue histamine content of guinea-pigs following treatment with cortisone and metyrapone, Brit. J. Pharmacol., 24:574-578, 1965.

42. Levy, D. A., and Osler, A. G.: Studies on the mechanisms of hypersensitivity phenomena. XIV. Passive sensitization in vitro of human leukocytes to ragweed pollen antigen, J. Immunol., 97:203-212, 1966.

43. Lewis, T., and Grant, R. T.: Vascular reactions of the skin to injury. II. Liberation of a histamine-like substance in injured skin, the underlying cause of factitious urticaria and of wheals produced by burning. Observations upon the nervous control of certain skin reactions, Heart, 11:209-269, 1924.

44. Lindahl, K. M., Lindell, S-E., Nilsson, K., Schayer, R. W., and Westling H.: Studies on the metabolism of C^{14}-labeled histamine in human plasma during pregnancy, Arkiv f. Kemi, 38:379-384, 1958.

45. Lindahl, Kerstin: The histamine methylating enzyme system in liver, Acta physiol. scand., 49:114-138, 1960.

46. MacKay, D., Riley, J. F., and Shepherd, D. M.: Amino-acid decarboxylase activities in rat hepatoma, J. Pharm. Pharmacol., 13:257-261, 1961.

47. Marasas, L. W.: Formation of histamine and histamine-like substances from histidine by quartz dust, Nature, 192:872-873, 1961.

48. Marschke, G. R., and Beall, G. N.: The effects of two histidine decarboxylase inhibitors in guinea pig anaphylaxis, Biochem. Pharmacol., 14:192-194, 1965.

49. Middleton, E., Jr., and Phillips, G. B.: Release of histamine activity in human skin by lysolecithin, J. Lab. and Clin. Med., 64:889-894, 1964.

50. Nadel, J. A., Colebatch, H. J. H., and Olsen, C. R.: Location and mechanism of airway constriction after barium sulfate microembolism, J. Appl. Physiol., 19:387-394, 1964.

51. Oates, J. A., Marsh, E., and Sjoerdsma, A.: Studies on histamine in human urine using a fluorometric method of assay, Clinica Chimica Acta, 7:488-497, 1962.

52. Oates, J. A., and Sjoerdsma, A.: A unique syndrome associated with secretion of 5-hydroxytroptophan by metastic gastric carcinoids, Amer. J. Med., 32:333-342, 1962.

53. Padawer, Jacques, Editor: Mast cells and basophils, Ann. N.Y. Acad. Sci., 103:1-492, 1963.

54. Parrot, J.-L., Laborde-Burtin, B., and Saindelle, A.: The allergic terrain, Ann. Allerg., 22:511-526, 598-610, 1964.

55. Paton, W. D. M.: Histamine release by compounds of simple chemical structure, Pharm. Rev., 9:269-328, 1957.

56. Paton, W. D. M.: Lobster's lymph and histamine liberation, Int. Arch. Allergy, 15:35-46, 1959.

57. Reid, J. D., and Shepherd, D. M.: Inhibition of histidine decarboxylases, Life Sciences, 1:5-8, 1963.

58. Robinson, B., and Shepherd, D. M.: The inhibition of the L-histidine decarboxylases of guinea-pig kidney and rat hepatoma, J. Pharm. Pharmacol., 14:9-15, 1962.

59. Rocha e Silva, M.: Histamine: Its Role In Anaphylaxis and Allergy, Charles C Thomas, Springfield, Ill., 1955.

60. Schayer, Richard: Catabolism of physiological quantities of histamine in vivo, Phys. Rev., 39:116-126, 1959.

61. Schayer, Richard: Induced synthesis of histamine, microcirculatory regulation and the mechanism of action of the adrenal glucocorticoid hormones, Progr. Allerg., 7:187-212, 1963.

62. Schayer, Richard: Histamine and circulatory homeostasis, Fed. Proc., 24:1295-1297, 1965.

63. Shore, P. A., Burkhalter, A., and Cohn, V. H., Jr.: A method for the fluorometric assay of histamine in tissues, J. Pharmacol. Exp. Therap., 127:182-186, 1959.

64. Silen, W., and Eiseman, B.: Evidence for histamine as the agent responsible for gastric hypersecretion after portacaval shunt, Surgery, 50:213-219, 1961.

65. Snyder, S. H., and Axelrod, J.: Tissue metabolism of histamine-C^{14} in vivo, Fed. Proc., 24:774-776, 1965.

66. Southren, A. L., Kobayshi, Y., Sherman, D. H., Levine, L., Gordon, G., and Weingold, A. B.: Diamine oxidase in human pregnancy, Am. J. Ob. Gyn., 89:199-203, 1964.

67. Stone, S. H., Liacopoulos, P., Liacopoulos-Briot, M., Neveu, T., and Halpern, B. N.: Histamine: differences in amount available for release in lungs of guinea pigs susceptible and resistant to acute anaphylaxis, Science, 146:1061-1062, 1964.

68. Szego, C. M., and Gitin, E. S.: Ovarian histamine depletion during acute hyperaemic response to luteinizing hormone, Nature, 201:628-684, 1964.

69. Telford, J. M., and West, G. B.: Some aspects of experimental allergy, Int. Arch. Allergy, 22:11-30, 29-58, 1963.

70. Tham, Richard: Gas chromatographic analysis of histaminemetabolites in urine. Quantitative determination of ring methylated imidazole acetic acids in healthy man, J. Chromatog., 23:207-216, 1966.

71. Tham, Richard: Liberation of histamine in man. Gas chromatography of ring methylated imidazoleacetic acids in urine, Scand. J. Clin. and Lab. Invest., In Press, 1966.

72. VanArsdel, P. P., Jr., Middleton, E., Jr., Sherman, W. B., and Buchwald, H.: A quantitative study on the in vitro release of histamine from leukocytes of atopic persons, J. Allergy, 29:429-437, 1958.

73. VanArsdel, P. P., Jr., Beall, G. N.: The metabolism and functions of histamine, Arch. Int. Med., 106: 714-733, 1960.

74. Weiss, S., Robb, G. P., and Ellis, L. B.: The systemic effects of histamine in man, Arch. Int. Med., 49: 360-396, 1932.

75. Weissbach, H., Lovenberg, W., and Udenfriend, S.: Characteristics of mamellian histidine decarboxylating enzymes, Biochem. Biophys., Acta, 50:177-179, 1961.

76. West, G. B., and Harris, J. M.: Pharmacogenetics —A fresh approach to the problem of allergy, Ann. N.Y. Acad. Sci., 118:441-452, 1964.

77. Westling, H., and Wetterqvist, H.: Further observations on the difference in the metabolism of histamine in male and female rats, Brit. J. Pharmacol., 19:64-73, 1962.

78. White, T.: Biosynthesis, metabolism and function of histamine in the nervous system, Fed. Proc., 23:1103-1106, 1964.

79. Wilhelm, D. L.: The mediation of increased vascular permeability in inflammation, Pharmacol. Rev., 14: 251-280, 1962.

80. Windsor, C. W. O., Thompson, J. C., and Orloff, M. J.: Effects of experimental liver disease and portacaval shunts on gastric acid secretion and blood histamine, Surg. Forum, 16:322-324, 1965.

81. Winkelmann, R. K., Wilhelnij, C. M., and Horner, F. A.: Experimental studies on dermographism, Arch. Derm., 92:436-442, 1965.

82. Wolstenholme, G. E. W., and O'Connor, C. M., Editors: Ciba Foundation Symposium, Histamine, Little, Brown & Co., Boston, 1956.

83. Zachariae, H.: Histamine in human skin, Acta derm.-venereol., 44:219-222, 1964.

84. Zeller, E. Albert: Identity of histaminase and diamine oxidase, Fed. Proc., 24:766-768, 1965.

Heritable Disorders of Mucopolysaccharide Metabolism

HIBBARD E. WILLIAMS, M.D., *San Francisco*

■ *The heritable diseases of mucopolysaccharide metabolism have undergone reclassification during the past several years as a result of advances in knowledge. Recognition of clinical, genetic and biochemical differences has made it possible to delineate six types of mucopolysaccharidosis. All types are characterized by excessive excretion of one or more acid mucopolysaccharides in the urine.*

The underlying defect is not known at present, but recent investigations have suggested possible defects in protein binding and increased biosynthesis and storage of the various mucopolysaccharides.

Treatment of these disorders has been unrewarding, although administration of corticosteroids may be of some benefit.

Several diagnostic tests are now available for the determination of excessive urinary acid mucopolysaccharide excretion.

DURING THE PAST 10 years, the heritable disorders of mucopolysaccharide metabolism have been investigated extensively, leading to reclassification and a broader pathophysiologic understanding of them. As in the case of many inborn errors of metabolism, advances in biochemical techniques have allowed separation of these disorders into specific types differing by both biochemical and clinical criteria. This review will discuss these recent advances and their clinical and biochemical implications. Two extensive reviews of this subject have appeared recently.[7,12]

Biochemical Considerations

The acid mucopolysaccharides found in human tissues are high molecular weight polymers of repeating disaccharide units composed of an amino sugar and a uronic acid moiety. The amino sugar is usually N-acetylated and in some cases is sulfated through an ester linkage. The structure of these compounds has been defined in the past several years by Hoffman and coworkers.[8] The typical repeating disaccharide units of chondroitin sulfate-A and chondroitin sulfate-C are shown in Figure 1. The components of the major acid mucopolysaccharides are listed in Table 1. One of these compounds, hyaluronic acid, is a nonsulfated polysaccharide; in contrast, heparin has three sulfate molecules per disaccharide unit. Kerato-sulfate differs in that the free sugar galactose replaces the uronic acid in the disaccharide unit. Recent evidence shows that this galactose moiety is also sulfated in keratosulfate derived from cornea.[2]

Most of the known mucopolysaccharides, with the exception of hyaluronic acid, are firmly bound to protein through a covalent linkage. In their

---

From the Department of Medicine, University of California School of Medicine, San Francisco.

Reprint requests to: Department of Medicine, University of California Medical Center, San Francisco 94122.

studies, Muir [14] and Rodén and Mathews[16] have defined the nature of this linkage. Serine is essential in the linkage, and two molecules of galactose plus one of xylose appear to be interposed between the serine of the protein and the uronic acid component of the polysaccharide (Figure 2). Some heterogeneity of protein-polysaccharide complexes exists, and in addition to O-glycosidic bonds between serine and xylose, glycosylamine linkages and amide linkages between glucosamine and aspartic and glutamic acids also occur.

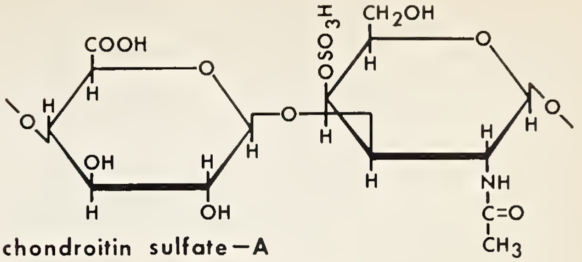
The synthesis of mucopolysaccharides involves two major pathways, each utilizing a uridine nucleotide-linked system. The uronic acid component is contributed by uridine diphosphate glucuronic acid and the hexosamine component by uridine diphosphate-N-acetyl glucosamine. Evidence now suggests that in the biosynthesis of the mucopolysaccharide-protein complex, carbohydrate units are added to pre-formed protein. The addition of sulfate molecules to the mucopolysaccharide occurs through a soluble sulfotransferase after polymerization of the disaccharide units has taken place.

Clinical Features

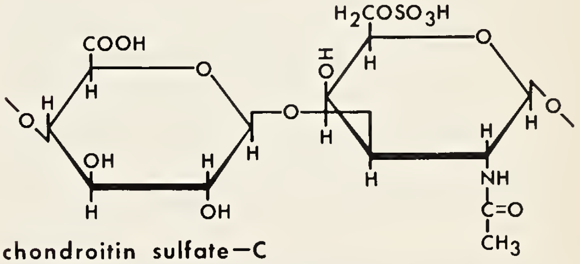
The classification of the genetic mucopolysaccharidoses is based on characteristic clinical findings and specific biochemical abnormalities consisting of the excretion of excessive amounts of one or more acid mucopolysaccharides in the urine (Table 2). The prototype of these disorders is the Hurler syndrome (mucopolysaccharidosis, type I), which will be described in some detail.

Mucopolysaccharidosis, Type I

The clinical manifestations of the Hurler syndrome become evident during the first year or two of life. The child may appear to develop normally during early infancy but deterioration is usually evident by the end of the first year. Dwarfism, grotesque facial features and mental retardation are the predominant features. The head is large and hydrocephalus may be present. The eyes are widely spaced, the ears low set and the lips thick. The bridge of the nose is depressed. The tongue is huge and the teeth are peg-like and widely spaced in hypertrophic gums. Thick, bushy eyebrows are common, as is increased hair over the forehead.



chondroitin sulfate—A



chondroitin sulfate—C

Figure 1.—Repeating disaccharide units of chondroitin sulfate-A and chondroitin sulfate-C.

The hands are broad with stubby fingers. The joints of the hands often show limitation of movement and contracture, but other joints may be involved as well. A gibbus of the lower thoracic or upper lumbar spine is common, as is pectus excavatum. The abdomen is prominent, the liver and spleen are enlarged, and umbilical and inguinal hernias may be present. Thickened skin with characteristic white nodules over the scapulae is seen frequently, as are hirsutism and coarseness of the hair.

One of the most characteristic features of this syndrome is progressive clouding of the cornea resulting in early blindness. Other ocular manifestations include retinal degeneration, optic atrophy

TABLE 1.—*Components of the Major Acid Mucopolysaccharides*

Mucopolysaccharide	*Amino Sugar*	*Uronic Acid*	*Sulfate (Molecules/ Disaccharide Unit)*
Chondroitin sulfate-A	Galactosamine	Glucuronic acid	1
Chondroitin sulfate-B	Galactosamine	Iduronic acid	1
Chondroitin sulfate-C	Galactosamine	Glucuronic acid	1
Hyaluronic acid	Glucosamine	Glucuronic acid	0
Heparin	Glucosamine	Glucuronic acid; iduronic acid	3
Heparin monosulfate	Glucosamine	Glucuronic acid; iduronic acid	1
Keratosulfate	Glucosamine	(Galactose)	1

TABLE 2.—*Classification of the Genetic Mucopolysaccharidoses**

Type	*Eponym*	*Clinical Features*	*Mucopolysaccharide*	*Inheritance*
I	Hurler	Severe mental retardation; dwarfism; progressive corneal clouding; enlarged liver and spleen; lumbar gibbus	Chondroitin sulfate-B; heparin monosulfate	Autosomal recessive
II	Hunter	Less severe mental retardation; no corneal clouding; no gibbus; deafness common	Chondroitin sulfate-B; heparin monosulfate	Sex-linked
III	Sanfilippo	Severe mental retardation; no corneal clouding; dwarfism; less enlargement of liver and spleen	Heparin monosulfate	Autosomal recessive
IV	Morquio-Brailsford-Ullrich	Normal intellect; severe bone change; aortic regurgitation	Keratosulfate	Autosomal recessive
V	Scheie	Normal intellect; corneal clouding; no dwarfism; aortic regurgitation	Chondroitin sulfate-B	Autosomal recessive
VI	Maroteaux-Lamy	Normal intellect; severe bone change; corneal clouding	Chondroitin sulfate-B	Autosomal recessive

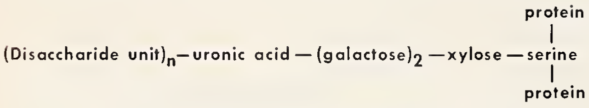
*Modified from McKusick and coworkers.[12]

and buphthalmos. Deafness may occur, but is not usual in this syndrome. Mental retardation is a consistent feature; like the corneal clouding, it is severe and progressive, often so severe that the child is put into an institution at an early age.

Cardiac involvement is one of the major causes of death. Congestive heart failure often occurs in infancy. Valvular, endocardial, pericardial and myocardial involvement, as well as coronary artery disease with angina pectoris, may be present at an early age. Respiratory infections are a common complication and another cause of death in these patients.

Roentgenographic characteristics include spatulate ribs, gibbus formation of the spine associated with wedge-shaped vertebrae with beaking of the anterior surface, an elongated "shoe-shaped" sella turcica and short, thick phalangeal bones.

The pathological features result from cellular deposition of acid mucopolysaccharides in cartilage, fascia, tendons, heart valves, blood vessels, meninges, cornea and periosteum. Electron micrographs of hepatic cells suggest that the mucopolysaccharides accumulates in the lysosomes of these cells. Cells of the central nervous system and the heart valves may also contain deposits of an abnormal lipid material. Metachromatic inclusion bodies may be found in the histiocytes and lymphocytes of the bone marrow. Similar inclusions, termed Reilly or Alder bodies, are found less frequently in circulating polymorphonuclear leukocytes. Metachromatic granules have also been demonstrated in both macrophages and granulocytes by the Rebuck skin window technique.[15]



$$\text{(Disaccharide unit)}_n\text{—uronic acid — (galactose)}_2\text{ —xylose —}\overset{\overset{\text{protein}}{|}}{\underset{\underset{\text{protein}}{|}}{\text{serine}}}$$

Figure 2.—Proposed linkage of mucopolysaccharide to protein.

The *sine qua non* of the Hurler syndrome is the excretion of excessive amounts of acid mucopolysaccharides in the urine. These mucopolysaccharides have been identified as chondroitin sulfate-B and heparin monosulfate. They are excreted in amounts four to 10 times those found in normal urine. They have also been recovered from tissues of patients with the disease, but their relative proportions in different tissues may vary considerably.

The Hurler syndrome is inherited as an autosomal recessive trait. It has been estimated to occur about once in every ten thousand births.

Mucopolysaccharidoses, Types II-VI

Type II mucopolysaccharidosis, termed the Hunter syndrome, differs clinically and genetically from the Hurler syndrome (Table 2). The clinical differentiation is based on less severe mental retardation, absence of lumbar gibbus, frequency of deafness, absence of corneal clouding and the occurrence of atypical retinitis pigmentosa. Dwarfism, enlargement of the liver and spleen, grotesque facial features and limitations of joint motion are common to both the Hurler and the Hunter syndrome. In both syndromes excessive amounts of chondroitin sulfate-B and heparin monosulfate are excreted in the urine, and therefore no distinct biochemical differentiation can be made, although some variation in the relative proportions of the two urinary mucopolysaccharides in the two syndromes has been reported. The two types of mucopolysaccharidosis are genetically distinct, however, since the Hunter syndrome is inherited as an X-linked trait.

Type III mucopolysaccharidosis (Sanfilippo syndrome) is characterized by severe mental retardation but less severe somatic changes, particularly those affecting the facial features and joints. Roentgenographic abnormalities are less pronounced and corneal clouding does not occur. Enlargement of liver and spleen and dwarfism are less prominent than in types I and II. Differentiation of this form of mucopolysaccharidosis is chiefly biochemical, since only heparin monosulfate is excreted in excessive amounts in the urine. This type is inherited as an autosomal recessive trait.

The designation type IV mucopolysaccharidosis has been applied to the Morquio-Brailsford-Ullrich syndrome. This disorder is related biochemically to the mucopolysaccharidoses but is clinically distinct. It is manifested primarily by dwarfism and severe deformities of bone, consisting of platyspondyly of the vertebrae, enlarged wrists, pigeon breast, misshapen hands and dental abnormalities. Some patients also have slowly progressive clouding of the cornea, aortic regurgitation, deafness and enlargement of the liver and spleen. Fine granulations in circulating lymphocytes are demonstrable in some patients. Intelligence is usually normal. Large amounts of keratosulfate are excreted in the urine. Inheritance is autosomal recessive.

Type V mucopolysaccharidosis (Scheie syndrome) is manifested by severe corneal clouding, retinitis pigmentosa, a "broad-mouthed" face, aortic regurgitation, limitation of joint motion and some roentgenographic features of the Hurler syndrome. Intelligence is normal. The patients excrete large amounts of chondroitin sulfate-B in the urine. Inheritance appears to be autosomal recessive.

Type VI mucopolysaccharidosis, or the Maroteaux-Lamy syndrome, is characterized by severe abnormalities of bone, radiologically similar to those seen in the Hurler syndrome, and by corneal clouding, deafness and enlargement of the liver and spleen. The patients have normal intelligence. Chondroitin sulfate-B is excreted in the urine in excessive amounts, and the inheritance pattern is assumed to be autosomal recessive.

Unclassified Mucopolysaccharidoses

A number of cases of apparent mucopolysaccharide disorders cannot be categorized under the present classification (Table 2) because of variations in clinical, biochemical or genetic features. In such patients mucopolysaccharide excretion, when it has been determined, has been normal. As more knowledge of mucopolysaccharide metabolism is obtained, definitive classification of these cases may be possible. Finally, mucopolysaccharide excretion may be abnormal in some acquired disorders, namely lupus erythematosus, rheumatoid arthritis, diabetes mellitus, neoplasia and systemic mastocytosis. In these conditions, however, the degree of mucopolysaccharide excretion does not approach that found in the genetic mucopolysaccharidoses.

Pathogenesis

A number of theories have been proposed to explain the basic defect in the mucopolysaccharidoses. These include (1) faulty embryonic differentiation of mesenchymal cells, (2) defective binding of mucopolysaccharide to protein or production of an abnormal protein moiety, (3) mutation of a regulator gene controlling mucopolysaccharide synthesis, and (4) a block in the degradation of mucopolysaccharides.

The first theory, proposed by Meyer and Hoffman,[13] is plausible but unproved. According to this hypothesis, faulty embryologic differentiation of mesenchymal cells would lead to two groups of cells, each capable of producing a single mucopolysaccharide—a mechanism that could explain the accumulation of two mucopolysaccharides in type I and type II mucopolysaccharidosis.

The recent studies of Knecht and Dorfman[10] have suggested the possible role of abnormal protein binding in the pathogenesis of the mucopolysaccharidoses. These investigators showed that heparin monosulfate isolated from hepatic tissue from patients with the Hurler syndrome is of low molecular weight and contains only small amounts of amino acids. The mucopolysaccharide was resolved into two fractions: One contained serine in a ratio of one serine residue to each 10 polysaccharide chains, and the second contained small amounts of amino acids with no predominance of serine. From such data it has been hypothesized that a defect in binding of polysaccharide to serine may account for the accumulation of mucopolysaccharide in tissues. An alternate explanation suggests that a defect in polysaccharide-peptide production prevents normal formation of macromolecular structure in tissues.[10]

The third possibility, a defect in a regulator gene controlling mucopolysaccharide synthesis, is purely theoretical at present. Regulator genes controlling mucopolysaccharide synthesis have been demonstrated in bacteria, but proving the presence of such a mechanism in mammalian systems must await further study.

Most inborn errors of metabolism result from a deficiency in degradative enzymes, and a similar defect seems a logical mechanism in the genetic mucopolysaccharides. The demonstration by Van Hoof and Hers[17] that lysosomal inclusion bodies may be found in hepatic cells from patients with mucopolysaccharidoses raises the possibility of a defect in a lysosomal enzyme necessary for mucopolysaccharide breakdown. Such an enzyme has been demonstrated in lysosomes of hepatic tissue from the rat by Hutterer.[9]

In 1966 Danes and Bearn[3] demonstrated the accumulation of metachromatic granules in fibroblasts cultured from the skin of patients with the Hurler syndrome and from their phenotypically normal parents. Metachromatic granules were also present in fibroblasts from the skin of the mothers, but not of the fathers, of patients with the X-linked Hunter syndrome. These significant studies demonstrate that skin fibroblasts are capable of synthesizing mucopolysaccharides for prolonged periods in tissue culture and offer a means of determining the presence of the heterozygote state in relatives of patients with type I and II mucopolysaccharidoses. These findings have been confirmed by Matalon and Dorfman,[11] who also demonstrated that cultured fibroblasts from patients with Hurler's syndrome accumulated five to 10 times more acid mucopolysaccharides than cells from normal individuals. These investigators also presented evidence obtained from *in vitro* isotopic studies that the rate of synthesis of acid mucopolysaccharides, as well as the storage of these compounds within cells, is augmented in patients with Hurler's syndrome.

Treatment

Treatment of the genetic mucopolysaccharidoses has been unrewarding in most instances. Although large doses of corticosteroids have been shown to inhibit synthesis of mucopolysaccharides in animals, trial of such treatment in several patients has given varying results. A decrease in the urinary excretion of mucopolysaccharide, together with clinical improvement, has been claimed in some reports. Other more extensive long-term studies, however, have indicated that mucopolysaccharide excretion gradually returns to elevated levels despite prolonged corticosteroid therapy. Attempts to improve the vision of patients with corneal clouding have also been generally unsuccessful. Corneal transplants have become opacified in patients with type V mucopolysaccharidosis. Lamellar corneal grafts may be of benefit in the Hurler syndrome.

Diagnostic Tests

Several relatively simple laboratory tests are available for detecting increased levels of acid mucopolysaccharides in the urine.

Urine Spot Test[1]

Whatman No. 1 filter paper is spotted in three areas with 5, 10 and 25 microliters of urine, added 5 microliters at a time. After each addition the spot is allowed to dry thoroughly before the next 5 microliters of urine are added. The paper is dipped in an aqueous solution of toluidine blue (0.04 per cent, buffered at pH 2); after about one minute it is rinsed in 95 per cent ethanol. A purple spot against the blue background of the filter paper indicates the presence of abnormal amounts of mucopolysaccharide.

Acid Albumin Turbidity Test[6]

Urine is centrifuged and dialysed against distilled water for at least 3 hours. A mixture of 1.5 ml of the dialysed urine, 0.5 ml of 0.3 M phosphate-citrate buffer (pH 5.6) containing 0.45 M sodium chloride, and 10 ml of acid albumin reagent (0.1 per cent purified serum albumin in 0.1 M acetate buffer, pH 3.75) is prepared and allowed to stand for 10 minutes. Control solutions containing known amounts of chondroitin sulfate are handled similarly. Turbidity is then measured in a colorimeter at 540mμ with a 1 cm light path. The result is positive if the optical density of the test solution is greater than 0.05 O.D. units.

Cetyl Trimethyl Ammonium Bromide Precipitation Test[4]

With this method urinary acid mucopolysaccharide is determined by measuring its glucuronic acid content after precipitation with a quartenary ammonium salt. Duplicate 30 ml aliquots of a 24-

hour urine specimen are placed in tubes and brought to pH 5 with 2 N hydrochloric acid. To each tube is added 1 ml of cetyl trimethyl ammonium bromide (2 per cent aqueous solution). The contents are mixed and allowed to stand overnight at 2°C (35°F). The precipitate is then centrifuged at room temperature for 15 minutes at 2,000 times gravity. The supernatant fluid is removed, and the tubes are allowed to drain for one minute. The precipitate is then washed twice with 30 ml of 45 per cent ethanol saturated with sodium chloride. About 1.5 ml of distilled water and 2 drops of 0.1 N sodium hydroxide are added, and the precipitate is dispersed. The solution is transferred by washing to a 5 ml volumetric flask and brought to volume with water. Any insoluble material is removed by centrifugation. The glucuronic acid content of the solution is determined on 1 ml aliquots by the carbazole method of Dische.[5] With this method the normal 24-hour urinary excretion of acid mucopolysaccharide, expressed as milligrams of glucuronic acid, is 4.40 to 7.28 for men and 2.73 to 5.40 for women. The normal values for children are somewhat higher.

REFERENCES

1. Berry, H. K., and Spinanger, J.: A paper spot test useful in study of Hurler's syndrome, J. Lab. Clin. Med., 55:136-138, January 1960.

2. Bhavanandan, V. P., and Meyer, K.: Mucopolysaccharides: N-acetyl-glucosamine- and galactose-6-sulfates from keratosulfate, Science, 151:1404-1405, 18 March 1966.

3. Danes, B. S., and Bearn, A. G.: Hurler's syndrome: A genetic study in cell culture, J. Exper. Med., 123:1-16, 1 January 1966.

4. DiFerrante, N., and Rich, C.: The determination of acid amino polysaccharide in urine, J. Lab. Clin. Med., 48:491-494, September 1956.

5. Dische, Z.: A new specific color reaction of hexuronic acids, J. Biol. Chem., 167:189-198, January 1947.

6. Dorfman, A.: Studies on the biochemistry of connective tissue, Pediatrics, 22:576-589, September 1958.

7. Dorfman, A.: Heritable diseases of connective tissues: The Hurler syndrome; From The Metabolic Basis of Inherited Disease, Edited by J. B. Stanbury, J. B. Wyngaarden, and D. S. Fredrickson, Second Edition, McGraw-Hill, New York, 1966, pp. 963-994.

8. Hoffman, P., Linker, A., and Meyer, K.: Chondroitin sulfates, Fed. Proc., 17:1078-1082, December 1958.

9. Hutterer, F.: Degradation of mucopolysaccharides by hepatic lysosomes, Biochim. et Biophys. Acta, 115: 312-319, 28 February 1966.

10. Knecht, J., and Dorfman, A.: Structure of heparitin sulfate in tissues of the Hurler syndrome, Biochem. Biophys. Res. Comm., 21:509-515, 9 December 1965.

11. Matalon, R., and Dorfman, A.: Hurler's syndrome: Biosynthesis of acid mucopolysaccharides in tissue culture, Proc. Nat. Acad. Sci., 56:1310-1316, October 1966.

12. McKusick, V. A., Kaplan, D., Wise, D., Hanley, W. B., Suddarth, S. B., Sevick, M. E., and Maumanee, A. E.: The genetic mucopolysaccharidoses, Medicine, 44:445-483, November 1965.

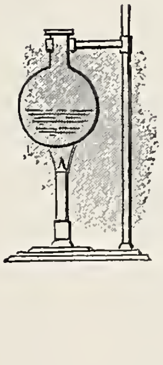
13. Meyer, K., and Hoffman, P.: Hurler's syndrome, Arth. and Rheum., 4:552-560, October 1961.

14. Muir, H.: The nature of the link between protein and carbohydrate of a chondroitin sulfate complex from hyaline cartilage, Biochem. J., 69:195-204, June 1958.

15. Rebuck, J. W., and Crowley, J. H.: A method of studying leukocytic functions *in vivo,* Ann. N.Y. Acad. Sci., 59:757-805, 24 March 1955.

16. Rodén, L., and Mathews, M. B., cited by Dorfman, A.: Heritable diseases of connective tissues: The Hurler syndrome; From The Metabolic Basis of Inherited Disease, Edited by J. B. Stanbury, J. B. Wyngaarden, and D. S. Fredrickson, Second Edition, McGraw-Hill, New York, 1966, p. 971.

17. Van Hoof, F., and Hers, H.-G.: L'ultrastructure des cellules hépatiques dans la maladie de Hürler (gargoylisme), Compt. Rend. Acad. Sci., 259:1281-1283, 3 August 1964.



Periodic Fever

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California Medical Center, San Francisco. Taken from transcriptions, they are prepared by Drs. Martin J. Cline and Hibbard E. Williams, Assistant Professors of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine.

DR. HOWARD MORRELLI*: The patient is a 33-year-old Caucasian housewife and hairdresser from Sacramento, California. She was admitted to the University of California Hospital because of recurrent chills and fever of lifelong duration. As a child, she had had intermittent fever of 39.4° to 40°C (103° to 104°F) every four to six weeks which was attributed to respiratory infections. As she grew older, these episodes became more frequent and more severe. At present, every 10 to 14 days she has fever of 39.4° to 40°C, occasionally as high as 41°C (106°F), lasting three to four days. During these episodes she has noticed tenderness and swelling of lymph nodes. On biopsy of one of the nodes a year before the present admission, non-specific lymphadenitis was noted. Occasionally with these critical episodes, the patient has had sharp abdominal pain, headaches or dysphagia. At the end of an attack she frequently has noticed diarrhea. She thinks that her father may have had a similar affliction. Both of her children have a similar problem in that every four to six weeks they have a fever of 40° to 41°C (104° to 106°F) lasting 24 hours.

On physical examination, rectal temperature was 38°C. Several discrete, slightly tender axillary, inguinal and cervical lymph nodes were felt. Results of examination of the chest, heart and abdomen were within normal limits.

The number and the types of leukocytes in the blood were within normal limits, the hematocrit was 36 per cent, and the erythrocyte sedimentation rate, uncorrected, 51 mm in one hour. Urinalysis showed a trace of protein, but a 24-hour collection revealed less than 100 mg of protein per 100 ml. Serum protein electrophoresis demonstrated slight elevation in alpha-2-globulins and gamma globulins. Serum fibrinogen during a febrile episode was elevated to 581 mg per 100 ml. Determination of urinary etiocholanolone excretion was done independently in two laboratories on two separate days when the patient was febrile, and in both instances the amount reported was at the lower end of normal range.

An x-ray film of the chest showed slight prominence of the main pulmonary artery. Antinuclear antibodies and febrile agglutinins, and results of renal function and liver function tests were all within normal limits. Blood volume studies by the Evans blue method, the iodinated albumin method and chromium-labeled blood cell techniques were normal, indicating no discrepant uptake of dye which might indicate systemic amyloidosis. The absence of amyloidosis was further substantiated by rectal biopsy.

By the fourth hospital day the patient was afebrile. (The highest level the temperature had reached was 39°C. She was discharged on a 20-gm fat diet in the hope that this might ameliorate the symptoms. Although febrile episodes recurred at 10- to 14-day intervals, the temperature would reach only 38.4°C (101°F) rather than 39.4° to 40°C (103° to 104°F), but the patient did not think the improvement in symptoms warranted the very restrictive 20-gm fat diet.

DR. LLOYD H. SMITH, JR.*[1]: How are you feeling today?

PATIENT: Very good.

DR. SMITH: Could you describe for the doctors here what happens when one of these attacks begins?

PATIENT: I feel very cold at first, and I shake. I have a fever while I am cold.

DR. SMITH: How long does it last?

PATIENT: About three days. I don't think it has ever lasted longer than that.

DR. SMITH: Does it really incapacitate you? Do you have to go to bed and give up your ordinary activities?

---

*Instructor in Medicine and Pharmacology.

*[1] Professor and Chairman, Department of Medicine.

PATIENT: Sometimes I do; not all the time. It depends on the height of the fever.

DR. SMITH: Are you bothered by pain in your abdomen or in your chest? Do you have difficulty breathing when you have these attacks?

PATIENT: No, just headaches; or sometimes aching in the stomach or abdomen.

DR. SMITH: You were put on a low fat diet after your visit here. Do you believe this gave you any improvement in the frequency or the severity of these attacks?

PATIENT: I think they happened just as often. The fevers didn't get as high.

UNKNOWN PHYSICIAN IN AUDIENCE: What is the patient's family background?

DR. SMITH: Do you want to describe the origin of your family?

PATIENT: My father was born in Rome. He died when I was 10. My mother is 77 and very healthy.

DR. SMITH: Where was she from originally?

PATIENT: She was born in South Dakota.

DR. SMITH: Thank you very much for coming in this morning. (The patient leaves.) Now I will ask Dr. Morrelli, who has been interested in this syndrome, to review it for us.

DR. MORRELLI: The patient has a very rare disease; only 275 cases have been reported in the literature through 1964. It was first characterized clearly by Janeway[7] in 1908 and I think his description of a young girl with paroxysmal peritonitis is the best available in literature. It was recognized at this hospital by Althausen in 1930, and a case report was written describing it as the "false acute abdomen." Beginning in the 1940's, large series of cases began to appear in the literature, the disease variously called benign paroxysmal peritonitis, periodic disease, familial Mediterranean fever and familial recurrent polyserositis. There is no clearly defined etiologic explanation for the disease. As will be emphasized later, it is probably genetic, transmitted either as an autosomal dominant with incomplete penetrance or a recessive trait. Since we have no means of substantiating this etiologic conjecture by means of a specific laboratory test, it is at this time a symptom complex only.

Clinical Features

The symptom complex consists of episodes of recurrent fever. The fever is not an equivocal or trivial fever but usually is in the range of 39.4° to 40°C (103° to 104°F), occasionally reaching 41° (106°). It is always reported to be periodic, the patients reporting variously that they have fever once a month or once every seven days; but when an accurate log is kept, it is found that in an individual case the fever has a variation within the time period that the patient has related. The disease is ordinarily of such long duration that serious diseases such as tuberculosis and lymphoma can be excluded. In addition to the fever, the patients most commonly have pain, particularly severe abdominal pain, characteristic of peritonitis. Early in the history of this disease, most patients have had an appendectomy or cholecystectomy because of the pronounced signs of peritoneal irritation, fever and leukocytosis which are characteristic of the syndrome. A large number of patients have stethalgia or chest pain of a pleuritic nature. On x-ray examination there may be evidence of a small pleural effusion or disc atelectasis on the involved side. The arthritis of familial Mediterranean fever is less impressive, both numerically and as an individual symptom. The joints do not usually seem to be as severely involved as in gout or rheumatoid arthritis. During the asymptomatic intervals, the patient is perfectly well—as the patient was today—and in this respect an analogy can be drawn between periodic fever and gout: In the intercritical periods, a patient with either disease might well run in the Olympic Games.

No pathologic state is found after exhaustive examination that would account for the symptoms. The patients have recurrent attacks at intervals from once a year to once a week. In any given individual, this interval is fairly characteristic. During the course of the disease, the interval tends to shorten; however, the intervals may become longer and spontaneous remissions may occur. In recent years, it has become apparent that in a fair number of patients with this disease amyloidosis develops, and they may die of renal failure.[4,17] Unfortunately, there has been no constant relationship between the clinical manifestations in a given patient and the likelihood that amyloidosis will develop.

Table 1 is a summary of the clinical features in patients reported in the literature through 1964. There are three major reviews: By Heller in 1958,[6] by Ehrenfeld in 1961,[3] and by Siegal in 1964.[15]

TABLE 1.—*Frequency of Signs and Symptoms of "Familial Mediterranean Fever"*

	Heller 1958	*Ehrenfeld 1961*	*Collected '30 to '62*	*Siegal 1964*	*Totals*	*Per Cent*
Number of patients	74	55	92	50	271	......
Fever	74	55	92	50	271	100
Abdominal pain	68 (92)	34 (62)	88 (96)	49 (98)	239	88
Chest pain	60 (81)	21 (38)	42 (45)	41 (82)	164	60
Joint pain	61 (82)	17 (31)	56 (61)	22 (44)	156	57
Erysipeloid	27 (36)	4 (7.2)	4 (4.3)	4 (8)	39	14
Positive family history	42 (57)	16 (29)	41 (44)	14 (28)	113	42
Leukocytosis	Usual	Usual	Usual	47	......	......
Sedimentation rate decidedly elevated	Usual	Usual	Usual	35 per cent	......	......
Jewish	73 (98)	55 (100)	60 (66)	38 (76)	226	83
Mediterranean	74 (100)	55 (100)	65 (70)	12 (24)	206	76
Proven amyloid	3	4	20 (most recent)	1 (2)	28	12
Men/women	50/24	36/19	59/33	36/14	181/90	......
Age at Onset						
1- 9	46	14	......	12	72	Preponderance of onset at less than 20 years
10-19	27	17	......	19	63	
20-29	1	13	......	14	28	
30-39	0	7	......	4	11	

Fever was the universal complaint. In the combined group the incidence of abdominal pain, characteristic of peritonitis, was 88 per cent. Chest pain occurred in 60 per cent of the patients, joint pain in 57 per cent.

Heller's article in 1958 described an erysipeloid lesion of the lower extremities in 27 of 74 patients. However, observers reporting since then have found this to be a fairly infrequent manifestation of the disease—the overall incidence 14 per cent. Similarly, with regard to the family history, if collected by an observer who sees family members from a small local geographic area, familial incidence of the disease will be considerably higher than that shown in a combination of individual case reports.

Leukocytosis, between 10,000 and 20,000 per cu mm of blood, is expected. The erythrocyte sedimentation rate is usually quite high, commonly between 50 and 75 mm in an hour.

The first two series by Heller and Ehrenfeld are possibly skewed toward a very high incidence among the Jewish population. At one time there was a great concern about the relative preponderance of occurrence in Sephardim amongst the Jews, but this has not proved to be of great diagnostic importance. In the remainder of the case reports an inordinately high proportion of patients are Jews, but there is also a significant proportion of Armenians and Italians. The same can now be said also of peoples remote from Mediterranean origin. Because the first review articles were published from the Mediterranean area, it was felt that all patients were Mediterranean; hence the epithet *familial Mediterranean fever*. In later publications, it has become quite clear that this is not the case.

About twice as many men as women have been reported with this disease. Proved amyloidosis in these patients was infrequently recognized early, but in the later case reports it has become apparent that amyloidosis is a significant complication of the illness, and may appear very early. In recent years the reported incidence of amyloidosis has become higher.

Additional symptoms that are mentioned with some frequency in the review articles and case reports are constipation, vomiting, disturbances in micturition, severe headaches—periodic meningitis as a manifestation of this disease has been reported[5]—diarrhea and, sometimes, precordial pain. There is only one documented case in which precordial pain was simultaneous with electrocardiographic changes of pericarditis. Sialorrhea has been reported; profuse diaphoresis with the termination of the attack is characteristic. As a manifestation of serous membrane involvement the serous membranes of the testes may be affected, causing testicular pain. Lymphadenopathy is not frequently described in the syndrome, but certainly it was a prominent feature in the patient presented today. Splenomegaly is occasionally reported.

An interesting physical finding described in 1961[11] was an increased incidence of cystic degeneration of the elastic lamina of Bruck. On examination of the choroid, a "domino" appearance was seen. I have not seen later reports confirming this as a frequent finding in Mediterranean fever. As mentioned earlier, the symptoms have usually been present for many years before the diagnosis is established. This is helpful in establishing the diagnosis. Table 2 outlines the data of Heller and Ehrenfeld[3,6] indicating that the disease has commonly been present for many years before diagnosis is made.

At laparotomy or peritoneoscopy, the gross appearance of the serosal membranes is that of edema and hyperemia. On biopsy, or if the appendix is removed, one sees edema and bland infiltration with mononuclear, plasma and mast cells.[18] Fibrillar change in collagen has been described in some cases.

Treatment

I would like to expound on the pathological physiology and the rational approach to drug therapy of this disorder but I am unable to do that, since the basic physiologic disturbance is unknown. The treatment is generally symptomatic—aspirin for the fever, usually partially effective, meperidine (Demerol®) or morphine for severe painful episodes. (Narcotic addiction is uncommon in this condition.) The 20-gm fat diet recommended by Mellinkoff[9,10] is probably worthy of trial pending definitive etiologic knowledge. Antihistamines, antibiotics, corticosteroids, antiepileptic agents, sympathetic blocking agents, antimalarial drugs, ergotamine, paraminobenzoic acid, ephedrine, phenylbutazone, phenothiazines, atropine and appendectomy are all unsuccessful in therapy. This disease is so chronic and recurrent that it leads physicians to try many drugs seriatim in an attempt to ameliorate the symptoms.

TABLE 2.—*Duration of Symptoms in 129 Cases of "Familial Mediterranean Fever" at Time of Diagnosis (Heller and Ehrenfeld)*

	Number of Cases
1- 4 years	24
5- 9 years	48
10-14 years	26
15-19 years	14
20-24 years	8
25 and more	9
	129

Etiology

The causes postulated for this illness are numerous. A genetic basis has been suggested[13,16] but there is no objective proof for this. Since the original case reports were from the Mediterranean area, it was suspected that there might be an agent in the environment which caused the symptoms. An analogous situation is now coming about in the etiologic conjectures about Burkett's lymphoma in Africa. Infection and infestation are always attractive but no etiologic agent has been found. Allergic sensitivity is sometimes mentioned as a possibility, but there have been only one or two case reports in which patients were greatly improved by being on a milk-free diet.

Physical agents have been suggested (analogous to, say, the aggravation of porphyria cutanea tarda by sunlight). Chemical agents such as pronestyl may induce a lupus-like syndrome, but there has been no unifying historical exposure to either physical or chemical agents that would permit us to explain this syndrome on these bases.

Etiocholanolone fever[1] was thought to be identical with familial Mediterranean fever for a very short time—until a number of patients with the latter were found to have normal etiocholanolone excretion rates. Neoplastic diseases have been suggested as a possibility, since myeloma may be complicated by amyloidosis and since Hodgkin's may present as fever, but I find no closer relationship than these symptomatic examples. The degenerative diseases probably do not pertain here, since the disease begins as a rule early in childhood; about 75 per cent of patients have symptoms before age 20. In the middle 1940's there was discussion attributing the disease to some periodic or natural rhythm of the body itself.[14] This has not withstood the test of time. I have included as reference[12] a bit of interesting reading about periodicity as a cause of the disease. It is a very lengthy expostulation on host factors that might cause symptoms to recur in a periodic fashion. These theories attempted to explain the periodic fever of malaria, the cause and pathogenesis being then unknown.

DR. SMITH: I think Dr. Morrelli has covered all the pertinent information now available concerning this very distressing and mysterious illness. Are there any questions?

DR. OTTO GUTTENTAG*[2]: Would you mind elaborating on the etiocholanolone findings in the patient presented today?

---

*[2] Samuel Hahnemann Professor of Medical Philosophy.

DR. MORELLI: Etiocholanolone fever was found in a young man who had periodic fever. His etiocholanolone excretion rate was high. On injection of etiochololanolone into this patient, he had febrile episodes. He did not, however, have the other concomitants of Mediterranean fever nor have the other patients with proven etiocholanolone fever had peritonitis, chest pain or arthritis. Why today's patient had low etiocholanolone levels on two occasions as determined by independent laboratories is unexplained. One laboratory reported that perhaps there was an interfering substance, but did not specify what it might be.

DR. SMITH: How well has the inheritance of this disease been traced? It is quite clear that the pattern is at least vaguely that of a dominant; how much does it deviate from true dominant transmission?

DR. MORRELLI: There are families such as those in Heller's series and Ehrenfeld's series in which every generation for several generations will have a patient with the disease. Bouroncle[2] reported a German family in which three generations were involved, but fewer than half of all patients have a positive family history.

DR. KENNETH MELMON*[3]: When I heard that the patient had edema, sialorrhea, diaphoresis, diarrhea, leukocytosis and fever, I wondered whether there is any role for the kinins in this disease?

DR. MORRELLI: We sent specimens to your laboratory for determination. The results are pending.

DR. RICHARD HAVEL*[4]: You mentioned that a low fat diet was worth trying. Would you elaborate on that?

DR. MORRELLI: Mellinkoff says that it works. My limited experience is that it worked in one patient and did not work well in the second.

DR. JAMES HOPPER*[5]: It is worth pointing out that although rectal biopsy is a good way to get evidence of amyloidosis, renal biopsy is probably much better.

DR. SMITH: There are other forms of familial amyloidosis. I wonder whether patients with those forms have fever. Do you know of the other types of familial amyloidosis as a comparison with this syndrome? Do the patients have amyloidosis in the absence of any plasma protein abnormality, or do they show changes on electrophoresis or ultracentrification?

DR. MORRELLI: They may have minor changes in serum electrophoresis, notably an increase in alpha-2-globulin, but this is not diagnostic of the presence of amyloid. The plasma proteins may be normal by this method, and yet amyloid may be present. Ruicavina's cases of familial amyloidosis were not characterized by recurrent fever.

DR. SMITH: And yet they still have severe amyloidosis?

DR. MORRELLI: Yes.

GENERIC AND TRADE NAME OF DRUG

Meperidine—*Demerol.*

REFERENCES

1. Bondy, P. K., Cohn, G. L., and Gregory, P. B.: Etiocholanolone fever, Medicine, 44:249-262, 1965.
2. Bouroncle, B. A., and Doen, C. A.: Periodic fever: Occurrence in five generations, Amer. J. Med., 23:502-506, 1957.
3. Ehrenfeld, E. N., Eliakim, M., and Rachmilewitz, M.: Recurrent polyserositis (Familial Mediterranean fever; periodic disease), Amer. J. Med., 31:107-121, 1961.
4. Fox, M., and Morrelli, H.: Periodic fever with renal amyloidosis, New Engl. J. Med., 263:669-672, 1960.
5. Siegal, S.: Familial paroxysmal polyserositis, Amer. J. Med., 36:893-918, 1964.
6. Heller, H., Sohar, E., and Sherf, L.: Familial Mediterranean fever, Arch. Intern. Med., 102:50-71, 1958.
7. Janeway, T. C., and Mosenthal, H. O.: An unusual paroxysmal syndrome, Trans. Assn. Amer. Physicians, 23:504-518, 1908.
8. Mamou, H., and Cattan, R.: La maladie périodique (sur 14 cas personnels dont 8 compliqués de nephropathis), Sem. Hop. Paris, 28:1062-1070, 1952.
9. Mellinkoff, S. M., Schwabe, A. D., and Lawrence, J. S.: A dietary treatment for familial Mediterranean fever, Arch. Intern. Med., 108:80, 1961.
10. Mellinkoff, S. M., Snodgrass, R. F., Schwabe, A. D., Mead, J. F., Weimer, H. E., and Frankland, M.: Familial Mediterranean fever, low fat diet, plasma protein abnormalities, possible implications in pathogenesis, Ann. Intern. Med., 56:171, 1962.
11. Michaelson, I., Eliakim, M., Ehrenfeld, E. N., and Rachmilewitz, M.: Fundal changes resembling colloid bodies in recurrent polyserositis (Periodic Disease), Arch. Ophthalmol., 62:29-32, 1959.
12. Pallas, E., Audouard, M. F. M., De Mamers, G., and Bailly, E. M.: Analytical and critical review: Periodicity, The North American Medico-Chirurgical Review, 1:641, 1857.
14. Reiman, H. A.: Periodic disease: Observations on old cases and report of new cases and of therapeutic trials, Arch. Intern. Med., 92:494-506, 1953.
15. Siegal, S.: Familial paroxysmal polyserositis, Amer. J. Med., 36:893-918, 1964.
16. Sohar, E., Prass, M., Heller, J., and Heller, H.: Genetics of familial Mediterranean fever, Arch. Intern. Med., 107:529, 1961.
17. Sohar, E., Heller, H., Gafney, J., and Heller, J.: Amyloidosis in familial Mediterranean fever, Arch. Intern. Med., 10:539, 1961.
18. Tuquan, N. A.: Periodic disease: Clinicopathologic study, Ann. Intern. Med., 49:885-889, 1958.

---

*[3] Professor of Medicine and Pharmacology, Chmn. Sect. Clin. Pharm.

*[4] Professor of Medicine.

*[5] Professor of Medicine.

Editor
DWIGHT L. WILBUR, M.D.
•
Assistant to the Editor
ROBERT F. EDWARDS
For information on preparation of manuscript, see advertising page 2

Policy Committee—Editorial Board
JAMES C. MacLAGGAN, M.D., San Diego
JOHN G. MORRISON, M.D., San Leandro
WILLIAM F. QUINN, M.D.. Los Angeles
JOSEPH W. TELFORD, M.D., San Diego
CARL E. ANDERSON, M.D., Santa Rosa
HELEN B. WEYRAUCH, M.D.
San Francisco
DWIGHT L. WILBUR, M.D., San Francisco

California
Medicine

EDITORIAL

The Health Manpower Council

WITH THE FORMATION of the California Health Manpower Council, a sound beginning appears to have been made toward finding ways to meet increasing demands for health care services.

The new organization has been set up as a voluntary agency supported by all disciplines in the health care field to coordinate efforts in recruitment and training of personnel and the utilization of personnel and facilities involved in medical care. It grew from an idea put forward by the California Hospital Association, which also supplied the first impetus to get it under way. It is self-financing, the $100,000 estimated needed for the first year's expenses having been subscribed by members; and it will hire its own full-time executive secretary and whatever supporting staff is needed.

Formed following two general Health Manpower Conferences, the Council has a membership of 25 persons representing education, consumers, labor and government as well as the various suppliers of health services. Thirteen of the members of the Council are selected from nominations by all organizations interested in the health manpower problems. The remaining 12 are representatives of designated organizations, as follows: One each from the California State Departments of Public Health, of Education and of Employment; one each from the California Nurses Association and the California Nursing Home Association; one from the California Dental Association and the Southern California Dental Association; three from the California Hospital Association and three from the California Medical Association. Terms of office will be staggered.

From its make-up, the organization appears to be in a position to bring about an exchange of ideas and information. Each of the elements involved will have a place to make proposals of its own and to hear, weigh and shape the proposals of others. The Council will provide a place where each can influence—and be influenced. One great service it can perform for all its constituents is to eliminate the waste motion that is inherent in separate planning by individual organizations. From the information that will come to it from its variety of members, the Council should be able to determine what is needed and how best to go about supplying the need.

The California Medical Association may congratulate itself on the foresight that has equipped it well to fill its three places on the Council with physicians who already are well versed in the subjects on which the Health Manpower Council's proceedings will turn. They are Dr. Harold Kay, Dr. Frank C. Melone and Dr. C. Gerald Scarborough, who are the chairmen of, respectively, the CMA Commission on Community Health Services, the Committee on Paramedical Personnel and the Committee on Other Professions. The Commission and the two Committees have a substantial background of studious concern with many of the matters that must be involved in any general consideration of how to overcome the shortage of health care manpower. Behind these physicians are CMA staff members who are abreast of developments in these fields.

It is probable that, as with almost any organization as new and as far-reaching as the Council, shortcomings and inequities will be encountered from time to time. But they can be dealt with as they appear. The important thing is that we do have in California an organization that holds promise of bringing many interdependent elements into concert to solve a complicated problem that none of them could begin to solve alone.

Less important, of course, but still a matter of provincial pride, is that we in California are apparently the first among the states to have under way a full scale engagement with the problems of health manpower shortage.

California Medical Association

NOTICES AND REPORTS

Where We Stand on the Question of Therapeutic Abortion

CALIFORNIA LAW currently permits therapeutic abortion only to "preserve" the life of the mother. The law in California is typical of abortion statutes now in effect in 44 other states. In five states the law is slightly more liberal in that it calls for protecting a mother's health as well as her life.

California's law pertaining to therapeutic abortion was adopted by the Legislature in 1872. It has remained virtually unchanged since that time in spite of significant advances in medical science and changing patterns in social thinking.

Today's medical science bears little resemblance to that which was practiced in California in 1872. Yet, paradoxically, today's physician still finds himself bound to outdated abortion legislation which perpetuates needless suffering and fosters poor medical practices.

The California Medical Association has become increasingly concerned about the quality of medical care rendered under California's antiquated abortion statutes. On three separate occasions the CMA House of Delegates has called for modification of the law to provide for therapeutic abortion which is medically justifiable and takes into consideration the product of conception as well as the health of the mother.

In 1962, the CMA House of Delegates adopted Resolution 30 stating that they go on record in support of legislation "protecting the health of the mother in the consideration of therapeutic abortion and sterilization procedures." Also, that same year the delegates went on record as "strongly supporting the objectives of A.B. 2614 (The Therapeutic Abortion Act) and use every effort to bring forth positive action thereon." This bill later died during interim study.

Again, in 1963 the CMA House of Delegates adopted Resolution 93 which instructed the CMA's Legislative Committee to "attempt to modify the present law to relax the indication for therapeutic abortion from the present requirement that the mother's life be endangered to state 'that the mother's physical health be endangered' and that therapeutic abortion for mental health of mothers be restricted to serious psychiatric diagnosis such as severe psychosis and not for minor psychiatric diagnosis such as psychoneurosis."

JAMES C. MacLAGGAN, M.D.	President
JOHN G. MORRISON, M.D.	President-Elect
WILLIAM F. QUINN, M.D.	Speaker
JOSEPH W. TELFORD, M.D.	Vice-Speaker
CARL E. ANDERSON, M.D.	Chairman of the Council
ALBERT G. MILLER, M.D.	Vice-Chairman of the Council
HELEN B. WEYRAUCH, M.D.	Secretary
DWIGHT L. WILBUR, M.D.	Editor
HOWARD HASSARD	Executive Director

General Office, 693 Sutter St., San Francisco 94102 • 415 776-9400

JACK CURLEY	Southern California Office

1515 N. Vermont Ave., Los Angeles 90027 • 213 663-8071

RICHARD L. LEMOS	Sacramento Office

1127 11th St., Sacramento 95814 • 916 444-7496

Dated 27 February 1967.

During the 1964-65 rubella (German measles) epidemic, large segments of the public and the medical profession again questioned the wisdom and justice of California's therapeutic abortion statutes when it was clearly demonstrated that rubella during the first three months of pregnancy risks permanent damage to the fetus. Yet, California's rigid codes pertaining to therapeutic abortion prohibit abortion for medically justifiable reasons except when necessary to preserve the life of a mother.

So, again in 1966, the CMA's House of Delegates adopted another resolution "reaffirming its position and express to the State Legislature its belief in the broadening of the therapeutic abortion law, taking into consideration the health of both the mother and the product of conception and that such legislation should provide proper medical control through established hospital staffs or component medical society committees."

The current position of the California Medical Association on therapeutic abortion then is quite clear. We believe:

1. That the law should be broadened "taking into consideration the health of both the mother and the product of conception" and

2. That such legislation should provide for proper medical control through established hospital staffs or component medical society committees.

It is our belief that the best way to implement this would be by supporting Senator Anthony Beilenson's proposed legislation which closely parallels the recommendations of the American Law Institute's Model Penal Code on Therapeutic Abortion.

Section 230.3 of the ALI's Model Penal Code defines justifiable abortion to be the termination of pregnancy by a licensed physician for any of the following three reasons:

a. That continuance of the pregnancy would gravely impair the physical or mental health of the mother;

b. That the child would be born with a grave physical or mental defect, or

c. That pregnancy resulted from rape or incest.

Senator Beilenson's proposals for modifying our current California laws on therapeutic abortion are reasonable. Ample protection is afforded the patient to assure that only licensed physicians perform therapeutic abortions in hospitals which provide the highest quality of care and meet the exact standards of the Joint Commission on Accreditation of Hospitals (JCAH).

Furthermore, the procedure must be approved in advance by a special medical staff committee of the respective hospital which shall be established and maintained in accordance with standards promulgated by the JCAH.

Also, a therapeutic abortion can be performed only when this committee has found that one or more of the following conditions exist:

a. That there is substantial risk that continuance of the pregnancy would gravely impair the physical or mental health of the mother;

b. That there is substantial risk that the child will be born with grave physical or mental defects, or

c. That the pregnancy resulted from rape or incest.

We are fully aware that there is a wide divergence of opinion on the subject of therapeutic abortion. Some physicians and citizens believe the present laws should be retained while others call for the abolition of all abortion laws. The California Medical Association supports Senator Beilenson's moderate position as one that is reasonable, medically justified and in the best interest of the patient and quality medical care.

In Memoriam

ARONSTEIN, ERNST, Fresno. Died 16 November 1966, in Fresno, aged 72, of hemorrhage, duodenal ulcer, and myocardial infarction. Graduate of Universität Köln (Cologne) Medizinische Fakultät, Köln, Prussia, Germany, 1920. Licensed in California in 1935. Dr. Aronstein was a retired member of the Fresno County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✣

CLAMAN, ZALMAN BERNARD, Los Angeles. Died 2 March 1967, in Rochester, Minnesota, aged 45, of polyarteritis nodosa. Graduate of the University of Manitoba Faculty of Medicine, Winnipeg, Canada, 1945. Licensed in California in 1960. Doctor Claman was a member of the Los Angeles County Medical Association.

✣

FORTEZA, ROSENDO JR., Los Angeles. Died 31 January 1967, aged 60, of heart disease. Graduate of Ohio State University College of Medicine, Columbus, 1939. Licensed in California in 1948. Doctor Forteza was a member of the Los Angeles County Medical Association.

✣

GARN, DON CARLOS, Bakersfield. Died 17 February 1967, in Bakersfield, aged 66, of cancer. Graduate of the College of Osteopathic Physicians and Surgeons, 1932. Licensed in California in 1932. M.D. degree from California College of Medicine, 1962. Doctor Garn was a member of the Kern County Medical Society.

✣

GODDARD, EDWARD P., Oroville. Died 5 February 1967, in Ogden, Utah, aged 48, of heart disease. Graduate of the University of Oregon Medical School, Portland, 1944. Licensed in California in 1955. Doctor Goddard was a member of the Butte-Glenn Medical Society.

✣

GRIFFITH, WILLIAM HUGH, Los Angeles. Died 13 February 1967, in Los Angeles, aged 68, of cardiac failure, coronary occlusion. Graduate of the University of Minnesota Medical School, Minneapolis, 1925. Licensed in California in 1939. Doctor Griffith was a member of the Los Angeles County Medical Association.

✣

HAMRICK, JEWELL G., Santa Ana. Died 7 February 1967, in Orange, aged 62, of congestive heart failure. Graduate of the University of Tennessee College of Medicine, Memphis, 1940. Licensed in California in 1946. Doctor Hamrick was a member of the Orange County Medical Association.

JOSLIN, ERIC FRANCIS, Visalia. Died 11 February 1967, in Pacific Grove, aged 65. Graduate of Albany Medical College, New York, 1933. Licensed in California in 1957. Doctor Joslin was a member of the Tulare County Medical Society.

✣

LOW, CHIN Y., San Francisco. Died 5 March 1967, in San Francisco, aged 64. Graduate of Northwestern University Medical School, Chicago, Illinois, 1928. Licensed in California in 1928. Doctor Low was a member of the San Francisco Medical Society.

✣

MCGEE, WILLIAM (BUSTER), San Diego. Died 15 February 1967, in San Diego, aged 75. Graduate of the University of Maryland School of Medicine, Baltimore, 1928. Licensed in California in 1935. Doctor McGee was a member of the San Diego County Medical Society.

✣

MOORMAN, HENRY DEAN, Pasadena. Died 29 January 1967, in Crestline, aged 48, of acute coronary disease. Graduate of Harvard Medical School, Boston, Mass., 1943. Licensed in California in 1950. Dr. Moorman was a member of the Los Angeles County Medical Association.

✣

MUNOZ, RAFAEL, Rolling Hills. Died 23 February 1967, in Rolling Hills, aged 46. Graduate of Boston University School of Medicine, Massachusetts, 1945. Licensed in California in 1952. Doctor Munoz was a member of the Los Angeles County Medical Association.

✣

ROGERS, THOMAS L., Long Beach. Died 2 February 1967, aged 88, of congestive heart failure. Graduate of State University of Iowa College of Medicine, Iowa City, 1909. Licensed in California in 1910. Doctor Rogers was a member of the Los Angeles County Medical Association.

✣

ROSNER, SAMUEL, Santa Ana. Died 7 February 1967, in Anaheim. aged 59. Graduate of the University of Edinburgh Faculty of Medicine, Edinburgh, Scotland, 1934. Licensed in California in 1945. Doctor Rosner was a member of the Orange County Medical Association.

✣

SCHWARTZ, ARTHUR H., Beverly Hills. Died 16 February 1967, in Beverly Hills, aged 83, of coronary thrombosis. Graduate of Rush Medical College, Chicago, Illinois, 1904. Licensed in California in 1926. Doctor Schwartz was a member of the Los Angeles County Medical Association.

SHEPARDSON, HARRY CLARE, San Francisco. Died 5 March 1967, in San Francisco, aged 78. Graduate of the University of California School of Medicine, Berkeley-San Francisco, 1924. Licensed in California in 1924. Doctor Shepardson was a member of the San Francisco Medical Society.

✣

SKLEBA, LEONARD FREDERICK, Glendale. Died 1 February 1967, in Glendale, aged 75, of middle cerebral thrombosis. Graduate of Loyola University (Bennett Medical College), 1913. Licensed in California in 1952. Doctor Skleba was a member of the Los Angeles County Medical Association.

✣

VINCENT, ELMO H., Rolling Hills Estate. Died 8 October 1966, in Torrance, aged 48, of coronary infarction. Graduate of the College of Osteopathic Physicians and Surgeons, 1953. Licensed in California in 1954. M.D. degree from California College of Medicine, 1962. Doctor Vincent was a member of the Forty First Medical Society.

✣

WYCKOFF, HARRY A., Piedmont. Died 26 February 1967, aged 88. Graduate of Stanford University School of Medicine, Palo Alto-San Francisco, 1916. Licensed in California in 1916. Doctor Wyckoff was a retired member of the San Francisco Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✣

ZIEGLER, JULIUS, Los Angeles. Died 22 February 1967 in Arcadia, of myocardial infarction, aged 68. Graduate of Hamburgische Universität Medizinische Fakultät, Hamburg, Germany, 1924. Licensed in California in 1939. Dr. Ziegler was a member of the Los Angeles County Medical Association.

17th annual redwood regional conference

KONOCTI HARBOR INN
Clear Lake
Thurs. noon—Sat. noon
April 27-29, 1967

REDWOOD REGIONAL CONFERENCE

Presented cooperatively by North Coast Counties Medical Societies, Stanford University School of Medicine, and California Medical Association Committee on Continuing Medical Education. A 12-hour course.

HOST: **Mendocino-Lake County Medical Society**

Regional Chairman: **D. E. Coolahan, M.D.**

Institute Fee: $15.00. For additional information contact Continuing Medical Education office, California Medical Association, 693 Sutter Street, San Francisco 94102. All California Medical Association members and their families are cordially invited to attend.

PROGRAM THEMES:

CARDIOVASCULAR PROBLEMS . . . EAR, NOSE AND THROAT . . . ALLERGY . . . EYE PROBLEMS . . . MYOSITIS

THURSDAY, APRIL 27

Afternoon Session

2:00—**Office Procedures Involving the Ear, Nose and Throat**—John M. Fredrickson, M.D.

2:45—**Office Procedures Involving the Eye**—Frank C. Winter, M.D.

3:15—**Problems in the Therapy of the Allergic State**—William McKee, M.D.

4:15—CONCURRENT WORKSHOPS (you may go to one of your choice)

A. **Demonstration of ENT Techniques Usable in the Office**—John M. Fredrickson, M.D.
B. **Demonstration of Fundoscopic Examination**—Frank C. Winter, M.D.
C. **Seminar on the Specific Problems of the Allergic Patient**—William McKee, M.D.

FRIDAY, APRIL 28

Morning Session

8:30—**Cardiovascular Problems**—The Useful Studies for Patients Selected for Surgical Therapy

A. **Peripheral Vascular Disease**
B. **Cerebral Disease and Stroke**
C. **Cardiac Valvular Replacement**

Panel: Roy B. Cohn, M.D.
Edward J. Hurley, M.D.
Alfred P. Spivack, M.D.

11:00—CONCURRENT WORKSHOPS

A. **Use of the Defibrillator**—Edward J. Hurley, M.D.
B. **Wet Clinic: Patients with Cardiac Disease**—Alfred P. Spivack, M.D.

Afternoon Session

1:30—**Luncheon: New Horizons in Research**

DNA MOLECULES WITHOUT ENDS IN TUMOR VIRUSES AND TEMPERATE PHAGES—Dale Kaiser, Ph.D., Professor of Biochemistry, Stanford University School of Medicine

SATURDAY, APRIL 29

Morning Session

8:30—**Coronary Care Unit: Should a Small Hospital Have One?**—Alfred P. Spivack, M.D.

9:30—**Recent Additions to Laboratory Examinations Useful to the Clinician**—Frederick A. Fox, M.D.

10:15—**Useful Physiotherapeutic Measures in Rehabilitation**—Leslie W. Knott, M.D.

11:15—CONCURRENT WORKSHOPS

A. **Wet Clinic Patients with Peripheral Vascular Disease**—Roy B. Cohn, M.D.
B. **Cerebral Vascular Disease**—Edward J. Hurley, M.D.
C. **Physiotherapeutic Procedures**—Leslie W. Knott, M.D.

17th annual regional postgraduate institute

AHWAHNEE HOTEL
Yosemite
May 5-6, 1967

SAN JOAQUIN VALLEY COUNTIES

Presented cooperatively by San Joaquin Valley Counties Medical Societies, Continuing Education in Medicine and Health Sciences, University of California Medical Center, San Francisco, and the Committee on Continuing Medical Education, California Medical Association. A 12 hour course.

HOST: Fresno County Medical Society
***Regional Chairman:* Irwin Barg, M.D.**

Institute Fee: $15.00. For additional information contact Continuing Medical Education office, California Medical Association, 693 Sutter Street, San Francisco 94102. All California Medical Association members and their families are cordially invited to attend.

GUEST SPEAKER: Victor McKusick, M.D., Chief, Division of Medical Genetics, Johns Hopkins University School of Medicine.

PROGRAM THEMES:

PROGRESS IN CLINICAL PRACTICE: THE EMERGING GENETIC ASPECTS . . . GASTROENTEROLOGY

FRIDAY, MAY 5

Morning Session

9:00—**Constitutional and Environmental Interactions**—Piero Mustacchi, M.D.

9:20—**Genetic Determinants of Health and Disease**—Victor McKusick, M.D.

10:00—**Sex Chromosome Abnormalities**—Melvin M. Grumbach, M.D.

10:45—CONCURRENT WORKSHOPS (you may go to one of your choice)
- A. **Pinpointing the Causes of Maternal Mortality**—Gilbert A. Webb, M.D., and Joseph H. Kushner, M.D.
- B. **Metabolic Problems in Daily Practice**—Felix O. Kolb, M.D., and Melvin M. Grumbach, M.D.

12:15—**Wine Tasting and Get Acquainted Session**—Doctors and their wives are invited

Afternoon Session

2:00—**Genetic Aspects of Childhood Neoplasia**—Joseph H. Kushner, M.D.

2:30—**Cancer of the Stomach: Genetic and Environmental Interaction**—Leon Goldman, M.D.

3:00—**The Implications of Intestinal Polyposis**—Walter Birnbaum, M.D.

3:30—**Coffee Break**

3:50—CONCURRENT WORKSHOPS
- A. **An Overview of Contemporary Genetics**—Victor McKusick, M.D., and Nicholas L. Petrakis, M.D.
- B. **The Patient with a "Gastric Ulcer"**—Hugo C. Moeller, M.D., and Leon Goldman, M.D.

SATURDAY, MAY 6, 1967

Morning Session

9:00—**Genetic Control of Hypertension**—Victor McKusick, M.D.

9:30—**Genetic Factors in Renal Stone Formation**—Felix O. Kolb, M.D.

10:00—**Genetic Factors in Anemia**—Nicholas L. Petrakis, M.D.

10:30—**Coffee Break**

10:45—CONCURRENT WORKSHOPS
- A. **The Patient with Pancreatic Disease**—Leon Goldman, M.D., and Hugo C. Moeller, M.D.
- B. **Common Endocrinologic Problems in Daily Practice**—Gilbert A. Webb, M.D., Felix O. Kolb, M.D., and Melvin M. Grumbach, M.D.

Afternoon Session

2:00—**Ulcerative Colitis: The Point of View of the Internist**—Hugo C. Moeller, M.D.

2:45—**Ulcerative Colitis: The Point of View of the Surgeon**—Walter Birnbaum, M.D.

3:30—**Question Period**

3:45—**Coffee Break**

4:00—CONCURRENT WORKSHOPS
- A. **The Colostomy Patient, a Combined Approach**—Hugo C. Moeller, M.D., and Walter Birnbaum, M.D.
- B. **The Patient with an Abnormal Blood Count**—Joseph H. Kushner, M.D., Nicholas L. Petrakis, M.D., and Piero Mustacchi, M.D.

PUBLIC HEALTH REPORT

Lester Breslow, M.D., M.P.H.
Director, State Department of Public Health

WHILE ERADICATION of tuberculosis may seem a lofty goal, control of the disease is close at hand. The immense reduction of deaths due to this cause, coupled with easier management, has made us complacent. Yet 5,000 new cases of tuberculosis are reported annually, and 600 Californians die of it each year.

The disease has retreated into pockets of poverty, attacking the very young and the very old. Urban communities, especially depressed "Skid Row" neighborhoods, harbor the disease, but it is not alien to middle-class or rural settings. Medicare and Medi-Cal, by encouraging free choice of physicians, have placed increased responsibility on private-practicing physicians to achieve tuberculosis control.

The importance of maintaining consistent, quality care for tuberculous persons and stemming the infectious potential were reasserted recently in a Los Angeles symposium on Rational Therapy and Control of Tuberculosis.

Clinicians were urged to apply these practices:

- Sound bacteriologic procedures to establish an accurate diagnosis, to test drug sensitivity and to monitor response to drug therapy.
- Routine tuberculin skin testing of persons at high risks.
- Prompt chemotherapeutic treatment to render the disease in individual cases non-communicable.

Symposium speakers also advocated:

- Daily drug administration for a minimum of two years.
- Use of secondary drugs as needed for organisms resistant to primary drugs.
- Case reporting to expedite identification and treatment of the source case and contacts, and for lifetime follow-up of all infected persons.

Good laboratory support is fundamental to controlling tuberculosis. Skilled laboratory work can distinguish between cases of *M. Tuberculosis* and atypical mycobacteria, and can determine drug susceptibility. Most local health department laboratories, as well as some other laboratories, are equipped to perform diagnostic tests for tuberculosis. The Microbiology Laboratory of the State Health Department performs confirmatory identifications and susceptibility tests for secondary drugs used in treating tuberculosis.

Skin testing remains the most reliable index of tuberculous infection. The size of the tuberculin skin reaction roughly parallels the risk that clinical tuberculosis will develop. Patients with old untreated infections have a high risk of clinical disease. Others at high risk include those with diabetes, silicosis or subtotal gastrectomy, alcoholics, infants, young adults and those receiving long-term steroid therapy.

Pediatricians should perform tuberculin tests routinely on children when they begin to go to school, using a test dose of 5 tuberculin units. As these children have limited personal contacts, a source case can be found more easily, and contacts brought to examination. Adolescents should be similarly tested.

If a positive reaction is 10 mm or more in diameter, *M. Tuberculosis* is more likely the infective agent than one of the atypical strains. Mean positive reaction size to PPD-S (purified protein derivative, standard) has been shown to be 15 mm among tuberculous patients in hospital. Reactions under 10 mm to PPD-S are apt to reflect an infection with an atypical mycobacterium rather than *M. Tuberculosis.* If persons with reactions less than 10 mm are retested in one to two months, many will have negative response, indicating cross-reactions of atypical mycobacteria to PPD-S. Atypical strains, conversely, will react more strongly to specific antigens—PPD-R (Battey), PPD-K (Kansasii) and PPD-G (Scotochromagens).

Prompt administration of prophylactic drugs greatly reduces the risk that active disease will develop in contacts and "converters," that is, persons who convert from negative to positive reaction to tuberculin. Persons at greatest risk of active disease, for whom chemoprophylaxis or chemotherapy is essential, include: Those with old cases of tuberculosis lacking chemotherapy or adequate treatment; converters, especially under age six;

and persons with silicosis or diabetes who have been exposed to the disease. Recommended for converters and contacts is prophylactic treatment with isoniazid (INH) for one year. Benefits of INH greatly outweigh any deleterious side-effects, such as gastrointestinal irritation. As recently as 30 June 1966, only 14 per cent of known contacts with positive reactions and converters in the United States were receiving INH. If tuberculosis is to be controlled, we must take full advantage of effective drug therapy and chemoprophylaxis.

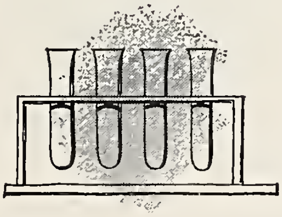
Drug resistance introduces another management difficulty. Laboratory test results indicate that 7 per cent of newly diagnosed and previously untreated cases of tuberculosis are drug-resistant to one or more of the primary drug triad. More significantly, over 60 per cent of reactivated cases previously treated—intermittently—with drugs are now drug-resistant. Sensitivity testing, plus administration of secondary drugs, can insure better control of this important disease reservoir.

Today's management of the recognized tuberculous patient is radically different from the isolation of the past. Yet problems of reinfection and lifetime follow-up remain unaltered. Shortening the stay in hospital has greatly improved patients' cooperation, even though longer periods of treatment as a hospital outpatient or in a physician's office is then necessary. As little as four to six months in hospital, often in a community general hospital rather than a remote sanitorium, may suffice, provided follow-up treatment is carried out. Breaks in medication, premature discontinuance or inadequate drug dosage substantially increase dangers of the development of resistant tubercle bacilli and of later reactivation.

Drug therapy should continue uninterrupted for 18 months or more after discharge from hospital. Lifetime follow-up and reexamination patterns must be firmly established if tuberculosis is to be controlled. A patient should be seen a month after discharge from hospital, then at least once every one to three months during therapy for tuberculosis and at least once every six to twelve months after discontinuance of therapy for the rest of his life. X-ray films of the chest should be made every six to twelve months, with bacteriologic examinations as indicated.

California's local health departments, where tuberculosis control programs are centralized, can offer clinicians considerable support. A prompt report of a verified case hastens finding the source case and the examination of contacts. Essential case follow-up is only possible if cases are reported for entry in the state's central registry. "Lost" patients are searched for and restored to treatment through procedures maintained by local and state health departments. In 14 of California's largest jurisdictions, project grants now support expanded tuberculosis control activities. These range from the provision of drugs, laboratory and x-ray services and medical consultation, to public health nursing and social service follow-up. These services are available to patients of private physicians who wish to use them.

Other activities include maintaining special neighborhood chest clinics, operated at times and in locations convenient to clusters of patients. Imaginative new approaches have greatly improved case-finding and treatment—and the early return of patients with tuberculosis to their communities.



WOMAN'S AUXILIARY

to the California Medical Association

THE MENDOCINO-LAKE Medical Society will again administer the Nurse Scholarship Loan from its Poliomyelitis Fund. The 25-cent charge per person for the Sabin vaccine administered in both counties left a reserve after all expenses were paid. The Medical Society decided to use this fund for a revolving nurse scholarship loan fund. If the student, upon graduating, returns to either Mendocino or Lake County to work for one year, her loan is forgiven. Otherwise she is to repay the loan, commencing six months after graduation, at the rate of $10 or more per month. The loan is interest-free.

The Mendocino-Lake Medical Auxiliary has again been asked to serve in fields of nurse recruitment and publicity. The Auxiliary is to act as a preliminary screening committee and the final decisions will be handled by the Executive Committee of this Fund, composed of representatives from lay groups from both counties as well as representatives from the medical society.

It was decided that $500 should be given each year, to be divided between two deserving applicants, preferably one from Mendocino County and one from Lake County.

College entrance examinations have recently been given and prospective nurse applicants are now applying to their prospective schools.

Letters regarding the availability of this scholarship loan fund have been sent to every high school in the area. In addition, articles regarding the loans have been sent to all newspapers in the two counties.

To date, one applicant has been interviewed.

MRS. THOMAS A. NICHOLSON
Chairman Mendocino-Lake Auxiliary
Nurse Recruitment Committee
350 North Spring, Ukiah 95482

INFORMATION

Professional Registered Nurses In the United States and California, 1962

A Report of the Bureau of Research and Planning, California Medical Association

• *Figures on professional registered nurses recently made available indicate that the ratio of active nurses to population in California in 1962 was 327 per 100,000 persons. The comparable figure nationally was 298/100,000. California ranked twentieth out of the 50 states and the District of Columbia.*

The percentage of all nurses actively engaged in nursing is considerably lower in California than in the total United States: 60.4 per cent compared with 65.3 per cent. This indicates that the shortage of nurses in California is more attributable to underutilization of potential manpower than to its absence.

There are some identifiable categories of nurses in California in which the utilization of personnel is particularly low, as compared with other states. In terms of rates of utilization, the group of nurses over 60 years of age are most noticeably underutilized, while in terms of numbers, married nurses could provide the greatest supply of extra nurses were their employment rate in the state equal to the national rate.

The recruiting potential is a very great one, however, as this report indicates.

The American Nurses' Association has recently published detailed information on professional registered nurses in the United States for the year 1962. The publication* contains considerable data from which a number of observations concerning the nursing shortage and approaches to remedies can be made. This report presents some of the basic information, and attempts to shed light on the situation in California as it compares both with that in the total United States and with the situation in states with particularly high nurse/population ratios as well as with those with particularly low ratios.

Table 1 shows that in 1962 there were a total of 847,531 professional registered nurses in the United States and 92,265 in California. Of these totals, 532,118 in the nation and 52,151 in California were, according to responses to the American Nurses' Association survey, active in the nursing field at that time. Excluding the proportion of nurses who did not state whether or not they were actively employed in nursing, the percentages employed were 65.3 per cent in the United States and a considerably lower 60.4 per cent in California.

The table also indicates employment status by two variables: marital status and age. The per cents of nurses in each category who are actively employed in the nursing field are parallel in the state and the nation; however, in almost all cases the percentage in California is somewhat lower than it is in the total United States. Such comparisons constitute a valuable indication of manpower pools of available personnel.

On the basis of marital status, it is apparent that the highest per cent employed are single persons, followed closely by those who are classified as divorced or separated. Nationally, over three out of four widows are actively employed and, ranking last by a fairly substantial margin, are those who are currently married. Slightly under three out of five of the nation's married nurses are active in the field. In California, the percentage of working, married nurses is even lower—just 51.4 per cent are actively engaged in nursing.

Figures are also shown by age group. The same pattern is indicated in California as in the rest of the nation, although the per cent employed is consistently lower in the state.

Young nurses (under 30) show a high proportion employed in the field, with almost seven in 10 (69.9 per cent) actively engaged in nursing in both the state and the nation. The proportion drops significantly during the next decade (30 to 39 years) when many women are raising their families. Nationally, the proportion who remain active dropped to 57.0 per cent and in California to 53.7 per cent.

After age 40, nurses begin returning to work. For nurses aged 40 to 49, the proportion actively employed jumped to 67.8 per cent nationally and to 62.4 per cent in California. The divergence in the activity pattern now becomes apparent, with California dropping behind in the proportion of

---

Reprint requests to: 693 Sutter Street, San Francisco 94102.

*American Nurses' Association: The Nation's Nurses—Inventory of Professional Registered Nurses, New York City, 1965.

TABLE 1.—*Numbers and Per Cent of Professional Registered Nurses in the United States and California in 1962: Marital Status and Age Group for All Nurses and for Those Employed in Nursing*

Professional Registered Nurses	*United States*				*California*			
	*Total**		*Employed*		*Total**		*Employed*	
	Number	*Per Cent*	*Number*	*Per Cent*	*Number*	*Per Cent*	*Number*	*Per Cent*
Total	847,531	100.0	532,118	62.8 (65.3)	92,265	100.0	52,151	56.5 (60.4)
Marital Status:								
Single	146,277	100.0	131,768	90.1	15,151	100.0	13,301	87.8
Married	545,112	100.0	313,993	57.6	57,647	100.0	29,652	51.4
Widowed	40,013	100.0	30,191	75.5	4,996	100.0	3,312	66.2
Divorced or Separated	29,817	100.0	26,599	89.2	5,160	100.0	4,436	86.0
Status not Reported	26,476	100.0	12,345	46.6	3,372	100.0	1,450	43.0
Age Group:								
Under 30	188,581	100.0	131,901	69.9	17,547	100.0	12,271	69.9
30-39	229,269	100.0	130,692	57.0	22,711	100.0	12,187	53.7
40-49	179,881	100.0	121,947	67.8	20,871	100.0	13,022	62.4
50-59	129,080	100.0	94,018	73.4	14,785	100.0	9,888	66.9
60 and over	61,541	100.0	36,666	59.6	7,980	100.0	3,706	46.4
Age not Reported	26,585	100.0	15,894	59.8	2,432	100.0	1,077	44.3

---

*Overall total includes nurses whose employment status was not reported; totals by variables shown exclude those whose employment status was not reported. Per cent in parentheses exclude unstated employment status.

nurses who return to an active status. The trend continues and magnifies in the next decade, during which a high point is reached nationally with 73.4 per cent of nurses between 50 and 59 actively employed in the field; in California, only 66.9 per cent are actively engaged in nursing during this period in their lives. The percentage in the state, in fact, is lower than that shown for nurses under 30, a situation opposite to the national pattern.

After age 60, nurses begin to retire from activity, and the proportion who remain actively engaged in the field drops rather sharply. Because of the open-ended nature of the category, it is, in fact, revealing that such a high proportion showed continued activity. Perhaps the same high proportion continue to engage themselves in nursing through age 65 as was shown for nurses aged 50 to 59, however, this cannot be determined from the information available. Almost three out of five (59.6 per cent) nationally indicated continued activity after age 60. In California, however, fewer than half of all nurses (46.4 per cent) over age 60 were actively employed in the profession.

Tables 2 and 3 present some comparative data for other selected states, showing (a) numbers of nurses and nurse/population ratios for all nurses and for those who are actively employed and (b) comparative employment ratios by selected variables in which was shown the most divergence between the state and the nation.

Table 2 shows numbers and rates per 100,000 population of total nurses and of active nurses for the United States, for the seven states which rank highest in active nurse/population ratios, for the seven which rank lowest and for California. This table, as well as Table 3, excludes the District of Columbia which led the nation in active nurse/population ratios; the District presents a situation which is atypical and, hence, would not be meaningful if used for comparative purposes.

In the total United States the 847,531 nurses in

TABLE 2.—*Numbers and Rates Per 100,000 Population of Total Nurses and Active Nurses, for the U.S., the Seven Highest- and Seven Lowest-Ratio States, and California, 1962*

	*Total Nurses**		*Active Nurses*	
Area	*Number*	*Rate Per 100,000†*	*Number*	*Rate Per 100,000‡*
United States	847,531	456	532,118	298
New Hampshire	4,832	777	2,947	494
Vermont	2,749	710	1,660	448
Massachusetts	42,854	826	21,928	514
Rhode Island	5,062	577	3,381	397
Connecticut	20,467	780	11,365	440
New York	103,026	589	67,199	388
Delaware	3,054	654	1,775	393
Kentucky	8,178	265	5,317	175
Tennessee	8,836	242	6,324	178
Alabama	7,201	217	5,047	158
Mississippi	4,128	183	3,141	142
Arkansas	3,229	175	2,194	121
Oklahoma	5,927	242	3,948	164
Texas	27,658	273	17,222	173
California	92,265	542	52,151	327

---

*Includes nurses whose activity status was unknown.
†Calculated by Bureau of Research and Planning based on U.S. Census estimates.
‡As shown in source reference, The Nation's Nurses.

1962 constituted a ratio of 456 per 100,000 civilians; the comparable ratio in California was 542 per 100,000. This measure, however, is not so important as the ratio of *active* nurses to population; in this category, the 532,118 nurses represented 298 for every 100,000 persons nationally; California had 327 active nurses per 100,000 persons. Observing both ratios, however, one can make some judgments about two important aspects of the nursing manpower situation. The first is that in the event of extreme emergency a substantial nurse-power reservoir could be tapped. The second relates to the question as to what extent existing manpower is being utilized. It is the second consideration to which the remainder of this report will be addressed.

California's active nurse/population ratio ranks twentieth among the 50 states and the District of Columbia. Table 2 shows the seven states which rank highest; these are all eastern seaboard states and, with the exception of Delaware, northern states. Five are in New England. The seven states with rank lowest include all of the south central states except Louisiana. It can be seen that the ratio in California is considerably closer to (though lower than) the top seven than to the bottom seven; however, it is well behind some of the leaders. It might also be noted that California's physician/population ratio ranked fourth in the nation at that time. Also noteworthy is the fact that in California there were 1.64 active registered nurses for every physician in 1962, while the national ratio was 1.99 and, in the northeast, 2.25 active nurses for every physician.*

Table 3 shows per cents of nurses who are actively employed in the nursing field in seven high nurse/population ratio states, seven low-ratio states and in California. It can be seen that the per cent employed tends to be considerably higher in low-ratio states, with an average of 70.0 per cent employed compared with 63.1 per cent in high-ratio states. It would appear, therefore, that in areas where demand is greatest, nurses tend to demonstrate a tendency to return to the ranks of the employed.

In any event, the percentage of nurses actively employed in California is below not only the low-ratio states and the national average, but also below the average of the seven high-ratio states. The meaning of this is that California's potential active nurse/population ratio is considerably higher than is its actual ratio. It can therefore be speculated that existing nursing talent in California is being underutilized and that the current shortage could be ameliorated considerably without any increase

---

*See Socio-Economic Report, Vol. V, No. 12, November 1965 for information concerning physician/population ratios (Also published in California Medicine, 104:334-337, April 1966), as well as data on other health occupations and Vol. VI, No. 4, May 1966 for data on hospital-based personnel.

TABLE 3.—*Per Cent of Professional Registered Nurses Employed in the Nursing Field, in Seven States with Highest Nurse/Population Ratios, Seven States with Lowest Ratios, and in California, by Selected Variables, 1962*

States	*Total, All Nurses Per Cent*	*Age or Marital Status Variable*				
		Age 40-49 Per Cent	*Age 50-59 Per Cent*	*Age 60 and Over Per Cent*	*Married Per Cent*	*Widowed Per Cent*
National average	65.3	67.8	73.4	59.6	57.6	75.5
—with highest ratios*						
Massachusetts	62.3	67.2	74.2	52.2	52.5	72.1
New Hampshire	63.6	69.1	74.7	39.1	57.1	76.2
Vermont	63.0	68.2	73.8	57.3	55.8	79.2
Connecticut	56.5	61.3	63.5	45.2	49.7	61.7
Rhode Island	69.1	72.8	78.3	66.2	61.2	75.3
Delaware	60.1	64.1	61.8	62.5	52.7	76.8
New York	65.9	66.0	74.5	64.9	54.4	77.2
—average	63.1	67.0	71.5	55.5	54.8	74.1
—with lowest ratios†						
Arkansas	68.8	72.9	73.2	53.1	63.6	76.4
Mississippi	77.8	79.4	85.1	71.7	73.8	84.0
Alabama	72.9	77.3	80.9	65.1	67.7	79.4
Oklahoma	67.6	70.8	77.9	69.4	63.3	80.3
Texas	63.2	64.2	68.7	59.7	‡	‡
Kentucky	65.9	68.7	70.2	54.6	58.3	71.2
Tennessee	73.5	75.5	82.5	75.2	67.9	83.2
—average	70.0	72.7	76.9	64.1	65.9	79.1
California	60.4	62.4	66.9	46.4	51.4	66.2

*Ranked from highest to lowest.
†Ranked from lowest to highest.
‡Data by marital status not available

in the number of new nurses entering the profession.

Per cents of nurses employed in several states are shown in Table 3 according to five selected age-marital status variables. An examination of these comparisons can be indicative of the areas wherein it is most likely that more nurses could be urged to return to activity.

A comparison between the percentage employed in California and in other states shows that the greatest disparity is among nurses aged 60 and over, followed by widows, those aged 50 to 59, married persons, and those in the age group 40 to 49. In other categories not shown in Table 3, California is average or above average in per cents employed. In all of the categories shown, even those states with the highest active nurse/population ratios exceed California in the per cent of nurses employed.

Similarities can be observed between some of the age-marital status categories; many of the same persons shown in the categories over 50 are widows, and many of those who are aged 40 to 49 are those who are married. Data were not cross-classified in the source material, however; therefore, these variables cannot be isolated or analyzed in greater depth.

Nurses aged 60 and over are probably underemployed in California. Only 46.4 per cent of all nurses in this age group are actively engaged in their profession, while the average of seven states with the highest active nurse/population ratios is 55.5 per cent; the national average is 59.6 per cent; and the average in states with low ratios is 64.1 per cent.

Widows constitute the next most promising pool of nurses in terms of the disparity between those employed and those who might be expected to be in the labor market. Fewer than two out of three California nurses who are widowed are employed, while over three-fourths (75.5 per cent) are employed nationally and almost four-fifths (79.1 per cent) are employed in low active nurse/population ratio states. The average per cent employed in high-ratio states also exceeds that in California by a considerable margin.

Among those 50 to 59 years of age, a group in which the activity level is generally quite high, only two out of three California nurses (66.9 per cent) are active. In high-ratio states the average is 71.5 per cent; nationally it is 73.4 per cent and in low-ratio states it is 76.9 per cent.

Married nurses show a greater tendency in California to be inactive than they do elsewhere in the nation. Although the differential is not so great as in the previous three categories, the potential in terms of numbers of nurses is far greater, since over two-thirds (66.8 per cent) of *all* nurses in California are married. If the percentage employed in the state matched the national average, it would add almost 3,600 nurses in the current supply. Married nurses appear to be a very important source of manpower in low-ratio states, judging from the disparity between the per cent employed nationally and those employed in these seven states.

Nurses aged 40 to 49, who often are the same ones as those shown in the prior category, provide an important source for potential manpower; however, they do not constitute a particularly well defined group. This is unfortunate, since even high-ratio states show a greater proportion of employed nurses in this age group than is shown in California.

It should be noted that these comparisons should only be used as general indicators of identifying unused nursing resources which could most easily be activated to ameliorate current shortages. Comparisons between and among various states are always subject to difficulties due to inherent interregional differences. Nevertheless, there appears to be strong evidence that, in California more than elsewhere, any nursing shortage which exists is more attributable to underutilization than to undersupply. Certainly, this important ratio lags behind that for other health manpower personnel. There is only one important area in the health manpower field where California ranks as low as twentieth nationally; unfortunately, nursing happens to be the one.

Medicolegal Responsibilities In Physician-Laboratory Relations

DON HARPER MILLS, M.D., LL.B., *Los Angeles*

HOW MANY PHYSICIANS could practice as good medicine and surgery without laboratory assist-

---

Dr. Mills is Associate Clinical Professor of Forensic Medicine and Pathology, University of Southern California, School of Medicine.

Reprinted with permission from Texas State *Journal of Medicine,* 61:865-866, December 1965.

ance? Indeed, has not laboratory medicine itself done much to extend medical competency in many fields? Though traditionally of diagnostic value, laboratories now can provide means for better understanding of disease process and its management. A glance at the advances in enzyme chemistry should convince most doubters of this, and more is on the way. Of course, the laboratory does not "treat" patients in the usual sense of that word, but it no longer has the status of being a mere tool for medical practitioners. This is something all doctors should realize, for the use of and knowledge about laboratory medicine is being injected more and more into the responsibilities expected of physicians.

Clinical laboratories seldom have been involved directly in malpractice cases, but this does not mean they lack potential liability problems.[1] Rather, their immunity seems to stem from a combination of factors over which they have little control. First, laboratories have less contact with patients than do practicing physicians, and this reduces the degree of interpersonal conflict from which many suits arise. Second, when actual contacts are made with patients, the procedures performed usually possess fewer risks of physical injury when compared with medical and surgical manipulations carried out by attending physicians. Third, with respect to specific test performance, patients find it extremely difficult to prove that an apparent incorrect result was due to negligent conduct (particularly when a test consumes the entire specimen, leaving nothing for subsequent checking). However, as attorneys representing patients become more medically oriented, I suspect this factor will afford diminishing protection for laboratories.

The fourth and perhaps most significant protector of laboratories is the medicolegal insulation provided by the "ordering" physician. It is he who decides a test is necessary despite whatever risk of injury it may involve. It is he who may have the responsibility of preparing the patient to assure an adequate specimen for dependable laboratory results. It is he who must interpret the results, sometimes to the extent of detecting incorrect information in light of other knowledge he possesses about his patient. That is, in spite of possible laboratory negligence, he may be liable for relying on a test answer which he should have known was false. Finally, he may even "protect" a substandard laboratory by exposing himself to primary liability for relying on such a laboratory when he knows or should know it does not deserve his reliance.

While completely divorced from possible laboratory liability, doctors face additional medical and legal risks by failing to order tests when indicated, by ordering the wrong test when another is mandatory, by failing to follow-up prior results with further testing to assure adequate management and disease response, or by failing to recognize the limitations of reliability inherent in many tests even when correctly performed.

Each of these points has posed liability threats to some physicians at one time or another; and, as laboratory technology becomes more intricate, these "protective" hazards will become more prominent in malpractice suits. Suits directly against laboratories may be on the increase, but this will offer no solace to practicing physicians.

Is it possible for active doctors to keep abreast of each new test that comes along or even with refinements of basic tests? If not, how are they to fulfill potential responsibilities of knowing what to order, when to order, how to assure proper specimens, how to interpret, and what to do subsequently? When these issues arise in malpractice cases, they can become very real threats to legal security.

Resolution of many pitfalls in the physician-laboratory relationship rests primarily on communication. How often do physicians consult laboratories concerning available tests, interpretations, and other information? If the laboratory director is a physician, he should be the best source of continuing education and assistance; yet it appears this is frequently overlooked. Clearly, utilization of this aspect of laboratory service will benefit both physicians and their patients.

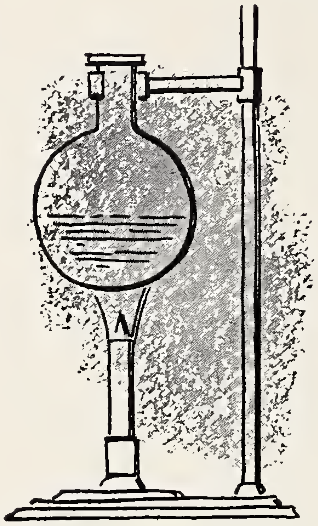
Many laboratories operated by non-physicians offer excellent technical services; however, adequate clinical advice from these sources is often lacking. Using them may be justified in the absence of physician-operated units, but it must be remembered that some of them do not meet the test of excellence. Therefore, a form of examination and certification is vital. Only then can doctors establish a right of reliance without personal evaluation of the facilities.

Economy alone is not a sound reason for eliminating necessary clinical-pathological consultation, yet it is distressing to note the trend of using out-of-state laboratories for tests which could be performed locally. Without an on-the-spot examination by the doctor, he may find it difficult, if not impossible, to prove his right to rely on that laboratory. Additionally, I have yet to find an out-of-state laboratory which is willing to commit itself in advance to local jurisdiction in the event of legal action.

The trend toward better medicine and the harassment by mounting malpractice litigation demand recognition of the value of physician-operated laboratories. Improved communication with physician-directors will do much to prevent liability risks which are now developing.

REFERENCE

1. Mills, D. H.: Malpractice and the clinical laboratory, Science, 144:638, 8 May 1964.



NEWS & NOTES

NATIONAL • STATE • COUNTY

ALAMEDA

The 1967 Postgraduate Seminar and **Clifford D. Sweet Memorial Lecture** will be held at Children's Hospital Medical Center of Northern California, Oakland, May 12 and 13. Pediatricians and general practitioners in the 10 western states are invited to attend.

The Sweet Lecture will be delivered May 12 at 6 p.m. by William Boyd, Ph.D., vice chancellor in charge of student affairs at the University of California. Dr. Boyd will talk in the general realm of schools, education and adolescence.

The lecture and seminar will touch heavily on adolescence. Participating in the program will be Dale C. Garell, M.D., head of the adolescent unit at Children's Hospital in Los Angeles, who will talk on **drugs and LSD;** Robert Carter, Ph.D., a former narcotics agent now with School of Criminology at the University of California, who will report on one year's study of **delinquency in suburbia;** Felix Heald, M.D., of Children's Hospital, Washington, D.C., now on a year's sabbatical at the University of California studying the physiology of obesity, who will discuss **obesity in the adolescent;** and John A. Knowles, M.D., assistant medical director of Children's Hospital Medical Center, who will discuss **sex education and teenage pregnancy.**

* * *

A three-day course in the **techniques of rehabilitation care** of patients will be offered to physicians May 25-27, 1967, at Fairmont Hospital, San Leandro.

The course, which is supported by a Traineeship Grant from the U.S. Public Health Service, will present, chiefly in a clinical setting, details of management of adult patients requiring the application of rehabilitation techniques. Applicants for enrollment must be physicians, U.S. citizens and not employed by the federal government.

Further information may be obtained from John D. Blair, M.D., Training Program Director, Fairmont Rehabilitation Training Program, Fairmont Hospital, 15400 Foothill Boulevard, San Leandro, California 94578. Phone: 415-351-8000, Extension 441.

LOS ANGELES

The appointment of **Dr. Findlay E. Russell** as professor of neurology (pharmacology) at the University of Southern California School of Medicine has been announced by Dr. Roger O. Egeberg, dean. An authority on venoms and the treatment of persons who have been bitten or stung by venomous animals, Dr. Russell has directed the Laboratory of Neurological Research, one of the largest and most widely known venom laboratories in the world, since 1955. Operation of this facility, which is located at Los Angeles County General Hospital, was recently transferred to USC from Loma Linda University.

* * *

The Annual Symposium of the Medical Staff of the **Memorial Hospital of Long Beach** will be held at the Memorial Hospital 17 May 1967, at 1 p.m.

The program will feature papers by Memorial Hospital staff physicians. Guest speakers will be Dr. Norman E. Shumway, professor of surgery and head of the Division of Cardiovascular Surgery, Stanford University, and Professor William Pickering, director, Jet Propulsion Laboratory, Pasadena.

SAN DIEGO

Dr. Richard S. Farr of La Jolla was elected treasurer of the American Academy of Allergy at the organization's 23rd Annual Meeting held at the Holiday Inn-Riviera Hotel in Palm Springs.

SAN FRANCISCO

The Eleventh Annual Western Industrial Health Conference will be held at the San Francisco Hilton Hotel, September 29 and 30, 1967. This will be an intradisciplinary meeting, to which professional personnel from fields related to industrial health are invited.

SANTA CLARA

Dr. Donald C. Harrison, assistant professor of medicine, has been appointed head of the division of cardiology at Stanford University School of Medicine. The appointment was announced by Dr. Robert J. Glaser, vice president for medical affairs and dean of the school of medicine. Dr. Harrison succeeds Dr. Herbert N. Hultgren who is on sabbatical leave at Dartmouth Medical School until September, when he will resume his teaching responsibilities at Stanford.

GENERAL

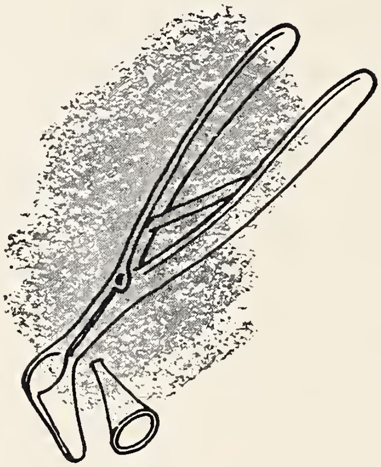
Dr. Forrest M. Willett, former chief of staff of the Veterans Administration Hospital in San Francisco, has been appointed medical director of the **California Division of the American Cancer Society.** The announcement was made by Dr. Lewis W. Guiss of Los Angeles, state president of the Society.

As state medical director of the American Cancer Society, Dr. Willett will coordinate the organization's professional education program, through which new research information is channeled to physicians in private and institutional practice. He will assist county branches of the American Cancer Society, county medical societies and other professional groups in arranging programs and conferences on the subject of cancer.

* * *

The California Research and Medical Education Fund of the Tuberculosis and Health Association of California is inviting **applications for research grants** for 1967-68. Projects may be proposed in clinical, social, epidemiological, laboratory, and related fields of investigation of tuberculosis and other respiratory diseases. This research program also includes investigations into social and psychological factors relating to the prevention and control of these diseases. The Fund has announced that it particularly wishes to encourage young investigators.

Deadlines for applications for 1967-68 are May 1 and November 1, 1967. Further information and application forms may be obtained from: Tuberculosis & Health Association of California, 424 Pendleton Way, Oakland, California 94621.



The Physician's BOOKSHELF

YEAR BOOK OF ANESTHESIA (1966-1967 Year Book Series)—Edited by Stuart C. Cullen, M.D., Professor of Anesthesia and Dean, School of Medicine, University of California Medical Center, San Francisco. Year Book Medical Publishers, Incorporated, 35 East Wacker Drive, Chicago, 1966. 397 pages, $8.50.

The contents are composed of lucid abstracts of articles of interest to anesthesiologists published late in 1965 and early in 1966. Figures, diagrams and tables are reproduced for more adequate presentation of the pertinent data but references are provided only for the original publications. The small size of the volume containing 392 pages makes it easily transportable and readily accessible for reading periods of short duration. For those readers interested in review of a particular subject, the abstracts are grouped under 12 major headings, including circulation, respiration, nonvolatile drugs, inhalation agents and technics, relaxants, regional anesthesia, obstetrics, complications, hyperoxia, clinical problems and inhalation therapy.

The volume is a boon to those students and clinicians who desire to follow what is being written about anesthesiology but who do not have access to many of the original publications or whose reading time is limited.

JOHN W. PENDER, M.D.

* * *

CONSUMERS UNION REPORT ON FAMILY PLANNING—A Guide to Contraceptive Methods and Materials for Use in Child Spacing, Techniques for Improving Fertility, and Recognized Adoption Procedures—2nd Ed.—Prepared by the Editors of Consumer Reports and the following distinguished consultants: Alan F. Guttmacher, M.D., President of the Planned Parenthood Federation, former Director of Obstetrics and Gynecology at Mount Sinai Hospital of New York, and former Clinical Professor of Obstetrics and Gynecology at the College of Physicians and Surgeons of Columbia University; Aquiles J. Sobrero, M.D., Director, Margaret Sanger Research Bureau; and Rael Jean Isaac, teacher, editor, and author of Adopting a Child Today. Consumers Union of U.S., Inc., Mount Vernon, New York, 1966. 191 pages, $1.75 (Paperback).

Many couples would profit from knowledge of the information contained in this inexpensive paperback book. The text is clearly written in nontechnical language which is easily understood by intelligent and inquisitive laymen. It is composed of three sections. Most patients will need to read only one section at a time although a few may have need for the information contained in each section at varying times in their married life.

The first section describes and rates contraceptive methods. The standard mechanical methods, the intrauterine devices, and the various oral contraceptives are discussed in terms of initial costs, yearly costs, effectiveness, side-effects and practicality. The brands and products within these groups are comparatively rated.

The second section describes the common causes of infertility and provides brief descriptions of the tests and treatment programs employed for the diagnosis and therapy both for the male and female infertility.

The third section explains adoption procedures, the legal problems, the costs involved and the criteria by which various adoption agencies select adoptive parents. The advantages and disadvantages of private and agency adoptions are discussed in an unbiased fashion.

In each section there is a list of clinics or agencies which provide these services and referral centers which will direct a patient to the proper source.

The book can be highly recommended to that large segment of the population which expects accurate, factual answers to its questions about medical care. For some, the book may contain more information than really is required but each section is brief, clearly written and well worth the investment in time.

The highest recommendation one can give to this type of book is to order copies for distribution to patients. I placed my order.

EMMET J. LAMB, M.D.

* * *

SCLERODERMA—By Marvin A. Sackner, M.D., Associate Director, Cardiopulmonary Laboratory, Mount Sinai Hospital, Miami Beach, Florida; Assistant Professor of Physiology and Medicine, University of Miami Medical School; American College of Physicians Research Fellow, 1961-1964. (Modern Medical Monographs: Irving S. Wright, M.D., Editor in Chief.) Grune & Stratton, Inc., New York and London, 1966. 174 pages, $13.75.

This is a monograph on the clinical aspects of scleroderma, a multisystem disease of interest to most physicians. The book reviews the many manifestations of the disease, and records the experience gained by the author in studying 65 patients at the Philadelphia General Hospital over a six-year period. Dr. Sackner is to be commended for recognizing the need for a single reference covering all aspects of scleroderma. As in most clinical reviews written by a single author the discussions are uneven. However, the chapters on the cardiopulmonary manifestations, representing the primary interest of the author, are excellent and thorough. The photographs which demonstrate the clinical, histological, and radiological features of the disease are quite useful. The bibliography consists of 516 references.

One criticism of the book is that recent biochemical and immunological advances in scleroderma have been scantily reviewed. The rather extensive literature concerning catecholamine and tryptophane metabolism receives only one paragraph. Immunologic changes, i.e., circulating antinuclear and antitissue antibodies and their possible relationship to pathogenesis, are not mentioned.

In the author's experience, 50 per cent of the patients died 2.2 years after diagnosis. This poor survival is contrary to the experience of most clinicians, and probably reflects the socio-economic group the author was dealing with. The chapter on therapy is quite concise, correct, and well written. The author reiterates that no therapeutic modality has altered the course of scleroderma, and that therapy can only be directed toward symptomatic treatment of individual organ systems.

Those physicians with a particular interest in this disease may find it inadequate, with the exception of the chapters on cardiopulmonary manifestations. The book is recommended for the practicing physician who wishes a single reference containing a review of the clinical manifestations of scleroderma.

DENNY L. TUFFANELLI, M.D.

* * *

FRIEBOES/SCHONFELD COLOR ATLAS OF DERMATOLOGY—By Joseph Kimmig, M.D., Professor of Dermatology, Chief of the Department of Dermatology and the Dispensary, and Michael Jänner, M.D., Research Assistant, Department of Dermatology and Dispensary, University Hospital, Hamburg. American Edition Translated and Revised by Herbert Goldschmidt, M.D., Assistant Professor of Clinical Dermatology, University of Pennsylvania School of Medicine. With a Foreword to the American Edition by Donald M. Pillsbury, M.D., Professor and Former Chairman of the Department of Dermatology, University of Pennsylvania School of Medicine. 304 pages, with 927 figures, mostly in color. W. B. Saunders Company, Philadelphia and London, 1966. $45.00

This is a handsome volume beautifully arranged and printed on pages measuring approximately 8x12". More than 75 per cent of its space is devoted to photographs, all but seven of which are in color. On the average, the reproductions are excellent. With rare exceptions, they serve their purpose of helping one to learn the clinical picture of the disease represented. The diagnosis and a note of two or three lines emphasizing the salient diagnostic features of the condition are printed under each photograph.

There is a table of contents which divides dermatological disorders into 22 groups. Under each group, the individual conditions are listed and the pages in the text on which they are discussed are noted.

The discussions are necessarily brief, but are most valuable. The name, common synonyms, incidence, etiology, character of primary and secondary lesions, distribution, course and sequelae are all mentioned where appropriate. The written test for each group of diseases immediately precedes their photographs. This makes for easy reference in studying a particular dermatosis and related conditions.

The index refers one not only to the text pages but also to the pages on which photographs are presented.

I find this atlas very much worth having. I believe other dermatologists will also wish to have it. It should be valuable in the libraries of medical schools and dermatological clinics and training centers. It would be a useful and popular volume in hospital staff libraries throughout the country.

The cost may discourage some individual physicians who otherwise would like it. However, considering the expense of producing an atlas devoted largely to color reproductions, this is understandable.

H. V. ALLINGTON, M.D.

* * *

MENTAL HEALTH IN A CHANGING COMMUNITY—Based on a Symposium Jointly Sponsored by the San Francisco Association for Mental Health and the Department of Continuing Education in Medicine and The Health Sciences, University of California Medical Center—Edited by Reed Brockbank, M.D., and Dorothy Westby-Gibson, Ed.D. Grune & Stratton, Inc., New York and London, 1966. 163 pages, $6.50.

This book is a real sleeper. While at first glance it looks like just another symposium on mental health put into book form, with all the expected pro forma contributors saying what they are supposed to for public consumption, instead we find a spirited and trenchant look at the changing community *and* the reciprocal changes induced in the studying professions of this changing community. The physical smallness of the book is deceptive for its scope is encyclopedic. Vital issues are addressed in this book and although not systematically dealt with they are raised and aired with clarity; both sides are often forcefully presented with either cogent arguments or weighty clinical evidence. Many of the issues revolve around the need to establish and make explicit normative values of adaptation that are inherent in different phases of life. Is the study and promotion of values the proper domain for the mental health profession? Whose responsibility is mental health? Where does helpfulness end and intrusion begin? Are mental health people supposed to change the whole fabric of our society? In essence, where are the boundaries of the mental health responsibility in this changing community? If we can establish the boundaries of our domain, who does what to whom? And who are the people that compose the mental health profession—are they all people who have contact with other people in a formal role as might be presumed from the inclusion of educators, lawyers and ministers in this forum?

With a striking clinical orientation even in the more theoretical appearing papers, this book approaches these problems in the context of the mental health problems of the people served. The centrality of people, rather than the more abstract "community," keeps the book viable and vibrant. The book is a MUST for physicians who would like to know the problems and the issues of mental health in a changing community, without the comfort of easy answers.

ROGER L. GOULD, M.D.

in many types of otitis externa—of varying etiology—as part of the treatment regimen

comprehensive
Coly▲Mycin® S Otic
with Neomycin and Hydrocortisone

Each ml. provides:

Colistin base activity (as the sulfate) 3.0 mg.
Neomycin base activity (as the sulfate) 3.3 mg.
for complementary broad-spectrum bactericidal action

Hydrocortisone acetate 10.0 mg. (1%)
for anti-inflammatory/antipruritic benefits

Thonzonium bromide 0.5 mg. (0.05%)
for more rapid dispersion and penetration

It also contains polysorbate 80, acetic acid and sodium acetate in a buffered (pH 5) aqueous vehicle and thimerosal 0.002% as a preservative. Buffered to pH 5 to reduce the abnormal alkaline pH which may be favorable to bacterial growth, and to help restore the normal acid pH of the ear canal.[8,9]

combats the bacterial cause
relieves the dermatologic manifestations

Recommended Dosage: Therapeutic—4 drops t.i.d. Prophylactic—4 drops before and after swimming.
Contraindication: A history of sensitivity to any of the components, or in tubercular, fungal and most viral lesions; especially herpes simplex, vaccinia and varicella.
Precautions: If sensitivity or irritation occurs, medication should be discontinued promptly. Overgrowth of resistant organisms is possible. Use with care in cases with perforated eardrum or in long-standing otitis media because of the possibility of ototoxicity caused by neomycin. Increased skin sensitivity to neomycin has been noted in current medical literature.
Adverse Reactions: A low incidence of mild burning or painful sensation in the ear has been reported. Such local effects do not usually require discontinuance of medication. Sensitivity reactions were reported in a few instances.
Full information is available on request.
References: 1. McLaurin, J. W.: J.A.M.A. *154*: 207, 1954. 2. Sulzberger, M. B., *et al.*: Dermatology: Diagnosis and Treatment, ed. 2, Chicago, The Year Book Publishers, Inc., 1961, p. 595. 3. Stewart, J. P.: J. Laryng. & Otol. 65:24, 1951. 4. Elia, J. C.: Southwestern Med. *42*:405, 1961. 5. McLaurin, J. W.; Raggio, T. P., and Simmons, M.: Laryngoscope 75:1699, 1965. 6. Mann, P. H.: Current Therap. Res. 6:101, 1964. 7. Saltzman, M.: Clin. Med. 70:559, 1963. 8. Ettenger, M. S.: Pennsylvania M. J.: 66:35, 1963. 9. Pullen, F. W. II: Eye Ear Nose & Throat Month. *42*: 38, 1963. 10. Jones, E. H., and McLain, P. G.: Eye Ear Nose & Throat Month. *43*:52, 1964.

WARNER-CHILCOTT W|C
CO-GP-72 Morris Plains, N.J.

OFFICERS

OF THE CALIFORNIA MEDICAL ASSOCIATION

(Continued from Page 5)

Committee on Health Facilities Planning

Laurence A. Mosier, Garden Grove	1967
John W. Gates, Visalia	1967
Joseph W. Telford, San Diego	1967
Jean F. Crum, Downey	1968
Glenn A. Pope, Sacramento (Chmn.)	1968
Richard Taw, Los Angeles	1968
James W. Dalton, Santa Barbara	1969
Paul C. Doehring, Glendale	1969
Pierre Salmon, San Mateo	1969

CONSULTANTS: Robert F. Schell, San Rafael; Jack Lomas, Beverly Hills.

COUNCIL COMMITTEES

Finance Committee

Harold Kay, Oakland (Chmn.); Roger C. Isenhour, San Diego; Frank C. Melone, Ontario; Joseph W. Telford, San Diego; Malcolm C. Todd, Long Beach.

Committee on Legislation

Dan O. Kilroy, Sacramento (Chmn.); Stuart C. Knox, Los Angeles; Harold E. Wilkins, Downey; John Rumsey, San Diego; Samuel R. Sherman, San Francisco.

Advisory Board to Woman's Auxiliary

James C. MacLaggan, San Diego (Chmn.); John G. Morrison, San Leandro; William F. Quinn, Los Angeles; Carl E. Anderson, Santa Rosa; Eugene Hoffman, Los Angeles.

Benevolence Fund Operating Committee

Clyde L. Boice, Palo Alto (Chmn.); Alexander Fraser, San Francisco; Elizabeth Mason Hohl, Los Angeles; Don C. Musser, San Francisco; George Wolf, Fresno; Dudley M. Cobb, Jr., Los Angeles.

Economical Long-term Therapy for Iron Deficiency Anemia
Tablets & Elixir

FAMOUS
Fergon®
brand of FERROUS GLUCONATE
C.M.A.P. CODE # 5261

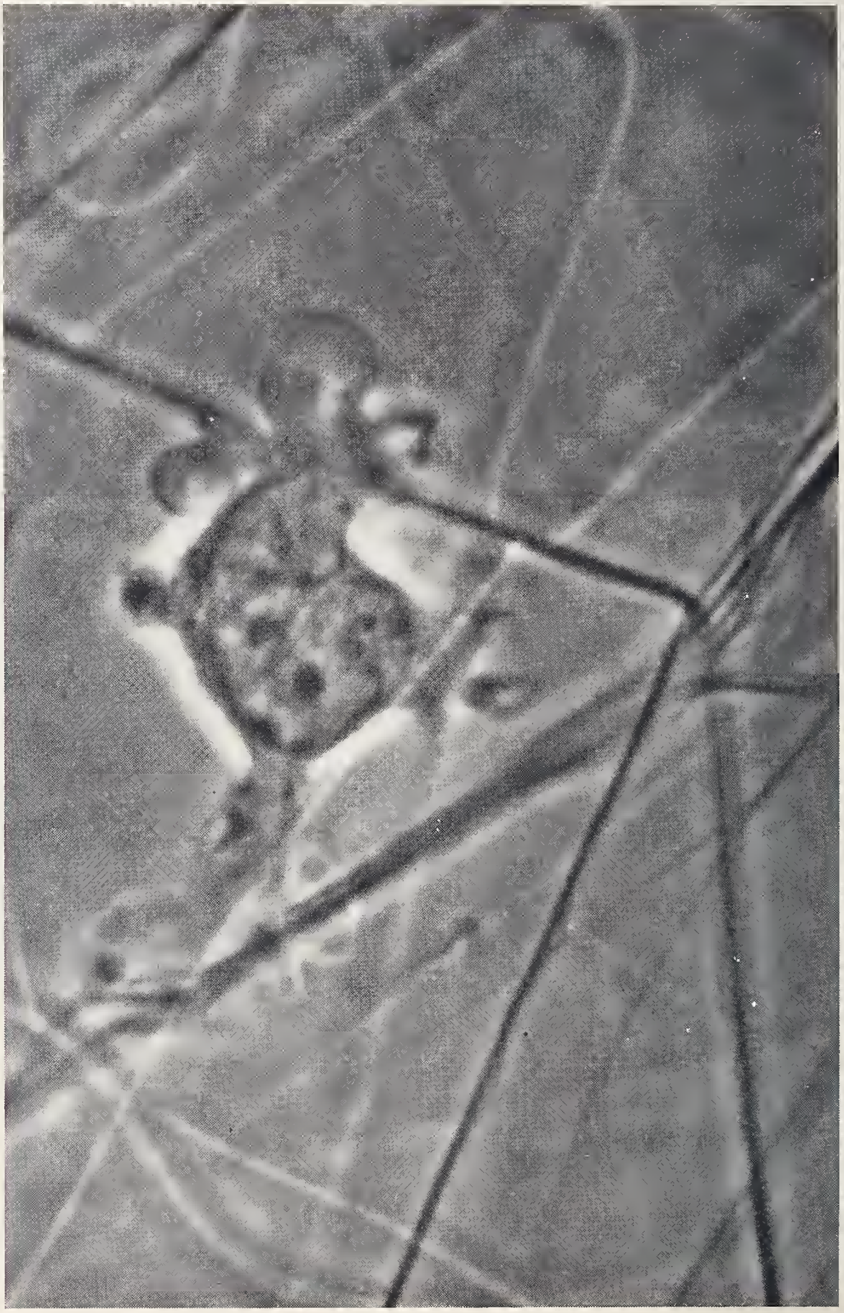
BREON
BREON LABORATORIES INC.
Subsidiary of Sterling Drug Inc.
90 Park Avenue, New York, N.Y. 10016

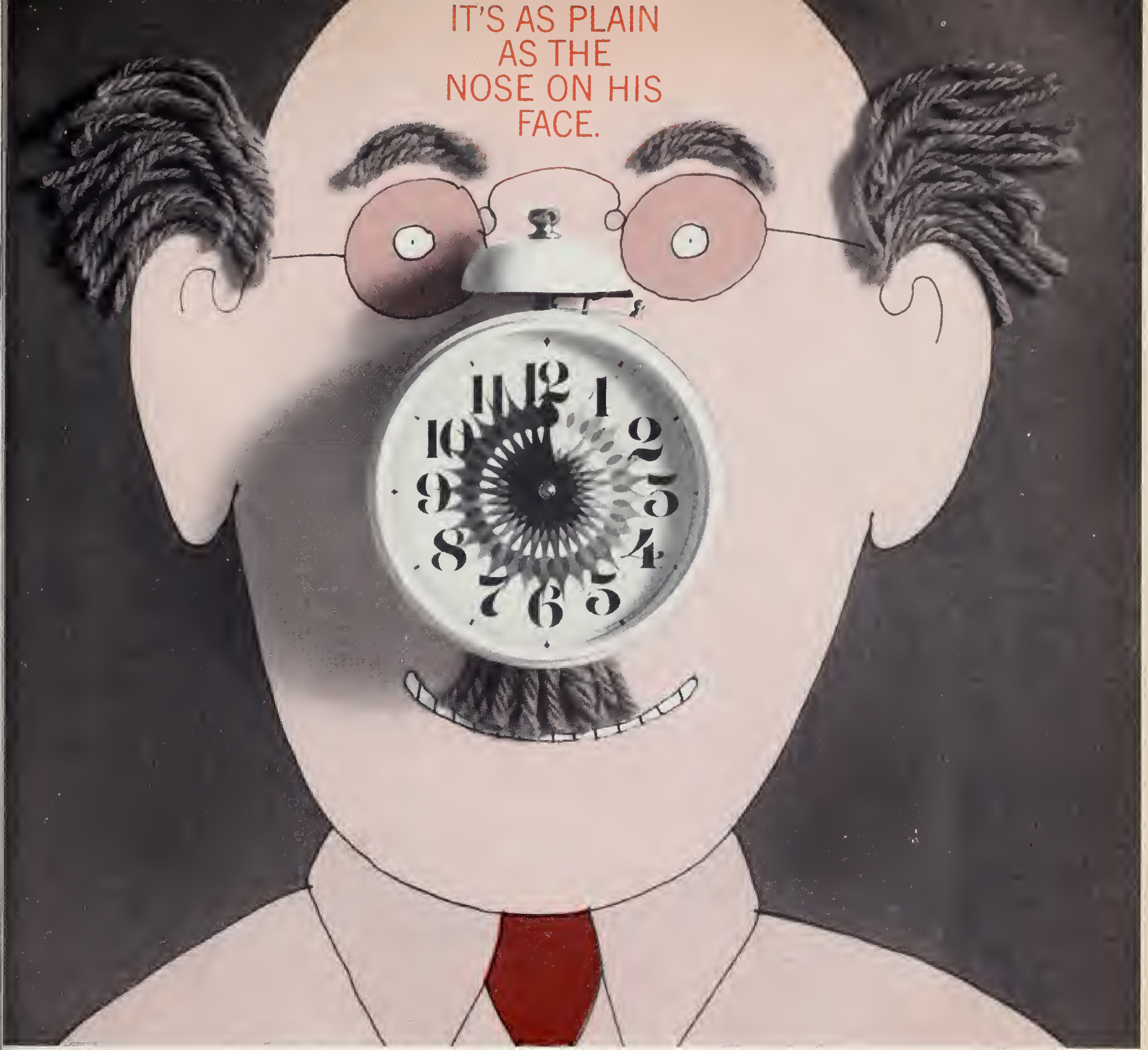
INFLAMMATION:
A cellular fight for life

A SYNTEX REPORT based on recently developed hypotheses about topical corticosteroids, including the cellular theories of inflammation by Thomas F. Dougherty, Ph.D., University of Utah.

You are looking at a fibroblast fighting for life. This cell—one of the most common found in connective tissue—has literally been poisoned by cytotoxins released from other cells that have ruptured. Soon, if the abnormal activity of this fibroblast does not cease, it, too, will rupture and die—one more casualty in the inflammatory wave of destruction precipitated by injury.

Until a short time ago no one had ever witnessed such a scene at the cellular level. Now, through advanced cinemicrographic techniques, it is possible to view and photograph the inflammatory process as produced experimentally in living animal tissue. This method permits new insight into the mechanism of inflammation and the role of corticosteroids in therapeutic management. Equally important, these techniques shed new light on factors that may make one corticosteroid more effective than another—factors that can be correlated with other chemical, biologic, and clinical parameters.





UP TO 10–12 HOURS' CLEAR BREATHING ON ONE TABLET

Dimetapp® Extentabs®

(Dimetane® [brompheniramine maleate], 12 mg.; phenylephrine HCl, 15 mg.; phenylpropanolamine HCl, 15 mg.)

In sinusitis, colds, or U.R.I., Dimetapp lets congested patients breathe easy again. Each Extentab brings welcome relief all day or all night, usually without drowsiness or overstimulation. Its key to success? The Dimetapp formula—Dimetane (brompheniramine maleate), a potent antihistamine reported in one study to have elicited side effects as few as the placebo,* teamed with decongestants phenylephrine and phenylpropanolamine—in a dependable 10- to 12-hour form.

Contraindications: Patients hypersensitive to antihistamines. Not recommended for use during pregnancy.

Precautions: Until the patient's response has been determined, he should be cautioned against engaging in operations requiring alertness. Administer with care to patients with cardiac or peripheral vascular diseases or hypertension.

Side Effects: Hypersensitivity reactions including skin rashes, urticaria, hypotension and thrombocytopenia have been reported on rare occasions. Drowsiness, lassitude, nausea, giddiness, dryness of the mouth, mydriasis, increased irritability, or excitement may be encountered.

Dosage: 1 Extentab morning and evening, or as needed.

Supplied: Bottles of 100 and 500.

Also available: Dimetapp® Elixir for conventional *t.i.d.* or *q.i.d.* dosage. See package insert for further details.

A. H. ROBINS CO., INC.
RICHMOND, VIRGINIA 23220

A·H·ROBINS

*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

mudrane®

for

- EMPHYSEMA
- ASTHMA
- CHRONIC BRONCHITIS
- BRONCHIECTASIS

The fast-disintegrating uncoated tablet gives relief in 15 minutes

Each tablet contains:

Potassium Iodide	195 mg.
Aminophylline	130 mg.
Phenobarbital, Caution: May be habit forming	21 mg.
Ephedrine HCl	16 mg.

FEDERAL LAW PROHIBITS DISPENSING WITHOUT PRESCRIPTION

Precautions: Usual for aminophylline-ephedrine-phenobarbital. Iodides may cause nausea, long use may cause goiter. Discontinue if symptoms of iodism develop.

Iodide contraindications: tuberculosis, pregnancy.

DOSAGE

One tablet, with full glass of water, 3 or 4 times daily.

Dispensed in bottles of 100 and 1000 tablets.

MUDRANE GG—Formula, dosage and package identical to Mudrane—*except*—100 mg. glyceryl guaiacolate replaces the potassium iodide. The value of Mudrane cannot be enjoyed by a small group in which K.I. is contraindicated. Mudrane GG is prepared for this group.

MUDRANE GG ELIXIR—Four 5 cc teaspoonfuls is equivalent to one Mudrane GG tablet. Dosage adjusted to age and weight of child. Mudrane GG Elixir is for pediatric patients and those who think they cannot swallow tablets. Dispensed in pint and half gallon bottles.

WM. P. POYTHRESS & CO., INC.
RICHMOND, VIRGINIA 23217
Manufacturers of ethical pharmaceuticals since 1856

New Snakebite Treatment

(Continued from Page 42)

Knowles, D.V.M., instructor in animal surgery at the University's department of surgery; Maxwell M. Sayet, M.D., clinical associate professor of pathology; James Pickens, a senior medical student at Miami, and James Emerson, sophomore dental student at Ohio State University. William Haast, herpetologist and director of the Miami Serpentarium, also is assisting in the work.

Myocardial Infarction, Smoking, and Activity

Among men, it's the smoker who is relatively inactive who is most likely to suffer a myocardial infarction and later, a fatal attack.

The amount of exercise men get seems to be the major factor in determining whether they survive the first heart attack. The more active they are, the better their chances.

The least active nonsmoker is nearly twice as likely to experience a rapidly fatal myocardial infarction as the more active smoker.

Inactive men who smoke cigarettes show a mortality rate nine times that of more active nonsmokers of similar age.

These findings are reported by the Health Insurance Plan of Greater New York in the December 19 *Journal of the American Medical Association.*

During an 18-month study of 110,000 men and women, 301 men suffered a first myocardial infarction, the report said.

"Fifty per cent of the least active men who smoked cigarettes were dead within four weeks of (the attack), compared with 28 per cent of the smokers with higher levels of physical activity," the report said.

For nonsmokers, the corresponding figures were 56 per cent for the least active men and 17 per cent for those who were relatively more active.

Speculating on possible causes of these differences, the authors said smoking may "accelerate the rate of development of coronary atherosclerosis and thereby increases the likelihood of (an attack)."

Smoking may be one of several "trigger" mechanisms which bring on acute myocardial deterioration in men with significant coronary atherosclerosis.

The rates for first infarction are: 7.39 attacks per 1,000 men for inactive nonsmokers; for active smokers, 5.36 per 1,000; least active nonsmokers, 5.24, and active nonsmokers, 2.52.

The authors are Charles W. Frank, M.D., of Albert Einstein College of Medicine, New York City, and Eve Weinblatt, Sam Shapiro, and Roger V. Sagar, M.D., of the Health Insurance Plan of Greater New York.

Soyalac

SOLVES THE PROBLEM

for the infant who requires a milk-free diet!

Loma Linda
Soyalac
CONCENTRATED
LIQUID
Hypoallergenic
For Infants
Children and Adults
Loma Linda
FOODS

- Soyalac satisfies! Baby is happy, mother is grateful, doctor is gratified.
- The nut-like taste is pleasing. Infants readily accept this hypoallergenic formula that is completely fibre-free. An exclusive process results in a consistency much like milk.
- Soyalac is strikingly similar to mother's milk in composition and ease of assimilation. Clinical data furnish evidence of Soyalac's value in promoting normal growth and development.
- Excellent for regular infant feeding, too—and for growing children and adults.

Soyalac

a product of

LOMA LINDA FOODS
MEDICAL PRODUCTS DIVISION
RIVERSIDE, CALIFORNIA
Mount Vernon, Ohio, U. S. A.

Free Booklet and Samples

A request on your professional letterhead or prescription form will bring to you complete information and a supply of samples.

A boy, a man,
a shared interest...

. . . vital elements in the Devereux therapeutic camping programs.

Opportunities for the development of meaningful individual relationships with mature, understanding staff members provide a fundamental treatment basis.

Residential camps in Pennsylvania, California, Texas, Massachusetts and Maine for emotionally disturbed and mentally retarded children and young adults.

DEVEREUX SCHOOLS
UNDER THE DEVEREUX FOUNDATION
A NON PROFIT ORGANIZATION

Helena T. Devereux *Edward L. French, Ph.D.*
Founder and Consultant *President and Director*

FOR INFORMATION AND LITERATURE
Santa Barbara (Box 1079), California
Keith A. Seaton, Director of Admissions

Devon, Pennsylvania and Rutland, Massachusetts
Charles J. Fowler, Director of Admissions
Devereux Schools, Devon, Penna. 19333

Victoria (Box 2666), Texas
Richard D. Grant, Registrar

Symposia on Iron Storage, Colitis, Among Scientific Programs at AMA Annual Convention

Symposia of interest to both the generalist and the specialist will be included in this year's Scientific Program of the American Medical Association's Annual Convention.

The Convention will be held in Atlantic City June 18 to 22; the Scientific Program Convention Hall and surrounding hotels and the House of Delegates at the Chalfonte-Haddon Hall Hotel.

A Symposium on Absorption and Storage of Iron will be presented as a joint meeting of the Sections on Pathology and Physiology, Internal Medicine, Experimental Medicine and Therapeutics, and Gastroenterology.

The Sections on Radiology, Proctology, Pediatrics, General Surgery, Internal Medicine, and Gastroenterology will join for a Symposium on Granulomatous Colitis and Ulcerative Colitis in Children.

The entire Scientific Program for the 1967 Annual Convention will be published in the May 8 issue of the *Journal of the American Medical Association.*

Patient Care One of Four Topics of General Sessions at 1967 AMA Annual Convention

Patient care, from the standpoint of standard methods as well as research, will be one of four topics presented in general scientific sessions at this year's Annual Convention of the American Medical Association.

The Convention is to be held in Atlantic City June 18 to 22; the Scientific Program will be at Convention Hall, and nearby hotels, and the House of Delegates will meet at the Chalfonte-Haddon Hall Hotel.

The General Scientific Meetings are open to all physicians attending the Annual Convention.

Other General Scientific Meetings on this year's Annual Convention program will be on the subjects of: backache, healing and sex.

In addition to the General Sessions, each of the 22 Scientific Sections will present scientific programs. Many of the Section programs will, as in past years, be joint meetings of two or more Sections and, in some instances, a specialty society.

Specialty societies joining AMA Sections will include:

—The American College of Chest Physicians, which will join the Section on Diseases of the Chest for a program.

—The American College of Cardiology, which will join the Section on Internal Medicine in a session.

—The Society for Investigative Dermatology, Inc., which will hold its meetings in conjunction with the Section on Dermatology.

Cold and The Common Cold

Does cold have an effect on the common cold?

A new study suggests that it doesn't, at least under normally encountered conditions.

The Medical News Section of the February 13 *Journal of the American Medical Association* reports the findings of three National Institutes of Health investigators who subjected 43 volunteers to a variety of cold-catching conditions.

Some persons spent 1½ to 2½ hours in a room whose temperature hovered around 40 degrees fahrenheit—the sort of short-term chilling commonly believed to bring on a cold.

Others were placed in cool baths which lowered their body temperature approximately two degrees. This was designed to duplicate the lowered body temperatures associated with some viral and bacterial infections.

Despite their exposure to cold temperatures, these volunteers didn't catch significantly more colds than a control group who stayed dry and warm.

When some persons were inoculated with cold-causing rhino-viruses, the illness rate was slightly higher among cold-exposed volunteers. But more controls than cold-exposed persons had fever.

Effects of cold temperature during illness also was studied. Three persons were immersed in cold baths for two to four hours at the height of their cold. The course of their illnesses didn't seem to differ from that of other persons under study.

A final study was of exposure to cold during recovery; that is, while viruses continued to be present in nasal areas, but the persons appeared to be well. For this, 13 volunteers went into the cold room or cold bath.

One cold-exposed man had a one-day relapse in his recovery from a cold. Further studies showed an increase in viruses shed by one cold-exposed volunteer, but also an increase in those shed by one of the controls.

The investigators are R. G. Douglas, Jr., M.D., and R. B. Couch, M.D., now both of Baylor University College of Medicine, and K. M. Lindgren, M.D., who continues his work at NIH.

Because cold temperature will lower resistance to some experimental infections in animals, "it remains possible that more prolonged and more severe cold exposures may result in such an effect on humans," Dr. Douglas said.

These experiments however, were designed to approximate cold-exposure found in natural circumstances.

"It seems unlikely," he said, "that more severe or prolonged (cold temperature) conditions would be encountered frequently."

Togetherness....

...can be rough when epidemics of nausea and vomiting strike a family. Emetrol offers prompt, safe relief. It is free from toxicity[1] or side effects[2,3] and will not mask symptoms of serious organic disorders.

1. Bradley, J. E., *et al.*: J. Pediat. *38*:41 (Jan.) 1951.
2. Bradley, J. E.: Mod. Med. *20*:71 (Oct. 15) 1952.
3. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. *65*:311 (Feb.) 1953.

RORER

WILLIAM H. RORER, INC.
Fort Washington, Pa.

Emetrol®
phosphorated carbohydrate solution
emesis control

The Mediatric Age:

There is a growing senescent body of people on their way to malignant inactivity, who sorely need your interest and direction to help them back to a more active and useful life. There are medicines too, designed to help. One such has proved useful in clinical practice.

"A steroid-nutritional compound (Mediatric) was used in 100 patients to relieve some of the symptoms caused by degenerative changes of aging . . . This therapy resulted in improvement of 75 per cent of the patients . . ."

McNeill, A. J.: Clin. Med. *8*:518 (Mar.) 1961.

"Mediatric (steroid-nutritional compound) capsules, one a day, seem to give definite help to debilitated patients."

Arnold, E. T., Jr.: Geriatrics *12*:612 (Oct.) 1957.

"Nutritional and hormone bolstering of function in the aged may have a useful place in geriatrics."

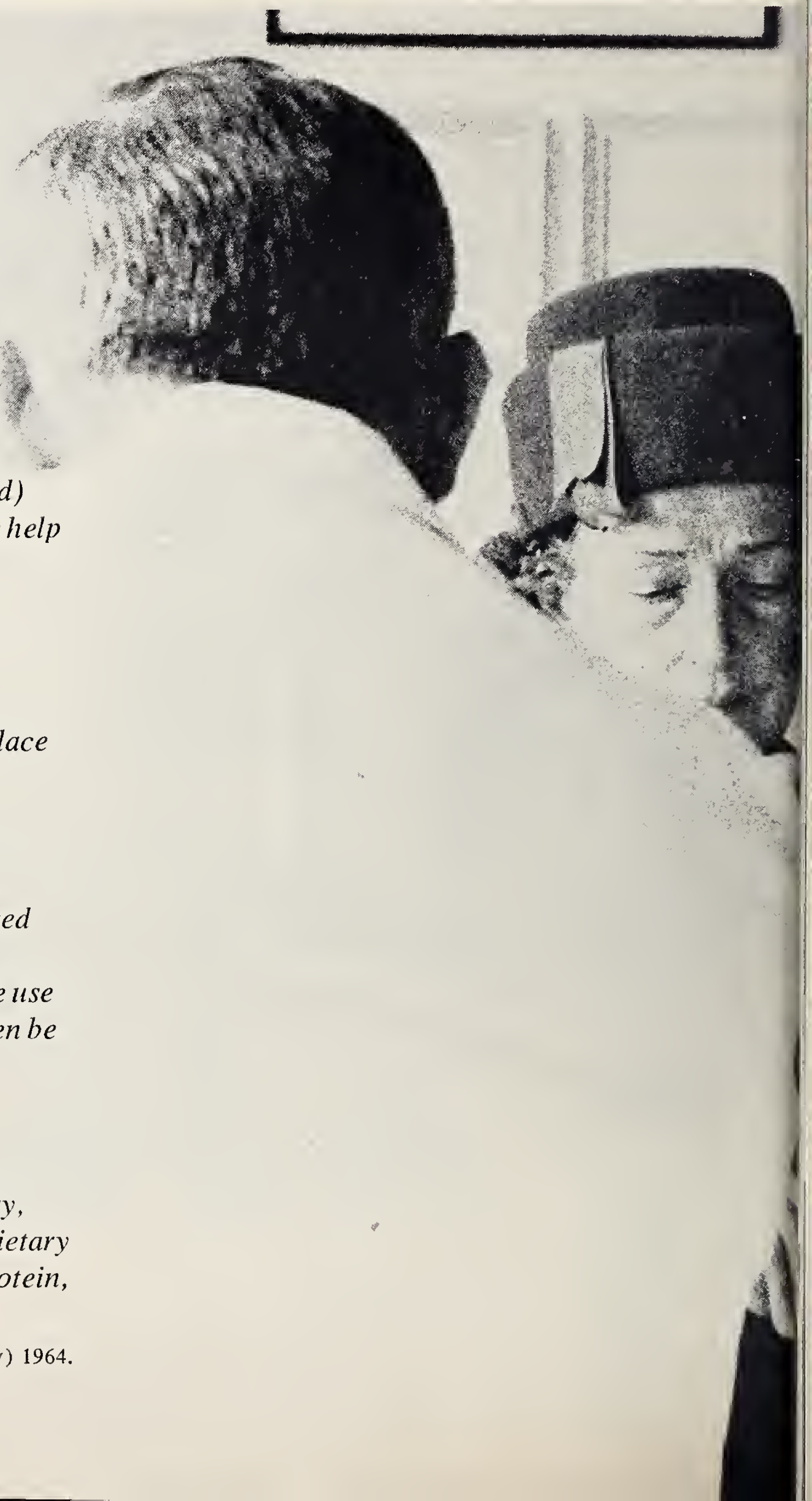
Morgan, A. F.: Gerontologist 2:77 (June) 1962.

"In diets which for any reason are restricted in calories, enough of these substances (B vitamins) may not be supplied . . . The use of B and C vitamin supplements may then be justified and indeed may be necessary."

Morgan, A. F.: Gerontologist *2*:77 (June) 1962.

"Intensive nutritional therapy is necessary, especially in elderly people, to correct dietary deficiencies created by large losses of protein, vitamins and other nutrients."

Riccitelli, M. L.: J. Am. Geriatrics Soc. *12*:489 (May) 1964.



BOOKS RECEIVED

Books received by CALIFORNIA MEDICINE *are acknowledged in this column. Selections will be made for more extensive review in the interest of readers as space permits.*

ALCOHOLISM—Mechanism and Management—By Max Hayman, M.D., Associate Clinical Professor of Psychiatry, Alcoholism Research Clinic, University of California at Los Angeles Center for the Health Sciences. Charles C Thomas, Publisher, 301 East Lawrence Ave., Springfield, Illinois, 1966. 315 pages, $10.50.

ATLAS OF VASCULAR SURGERY—Second Edition—By Falls B. Hershey, M.D., F.A.C.S., Associate Professor of Clinical Surgery, Washington University School of Medicine, St. Louis; and Carl H. Calman, M.D., F.A.C.S., Formerly Assistant in Clinical Surgery, Washington University School of Medicine, St. Louis. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis, Missouri 63103, 1967. 318 pages, $19.50.

CLINICAL PATHOLOGY—Interpretation and Application—Fourth Edition—By Benjamin B. Wells, M.D., Ph.D., Assistant Chief Medical Director for Research and Education in Medicine, Veterans Administration, Washington, D.C.; and James A. Halsted, M.D., Associate Chief of Staff for Research and Education, Veterans Administration; Clinical Professor of International Health and Nutrition, The George Washington University School of Medicine, Washington, D.C. W. B. Saunders Company, Philadelphia, West Washington Square, Philadelphia, 19105, 1967. 708 pages, illustrated, $11.50.

CNS DRUGS—A Symposium held at the Regional Research Laboratory, Hyderabad, India, January 24-30, 1966—Council of Scientific and Industrial Research, New Delhi, 1966. 367 pages, $10.00.

CONGESTIVE HEART FAILURE—By Ralph M. Myerson, B.S., M.D., F.A.C.P., Asst. Chief, Medical Service, VA Hospital, Philadelphia; Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; and Bernard H. Pastor (deceased), A.B., M.D., F.A.C.P., Formerly Chief, Cardiovascular Section, VA Hospital, Philadelphia; formerly Associate Professor of Medicine, University of Pennsylvania School of Medicine. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis, Mo. 63103, 1967. 174 pages, illustrated, $12.85.

DOCTOR'S HOUSE CALL—By James Rogers Fox, M.D., Collier Books, New York, Collier-MacMillan Limited, 866 Third Avenue, New York, N. Y. 10022, 1967. 406 pages, $1.95 (Paperback).

THE EXISTENTIALIST APPROACH TO PSYCHOTHERAPY WITH ADULTS AND CHILDREN—By Hanna Colm, Ph.D. With introduction by Edith Weigert, M.D. Edited by Claire M. Bloomberg. Grune & Stratton, 381 Park Ave. South, New York, N. Y. 10016, 1967. 229 pages, $6.50.

THE EXTREMITIES—Third Edition—By Daniel P. Quiring, Ph.D., Late Head of the Anatomy Division, Cleveland Clinic Foundation and Associate Professor of Biology, Western Reserve University; the Third Edition is Revised and Edited by John H. Warfel, Ph.D., Assistant Professor of Anatomy, State University of New York at Buffalo, School of Medicine. Lea & Febiger, 600 S. Washington Square, Philadelphia, Pa. 19106, 1967. 124 pages, 110 illustrations, $3.90.

THE HEAD, NECK AND TRUNK—Muscles and Motor Points—Third Edition—By Daniel P. Quiring, Ph.D., Late Head of the Anatomy Division, Cleveland Clinic Foundation and Associate Professor of Biology, Western Reserve University. Third Edition, Revised and Edited by John H. Warfel, Ph.D., Assistant Professor of Anatomy, State University of New York at Buffalo. Lea & Febiger, 600 S. Washington Square, Philadelphia, Pa. 19106, 1967. 1[illegible] pages, 111 illustrations, $3.90.

HEARTS—Their Long Follow-Up—By Paul Dudley White, M.D., and Helen Donovan. W. B. Saunders Company, West Washington Square, Philadelphia 19105, 1967. 357 Pages, illustrated. $12.00.

THE HUMAN HEART—The Layman's Guide to Heart Disease—By Brendan Phibbs, M.D., F.A.C.P., F.A.C.C., Cardiovascular Research Unit, Natrona County Memorial Hospital, Casper, Wyoming; with contributions from Lane Craddock, M.D., Denver, Colo., George Griffith, M.D., F.A.C.P., F.A.C.C., Los Angeles, Robert T. Patrick, M.D., F.A.C.Anes., Casper, Wyo., and Colin H. M. Walker, M.D., F.R.C.P. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis, Mo. 63103, 1967. 253 pages, 72 illustrations, $4.95.

A MANUAL OF CLINICAL ALLERGY—Second Edition—By John M. Sheldon, M.D., Professor of Internal Medicine, University of Michigan Medical School; Robert G. Lovell, M.D., Associate Professor of Internal Medicine, The University of Michigan Medical School; and Kenneth P. Mathews, M.D., Professor of Internal Medicine, University of Michigan Medical School. W. B. Saunders Company, 600 West Washington Square, Philadelphia, Pa. 19105, 1967. 550 pages, $15.00.

YOU ARE AS YOUNG AS YOUR SPINE—By Editha Hearn, Cyriax Physiotherapist, M.C.S.P., M.E., LET., London. Illustrations by Charlotta Adlerova. Doubleday & Company, Inc., Garden City, New York, 1967, 120 pages, $4.50.

Finer ALLERGENIC *Extracts*

- *specialized allergy service*
- *diagnostic and therapeutic extracts*

PROMPT • PERSONAL SERVICE

Frees you from details and delays.
Provides you with ready-to-use extracts.

Descriptive literature sent on request.

BERKELEY BIOLOGICALS
Second & Hearst Sts., Berkeley, Calif. 94710
2275 W. Lincoln Ave., Anaheim, Calif. 92801

Your public relations problem has been our prime consideration in collection procedures during two generations of ethical service to the Medical Profession.

THE DOCTORS BUSINESS BUREAU
Since 1916
821 MARKET ST., SAN FRANCISCO 94103
GArfield 1-0460

STACEY'S for any Medical Book in Print
Retail Stores in Palo Alto and San Francisco

NEW BOOK

LAW FOR THE PHYSICIAN. By Carl Erwin Wasmuth, M.D., Staff, The Cleveland Clinic Foundation, Associate Professor of Law, Cleveland-Marshall Law School of Baldwin Wallace College, Cleveland, Ohio. 583 pages. 1966. Lea & Febiger. $16.50.

Stacey's
326-9955
2575 HANOVER STREET
PALO ALTO, CALIFORNIA 94304

Please send me on approval:________
Name________
Street________
City________ State________

California
Medicine

CLASSIFIED ADVERTISEMENTS

Rates for each insertion are $10 for fifty words or less; additional words 10 cents each; Box number charge: 50c

Copy for classified advertisements should be received not later than *the fifth of the month preceding issue.* • Classified advertisers using Box Numbers forbid the disclosure of their identity. Your inquiries in writing will be forwarded to Box Number advertisers. The right is reserved to reject or modify all classified advertising copy in conformity with the rules of the Advertising Committee.

CLASSIFIED ADVERTISEMENTS ARE PAYABLE IN ADVANCE

PHYSICIANS WANTED

IMMEDIATE STAFF OPENINGS in psychiatry and general medicine in State hospitals and correctional facilities. Regular hours, attractive salaries and benefits. Qualifying interviews in Los Angeles and San Francisco, twice monthly. Write: William Webster, State Personnel Board, Sacramento, California 95814.

STAFF PHYSICIAN for 98-bed Country Hospital. Salaries $15,000 to $18,600 per year according to qualifications. California license required. Permanency for those preferring institutional practice, including excellent employment and retirement benefits. Write to Personnel Office, County Government Center, Madera, California 93637; or telephone collect (209) 674-4641.

PHYSICIANS-SURGEONS: Excellent appointments all areas of California and other western states. Administrative, industrial, general and specialties. We know our areas and our clients. Established in California since 1944. Continental-Pacific Coast Medical Bureau, Agency, 9777 Wilshire, Beverly Hills, California 90212.

SAN FRANCISCO: Elegant opportunity for internist in multi-phasic office. Salary to start, partnership after one year. Contact: Miss Schill, 450 Sutter Street, Suite #1137, San Francisco, California; or phone: (415) 781-3151 (Monday through Friday, between 9 and 6, only).

GENERAL PRACTICE GROUP/WOODLAND, CALIFORNIA—Seeking new partner to replace retiring fourth member. Salary $1,200-$1,600/month for 18 months followed by partnership. Equal sharing of on-call schedule. Prefer individual with two years postgraduate training. Further information on request. N. B. Smith, M.D., 500 First Street, Woodland, California 95695.

SOUTHERN MONTEREY COUNTY MEDICAL GROUP—INTERNIST, (1) With pediatric background preferred; Board eligible or Board qualified, 30-40.

GENERAL PRACTITIONERS, (2), ONE must have excellent knowledge of conversational Spanish, age 30-40.

Seven-man, family-oriented medical group. Accredited 46-bed general hospital and 25-bed nursing home nearby.

First-year salary, $18,000, plus H&A, life and business car insurance and other fringe benefits. Share in profits after first year. Excellent IRS approved pension plan after two years, and membership after three. No immediate investment. Located in hunting, fishing, boating and scenic recreational areas. Excellent school systems

For further details concerning the functioning of this group, please communicate with Len Hughes Andrus, M.D., F.A.C.P., Medical Director, enclosing resume. Address: 210 Canal Street, King City, California 93930.

GENERAL PRACTITIONER—For small community of 500, located in the high Sierras. Permanent residents about 1,000 in the area. Good winter and summer recreational area with skiing, hunting and fishing. Peak recreational population exceeds 25,000 per day. The nearest hospital is 40 miles. Contact: Mrs. D. O. Manes, Committee Chairman, June Lake Loop Chamber of Commerce, Inc., P.O. Box 2, June Lake, California.

ORTHOPAEDIC SURGEON: Growing San Gabriel Valley orthopaedic six-man group. Certified or Board eligible. Salary first two years. Partnership thereafter. H. H. Tyson, Business Manager, 632 W. Duarte Road, Arcadia, California 91007; (213) HI 6-4666.

POSITION NOW OPEN: General practitioner, preferably with GP residency to join a group of three other physicians and a Board certified surgeon in hospital-based group. Ample clinic facilities and material. Would take turn on call nights and week-ends. Prefer stable individual with family. Residence possibly available. Salary approximately $22,000 depending upon qualifications and experience. Contact: Chief County Physician, Humboldt Medical Center, 2200 Harrison Avenue, Eureka, California 95501, or telephone collect: (707) 443-4511.

GENERAL PRACTITIONER—LONE PINE, CALIFORNIA: To join surgeon and internist in large practice. Good 30-bed accredited hospital; good schools and churches; all outdoor recreational facilities. Contact: Donald L. Christenson, M.D. and Milton R. Jones, M.D., Box 38, Lone Pine, California 93545.

IMMEDIATE OPENING—For well-trained general practitioner with completed military obligations. Busy Southern California practice. Liberal financial arrangements first year, with partnership following. Send full resume to: Box No. 3020, California Medicine.

MULTISPECIALTY GROUP—DUE TO PLANNED EXPANSION immediate openings for staff physicians and some department heads in almost all specialties and general medicine; group comprises 60 physicians with planned expansion to 100, with 150-bed hospital expanding to 400 beds; partnership available in three years; excellent fringe benefits such as sick leave, vacation, educational allowance, disability and retirement programs. For further details, write or call John M. Mott, M.D., The Permanente Medical Group, 3240 Arden Way, Sacramento, California 95825; telephone (916) 482-8100.

GENERAL PRACTITIONER—Full time, small, growing clinic. Surgical and obstetrical consultants. Starting salary, $20,000; partnership to follow. Seattle suburb. Unlimited choice of salt-water recreation and mountain skiing. Position available the first of April 1967. Send inquiries to P.O. Box 3066, International Station, Seattle, Washington 98114.

SACRAMENTO—Several attractive medical practice openings are immediately available in the inpatient, outpatient and emergency departments; both short-term and yearly appointments available. Sacramento County Hospital is a 580-bed teaching institution affiliated with the University of California Medical School at Davis. Salary: $17,280 to $19,829 per year, based on education and experience; regular hours, night and weekend coverage on liberal rotation. Contact Director of Medical Services, Sacramento County Hospital, 2315 Stockton Boulevard, Sacramento, California 95817. Telephone (916) 454-2279.

GENERAL PRACTITIONER: Immediate opportunity, 52-bed county hospital. Starting salary, $1,500/month with range to $2,000. Air-conditioned home with utilities and maintenance provided; excellent fringes. Abundance of recreation. Small-city friendship and relaxation; no fog or smog. Write: Personnel Director, County of Imperial, Courthouse, El Centro, California 92243.

YOUNG SURGEON or GP interested in working permanently with active, young, surgically-oriented ENT man in San Francisco Bay area to manage general care of in- and out-patients, do minor ENT, organize and assist surgery. Start $1,000 monthly; yearly profit share. No special training required. Applicant agrees to work at least three years. Box No. 3055, California Medicine.

UPPER MIDWEST COLLEGE CITY—Expanding multi-specialty group seeks addition of internist, otolaryngologist, general surgeon, radiologist, urologist, and pediatrician. Unlimited income. Contact: John J. Eustermann, M.D., c/o Mankato Clinic, Ltd., 501 Holly Lane, Mankato, Minnesota 56001; (507) 387-1811.

LOS ANGELES AND ORANGE COUNTY: GP, $24,000 per year; GENERAL SURGEON, $30,000 per year. Other specialties, $30,000 per year. Opportunity for partnership. Only California residents with California license, or those available now, should apply. Call: D. Kelly, M.D., (213) 432-5911; 125 East 8th Street, Long Beach, California 90813.

EMERGENCY ROOM PHYSICIANS—Full-time, needed to staff active emergency department, JCAH-approved, 165-bed hospital in Torrance, California. Immediate openings; annual salary, $20,000 minimum. California State license required. Contact Administrator, Little Company of Mary Hospital, 4101 Torrance Boulevard, Torrance, California 90503.

ANESTHESIOLOGISTS; PATHOLOGIST; RADIOLOGIST; INTERNIST; PEDIATRICIAN: ANESTHESIOLOGISTS—Chief and staff, to $36,000; PATHOLOGIST—Chief, Bay Area, to $40,000+; RADIOLOGIST—Central California, $25,000; INTERNIST—Bay area, $20,000; PEDIATRICIAN—San Francisco area, $18,000. Apply: Conrad K. Howan, M.S.H.A., Director, Bay Medical Associates Agencies, 821 Market Street, Suite 518, San Francisco 94103; telephone: (415) YU 2-6191.

SEMI-RETIRED GENERAL PRACTITIONER wanted (in age group 50-60 years) to share a one-man established medical office 50 per cent of the time. Office completely equipped; staffed by registered nurse. Computerized billing system. E. A. Hahn, M.D., 1820 Professional Drive, Sacramento. California 95825.

(Continued on Page 79)

PHYSICIAN PLACEMENT SERVICE

The California Medical Association offers free placement assistance through the Physician Placement Service, 693 Sutter Street, San Francisco, California 94102. This service is for the use of all physicians seeking practice opportunities in California and for CMA members who are seeking an assistant or associate. A bulletin is published every other month. Opportunities in the Bulletin must be sponsored by a CMA member, an accredited California hospital or responsible persons in a California community. The Bulletin does not carry listings describing real estate for sale or office space for rent or lease or blind listings with box numbers.

CLASSIFIED ADVERTISEMENTS

(Continued from Page 78)

ASSOCIATES WANTED

YOUNG GENERAL PRACTITIONER—AAGP to associate with two active M.D.'s (one FACS), strictly general practice, in desirable coastal town with population area of 25,000; excellent opportunity. Hospital soon to be built, open staff policy. Well-equipped offices, pro-rated expenses. Box No. 845, California Medicine.

GENERAL PRACTITIONER for association with busy GP, age 55, in town of 5,000 in central California. Details of association open. Fully accredited, open-staff hospital with pathologist and radiologist. Reply: Box No. 2075, California Medicine.

ASSOCIATE WANTED—For medical chest practice. Internist with medical chest and pulmonary physiology training. Board qualified or certified. Practice limited to chest medicine, hospital consulting and research. Write: Suite 229—550 Washington Street, San Diego, California 92103.

WHITTIER, CALIFORNIA: GENERAL PRACTITIONER (A.A.G.P.) 42 years old desires male associate 25-45. Opening available July 1, 1967, to associate with GP established 14 years, as replacement for well-established associate of 11 years leaving for residency. Excellent income with salary guarantee and/or percentage, as desired, and eventual partnership possible. OB optional; excellent modern hospital facilities with teaching centers nearby. Modern, well-equipped, air-conditioned office—with six aides; located midway between nearby beach, mountain and desert resorts—all with outstanding recreational facilities. Nearby metropolitan area offers cultural and educational opportunities. Contact: J. C. Picklesimer, M.D., 1505 East Oak Street, Whittier, California 90605.

ASSOCIATE GP—Wide general practice, including surgery. Northwest Orange County, California. This large practice could be more than doubled with two physicians working together more efficiently than two separately. $1,500/month, for three months, then 60-40 per cent for 3 years and then equal partnership. Box No. 3005, California Medicine.

DEDICATED GENERAL PRACTITIONER OR INTERNIST—Wanted as associate in metropolitan Los Angeles, California, toward possible partnership in three years. Salary $18,000/year with consecutive yearly raises to $24,000 and $30,000. Consistent with compatibility, dedication and pace, possibilities of increased net earnings in partnership are great. Write Box No. 3025, California Medicine.

GENERAL PRACTICE ASSOCIATE—Needed immediately for busy general practice. Air-conditioned new office, beach city. Excellent hours and working conditions; well-trained practice. Salary; future partnership open. David Krugman, M.D., 630 West Third Street, Long Beach 90812.

LAKE TAHOE—Opening for general practitioner to join GP group. Salary guarantee, plus percentage. Spacious office with full lab and x-ray; 38-bed open-staff hospital three miles from office. High Sierra recreation at your doorstep. Two miles from Stateline. Tahoe Medical Center, Box 161, Al Tahoe, California 95702.

LOCUM TENENS

LOCUM TENENS: June 12 to July 12. General practice, $2,000 minimum guarantee per month or percentage if qualified. Free rent in luxury home, four bedrooms, if desired. On call one weekend per month and four nights per week. Richard J. Rihn, M.D., 166-24th Street, Richmond, California 94804.

LOCUM TENENS DOCTOR to cover general practice for 6-8 weeks in July & August or September. No OB or night calls. Daily 2-6:30 pm with mornings free. Guaranteed salary and percentage. Day or night phone: (213) 264-4111—Arthur P. Schneidman, M.D., 5016 Whittier Boulevard. Los Angeles, California 90022.

LOCUM TENENS needed for the month of June, 1967; requires California license. $2,000 per month. Contact: Frank J. Sykes, M.D., 786 H Street, Crescent City, California 95531 (Del Norte County).

MILL VALLEY—May 13 to June 23, busy general practice in beautiful Mill Valley, across the Golden Gate Bridge from San Francisco. Terms: 50 per cent of gross with $2,000 guaranteed; opportunity for partnership or independent practice. R. B. Hartman, M.D., 61 Camino Alto, Mill Valley, California 94941.

RESIDENTS WANTED

SURGICAL RESIDENCY—Third-year vacancy beginning July 1, 1967. Other AMA-approved residencies and internships. Large clinic and private service. California license necessary. Contact: Director, Medical Education, Mercy Hospital, San Diego, California 92103.

INTERNAL MEDICINE RESIDENCY—Vacancy first and third year. AMA-approved program; and other approved residencies and internships. California license necessary. Contact: Director, Medical Education, Mercy Hospital, San Diego, California 92103.

RESIDENT PHYSICIANS: First year residents in internal medicine; excellent three-year program with university teaching sessions. Three full-time teaching internists. Salary $625 per month, plus available work of approximately $200. Call (collect) or write James Reavis, M.D., Kern General Hospital, 1830 Flower, Bakersfield, California 93305; (805) 323-7651.

RESIDENT PHYSICIANS: One second-year resident to fill the only vacancy remaining in 1967-68 OB/GYN training program, Kern General Hospital, Bakersfield, California. Salary $675 per month, plus available work of approximately $200. Call (collect) or write James Reavis, M.D., 1830 Flower, Bakersfield, California 93305; (805) 323-7651.

INTERNS AND RESIDENTS OPENINGS—July 1967, all four years approved residency program in very active obstetrics/gynecology service. Excellent stipend. Kaiser Foundation Hospital, Santa Clara, California. Box No. 945, California Medicine.

PSYCHIATRY RESIDENCY POSITIONS AVAILABLE: July 1967 for three-year accredited program. A dynamically oriented, broadly based, well-balanced opportunity for sound training. Meadowbrook Hospital is the Nassau County General Hospital and is centrally located in the most desirable and beautiful area in the country. Easy commuting distance to New York City. 3,000 inpatient psychiatric admissions per year; 24-hour walk-in clinic and all psychiatric modalities. J. Martin Semer, M.D., Chairman, Department of Psychiatry, Meadowbrook Hospital, P.O. Box 175, East Meadow, Long Island, New York 11554.

LOCUM TENENS SITUATIONS WANTED

OPHTHALMOLOGIST: Need a vacation? Ophthalmology resident, age 34, will complete residency June 30, 1967. Would like locum tenens, July-August, or any part thereof. California licensed. Terms, open—will consider any reasonable offer. David Noorthoek, M.D., Highland General Hospital, 2701-14th Avenue, Oakland, California 94606.

SITUATIONS WANTED

PHYSICIAN PLACEMENTS—MD OFFICES, CLINICS AND HOSPITALS. No placement charge to employer. Physicians are available for placement in California and the West Coast. A professional placement service for physicians by a trained and experienced hospital administrator. For assistance with your physician vacancies, call or write: Conrad K. Howan, Director, Bay Medical Associates Agencies: 821 Market, suite 518, San Francisco. Telephone YU 2-6191.

PATHOLOGIST, 33, married, military obligation completed, licensed in California; will finish approved residency in a major metropolitan hospital in June 1967. Interested in association with individual or group. Write Box No. 2080, California Medicine.

ELECTROENCEPHALOGRAPHER seeks full or part-time position; extensive clinical and laboratory background. Interview can be arranged. Box No. 3045, California Medicine.

DERMATOLOGIST—Board certified, California license. University precepteeship trained at N.Y. Skin and Cancer; in successful private practice eight (8) years. Strongly motivated to make geographic change to Los Angeles or San Diego area. Association or purchase. Mutual confidentiality will be respected. Write: Professional Management-New York, 500 5th Avenue, New York, New York 10036.

CANADIAN QUALIFIED THORACIC AND GENERAL SURGEON—F.R.C.S. (C.), California license; age 44; currently conducting large consultative surgery practice in 700-bed hospital. Would like to make progressive change by moving to California. Bay area preferred, but not essential. Box No. 3035, California Medicine.

UROLOGIST—California licensed, Board eligible, in active practice —desires California location or association with individual or group. Box No. 3040, California Medicine.

OBSTETRICIAN-GYNECOLOGIST—Young, 36, married with family. Well-trained, Board certified A.C.O.G., California license. Four years satisfying group practice in mid-west; also stationed in Southern California in military. Desire to return because of child's allergy as well as climate with its associated advantages. Prefer associate(s) or group, however would consider solo. Reply Box No. 3070, California Medicine.

OFFICE ASSISTANTS

MD'S OFFICE ASSISTANTS—Well-trained, intelligent, dependable. Full-time or part-time. NO FEE. References. Skill-evaluation. Confidential interviews at your convenience. Inquire about our free internship program. Please call or write to Dental Nurses College of California, 1101 Masonic Avenue, San Francisco 94117; (415) 621-8761. "SERVING THE WEST WITH THE BEST SINCE 1935."

(Continued on Page 82)

...but doctor,
I eat like a bird!

IN WEIGHT CONTROL

OBETROL®

Each OBETROL-10 tablet contains: Methamphetamine Saccharate 2.5 mg., Methamphetamine Hydrochloride 2.5 mg., Amphetamine Sulfate 2.5 mg., Dextro-amphetamine Sulfate 2.5 mg. (OBETROL-20 tablets contain twice this potency) Pat. #2748052.

This combination of amphetamines may be useful as an adjunct in the management of certain forms of obesity where an appetite depressant is indicated.

CONTRAINDICATIONS: Hypertension, advanced arteriosclerosis, coronary artery disease, cardiac arrhythmias, peripheral vascular disease, states of undue restlessness, anxiety, excitement, agitated depression, hyperthyroidism, idiosyncrasy to amphetamine, concomitant administration of a monoamine oxidase inhibitor. PRECAUTIONS: Use with caution in individuals with anorexia, insomnia, vasomotor instability, asthenia, psychopathic personality, a history of homicidal or suicidal tendencies, and individuals who are known to be hyperreactive to sympathomimetic agents, or emotionally unstable individuals who are known to be susceptible to drug abuse. Certain monoamine oxidase inhibitors may potentiate the action of Obetrol. SIDE EFFECTS: The most common side effects attended with the use of amphetamines include nervousness, excitability, euphoria, insomnia, dryness of mouth, nausea, vertigo, constipation, and headache. DOSAGE AND ADMINISTRATION: Initial adult dose is one-half to one 'Obetrol-10' tablet daily, preferably one-half to one hour before meals. This may be gradually increased to one 'Obetrol-10' or 'Obetrol-20' tablet one to three times daily as indicated. SUPPLIED: Tablets scored, in bottles of 100, 500, and 1000.

REQUEST SAMPLES AND LITERATURE

OBETROL PHARMACEUTICALS

Div. of Rexar Pharmacal Corp. Brooklyn, N.Y. 11207

OBETROL PHARMACEUTICALS • BROOKLYN, N.Y. 11207

Dr. ____________________

Address ____________________

City ____________ State ________ Zip ______

Predicting Freedom From Heart Disease

It may be possible to assure a small group of persons between ages 39 and 59 that they won't suffer coronary heart disease for at least 4½ years.

This is suggested in a report in a December 12 *Journal of the American Medical Association.*

The authors say that after studying 3,000 healthy, middle-aged men for 4½ years, they can be reasonably certain of which ones will remain free of heart disease. They hasten to add, however, that the health assessment is not absolutely certain, and that many variables make such health predictions impossible for the general population.

The middle-aged man most likely to remain free of coronary heart disease, the report said, is one who isn't overly aggressive or ambitious. In addition, this man has low levels of serum cholesterol, triglyceride, and lipoproteins in his blood. These latter three lipids, or fat substances, have been associated with heart disease in many studies.

In 4½ years, there were only three cases of coronary heart disease among 499 men who displayed all of these characteristics, the authors said. Eighteen cases could have been expected in an average group of men the same age.

"The results of the present study suggest that apparently a relatively small group of persons between the ages of 39 and 59 can receive positive assurance valid for at least 4½ years that they . . . will not incur coronary heart disease," the authors said.

Persons who have low quantities of lipids, but are less relaxed and more ambitious cannot be given the same assurances. However, these people appear to have two to three times more resistance to heart disease than the average person.

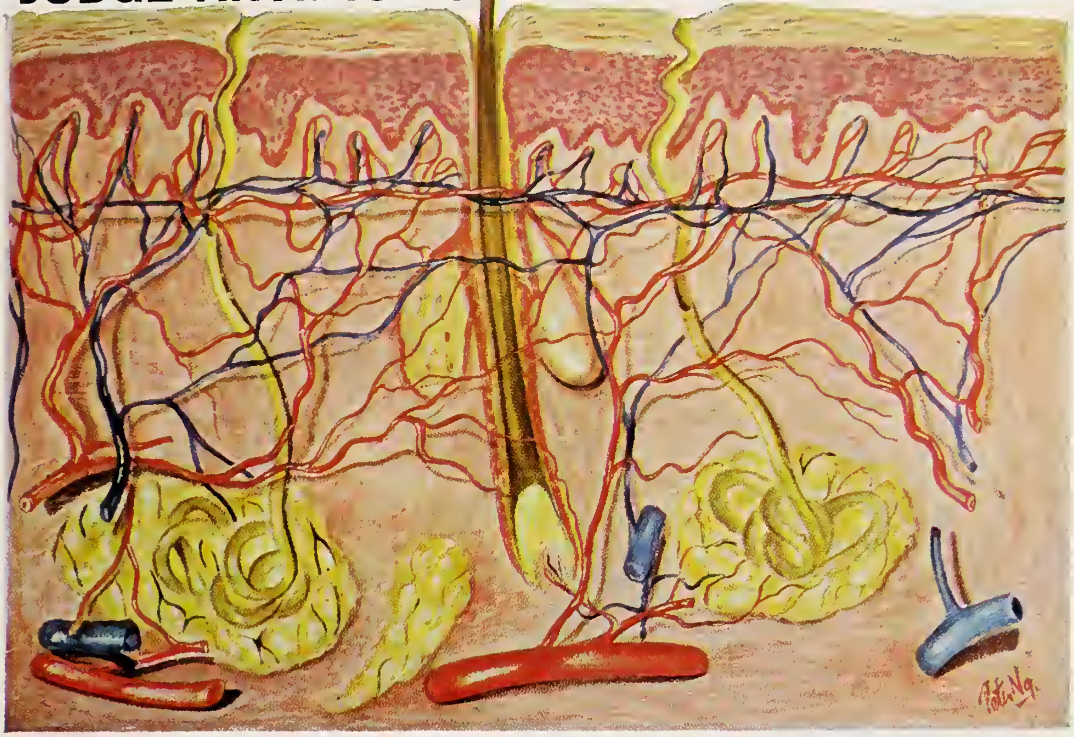
No one who is exceptionally aggressive, overly ambitious, or unusually conscious of deadlines, etc., can be given any assurance of freedom from heart disease, no matter what his levels of lipids, the authors said.

The authors are Ray H. Rosenman, M.D., and Meyer Friedman, M.D., of Mount Zion Hospital and Medical Center, San Francisco; C. David Jenkins, Ph.D., School of Public Health, University of North Carolina, Chapel Hill, and Reuben Straus, M.D., Moses Wurm, M.D., and Robert Kisitchek, M.D., of the Research Laboratories of St. Joseph Hospital, Burbank, Calif.

GIANT ULCER OF THE FACE FOLLOWING SURGERY FOR TRIGEMINAL NEURALGIA—I. S. Cliff, Jr., and D. J. Demis (Washington U. School of Medicine, St. Louis), Arch. Intern. Med., 119:218-222 (February) 1967.

A case of giant ulceration of the face, secondary to surgery for trigeminal neuralgia 12 years prior to ulceration, is reported, with clinical photographs and x-rays. This is an infrequent sequela. The frequency and occurrence of other postsurgical complications when the trigeminal nerve or parts of it are severed are discussed.

JUDGE ANTIBIOTIC OINTMENTS HERE



Results on skin are final proof of any topical antibiotic's effectiveness

No in vitro test can duplicate a clinical situation on living skin. 'Neosporin' (polymyxin B—bacitracin—neomycin) Ointment has consistently proven its effectiveness in thousands of cases of bacterial skin infection. The spectra of the three antibiotics overlap in such a way as to provide bactericidal action against most pathogenic bacteria likely to be found topically. Diffusion of the antibiotics from the special petrolatum base is rapid since they are insoluble in the petrolatum, but readily soluble in tissue fluids. The Ointment is bland and nonirritating.

Caution: As with other antibiotic preparations, prolonged use may result in overgrowth of nonsusceptible organisms and/or fungi. Appropriate measures should be taken if this occurs. Articles in the current medical literature indicate an increase in the prevalence of persons allergic to neomycin. The possibility of such a reaction should be borne in mind.

Contraindications: This product is contraindicated in those individuals who have shown hypersensitivity to any of its components.

Supplied: Tubes of 1 oz., ½ oz. with applicator tip, and ⅛ oz. with ophthalmic tip.

Complete literature available on request from Professional Services Dept. PML.

'NEOSPORIN'®
brand
POLYMYXIN B-BACITRACIN-NEOMYCIN
OINTMENT

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N.Y.

Officers OF THE CALIFORNIA MEDICAL ASSOCIATION

General Officers

President: JAMES C. MACLAGGAN, 525 Hawthorn Street, San Diego 92101. 714 233-6519

President-Elect: JOHN G. MORRISON, 333 Estudillo, San Leandro 94577. 415 483-1332

Past President: RALPH C. TEALL, 2626 L Street. Sacramento 95816. 916 448-1851

Speaker of the House of Delegates: WILLIAM F. QUINN, 1401 S. Hope Street, Los Angeles 90015. 213 749-0181

Vice-Speaker of the House of Delegates: JOSEPH W. TELFORD, 3255 Fourth Avenue, San Diego 92103. 714 295-5151

Chairman of the Council: CARL E. ANDERSON, 1150 Montgomery Drive, Santa Rosa 95405. 707 546-1922

Secretary: HELEN B. WEYRAUCH, 450 Sutter Street, San Francisco 94108. 415 392-6909

Editor: DWIGHT L. WILBUR, 655 Sutter Street, San Francisco 94102. 415 673-4080

Executive Director: HOWARD HASSARD, 693 Sutter Street, San Francisco 94102. 415 776-9400

General Counsel: PEART, HASSARD, SMITH and BONNINGTON, 44 Montgomery Street, San Francisco 94104. 415 982-8585

* * *

District Councilors

FIRST DISTRICT—Roger C. Isenhour, (1967), 2881 Fourth Avenue, San Diego 92103. San Diego County. 714 295-2137

SECOND DISTRICT—Imperial, Inyo, Mono, Orange, Riverside and San Bernardino Counties—
- Office No. 1—Henry V. Eastman (1967), 661 W. First Street, Tustin 92680. 714 544-4560
- Office No. 2—Frank C. Melone (1968), 527 N. Palm Avenue, Ontario 91761. 714 986-1104

THIRD DISTRICT—Los Angeles County—
- Office No. 1—Malcolm C. Todd (1968), 2865 Atlantic Avenue, Long Beach 90806. 213 426-1355
- Office No. 2—Elmer F. Gooel (1969), 465 N. Roxbury Drive, Beverly Hills 90210. 213 271-5745
- Office No. 3—Richard L. Taw (1967), 3741 Stocker Street, Los Angeles 90008. 213 293-7157
- Office No. 4—Lewis T. Bullock (1968), 3875 Wilshire Boulevard, Los Angeles 90005. 213 382-2209
- Office No. 5—Joseph P. O'Connor (1969), 595 E. Colorado Boulevard, Pasadena 91101. 213 792-2154
- Office No. 6—Franklin F. Ham (1967), 14556 Hamlin Street, Van Nuys 91401. 213 786-5711
- Office No. 7—Wilbur G. Rogers (1968), 1122 N. Brand Boulevard, Glendale 91202. 213 241-3125
- Office No. 8—Jean F. Crum (1969), 8355 E. 3rd Street, Downey 90241. 213 869-1091
- Office No. 9—Joseph F. Boyle (1967), 1136 W. Sixth Street, Los Angeles 90017. 213 482-9891

FOURTH DISTRICT—Joseph F. Maguire (1967), 2755 Loma Vista Road, Ventura 93003. San Luis Obispo, Santa Barbara and Ventura Counties. 805 648-2567

FIFTH DISTRICT—Ralph W. Burnett (1969), 515 Truxton Avenue, Bakersfield 93301. Calaveras, Fresno, Kern, Kings, Madera, Mariposa, Merced, San Joaquin, Stanislaus, Tulare and Tuolumne Counties. 805 327-4411

SIXTH DISTRICT—Monterey, San Benito, San Mateo, Santa Clara and Santa Cruz Counties.
- Office No. 1—Richard S. Wilbur (1967), 300 Homer Avenue, Palo Alto 94301. 415 321-4121
- Office No. 2—Albert G. Miller (1968), 77 San Mateo Drive, San Mateo 94401. 415 343-9074

SEVENTH DISTRICT—San Francisco County—
- Office No. 1—Malcolm S. M. Watts (1969), 909 Hyde Street, San Francisco 94109. 415 776-3838
- Office No. 2—Roberta F. Fenlon (1967), 490 Post Street, San Francisco 94102. 415 986-1391

EIGHTH DISTRICT—Alameda and Contra Costa Counties—
- Office No. 1—Harold Kay (1969), 411 Thirtieth Street, Oakland 94609. 415 832-4822
- Office No. 2—William F. Kaiser (1967), 3000 Colby Street, Berkeley 94705. 415 843-6515

NINTH DISTRICT—Carl E. Anderson (1967), 1150 Montgomery Drive, Santa Rosa 95405. Del Norte, Humboldt, Lake, Marin, Mendocino, Napa, Solano and Sonoma Counties. 707 546-1922

TENTH DISTRICT—James H. Yant (1968), 2600 Capitol Avenue, Sacramento 95816. Alpine, Amador, Butte, Colusa, El Dorado, Glenn, Lassen, Modoc, Nevada, Placer, Plumas, Sacramento, Shasta, Sierra, Siskiyou, Sutter, Tehama, Trinity, Yolo and Yuba Counties. 916 446-5278

ELEVENTH DISTRICT—Forest J. Grunigen (1967), 3919 Beverly Boulevard, Los Angeles 90004. Forty First Medical Society. 213 663-1161

SCIENTIFIC BOARD REPRESENTATIVE—William P. Longmire, Jr. (1967), UCLA Department of Surgery, Los Angeles 90024. 213 272-8911

* * *

COMMISSION ON *Medical Services*

Ralph Burnett. Bakersfield
Elmer F. Gooel, Beverly Hills (Secty.)
Arthur F. Howard, Fresno (Chmn.)
Charles B. Hudson, Oakland
Homer C. Pheasant, Los Angeles
Milo A. Youel, San Diego.

Committee on Fees

H. Dean Hoskins, Oakland	1967
Wallace A. Gerrie, Jr., Newport Beach	1967
Robert L. Watson, Jr., Los Angeles	1967
Roger C. Isenhour, San Diego	1968
Alfred J. Murrieta, Jr., Los Angeles	1968
Maurice S. Salomon, San Francisco	1968
Homer C. Pheasant, Los Angeles (Chmn.)	1969
Glenn A. Pope, Sacramento	1969
Leo Bell, San Mateo	1969
Joseph F. Boyle, Los Angeles	1969

CONSULTANT: William H. Thompson, San Mateo.

Committee on Federal Medical Care Programs

Charles B. Canby, Van Nuys	1967
Merlin A. Hendrickson, Rialto	1967
Ralph L. Hoffman, San Diego	1967
John S. McKee, Jr., San Diego	1968
Arthur Howard, Fresno (Chmn.)	1968

Committee on Veterans Affairs

Charles B. Hudson, Oakland (Chmn.)
Ben D. A. Miano, San Bernardino
Robert L. Watson, Los Angeles
CONSULTANT: Lloyd E. Gould, Napa.

Committee on Mediation

Eli A. Layon, Brawley	1967
Milo A. Youel, San Diego (Chmn.)	1967
Richard L. Taw, Los Angeles	1968
Albert G. Clark, San Francisco	1969
George Babbin, Sacramento	1969

Committee on Insurance and Prepayment

Oscar W. Hills, San Mateo	1967
Dave F. Dozier, Sacramento	1967
Ralph W. Burnett, Bakersfield (Chmn.)	1968
Stanley J. Murasky, Reseda	1968
Ivan Neubauer, San Jose	1969
Thayer A. Smith, Norwalk	1969

* * *

COMMISSION ON *Public Agencies*

Lewis T. Bullock, Los Angeles
Joseph P. Cosentino, Sacramento (Secty.)
William F. Kaiser, Berkeley
Howard W. Lindsey, Jr., San Mateo
Albert Miller, San Mateo (Chmn.)
Stanley A. Skillicorn, San Jose
Howell E. Wiggins, San Diego
Joseph P O'Connor, Pasadena
CONSULTANT: Malcolm C. Todd, Long Beach.

Committee on Public Health

Robert L. Garrett, Vallejo	1967
Karl L. Schaupp, Jr., San Francisco	1967
Allen F. Sterling, San Bernardino	1967
Austin W. Matthis, Sacramento	1967
Albert G. Miller, San Mateo	1968
Stanley A. Skillicorn, San Jose (Chmn.)	1968
Alfred F. Kandlbinder, Monterey	1969
Robert Watson, Jr., Los Angeles	1969
Samuel S. Woolington, Long Beach	1969

Committee on Mental Health

Emmanuel Silver, Palo Alto	1967
Ira H. Degenhardt, San Rafael	1967
Rudolph B. Toller, Stockton	1967
John A. Stroud, III, Sacramento	1967
Alfred Auerback, San Francisco	1968
David H. Payne, Los Angeles	1968
Jack B. Lomas, Beverly Hills	1968
Joseph J. Furlong, San Francisco	1969
Stuart C. Knox, Los Angeles (Chmn.)	1969

Committee on Welfare Medical Care Programs

Clyde L. Boice, Palo Alto	1967
Edgar Rosen, Oakland	1967
Thomas N. Elmendorf, Willows	1967
Vincent P. Carroll, Laguna Beach	1967
Orrin S. Cook, Jr., Sacramento	1968
Robert F. Schell, San Rafael	1968
William F. Quinn, Los Angeles	1969
James Dalton, Santa Barbara	1969
Arthur F. Howard, Fresno	1969
Howard W. Lindsey, Jr., San Mateo (Chmn.)	1969

SUBCOMMITTEE ON ADOPTIONS: Walter Beckh, San Francisco; Fred M. Kay, Fullerton; Robert J. McNeil, Los Angeles, (Chmn.); Donald Minkler, Berkeley; Dale W. Ritter, Chico; Ben J. Siebenthal, Sacramento.

Committee on Occupational Health and Rehabilitation

John H. Leimbach, Jr., San Francisco	1967
Herbert A. Holden, San Leandro	1967
Howell E. Wiggins, San Diego (Chmn.)	1967
Edward Zaik, Los Angeles	1968
Packard Thurber, Jr., Los Angeles	1968
Thomas Gucker, III, Los Angeles	1968
Herbert A. Lints, Oakland	1969
Joseph N. Vizzard, Los Gatos	1969
Patrick J. Maloney, Merced	1969

* * *

COMMISSION ON *Community Health Services*

Wayne P. Chesbro, Berkeley
Charles G. Hutter, Jr., Hollywood
Gerald G. Jampolsky, Tiburon (Secty.)
Elmer R. Jennings, Long Beach
Harold Kay, Oakland (Chmn.)
George A. Martin, Redding
Pierre Salmon, San Mateo
Leopold J. Snyder, Fresno
Beverly G. Stewart, Bakersfield

Committee on Rural Health

Mitry P. Ajalat, Calexico	1967
Richard W. Johnson, Arbuckle	1967
Leopold J. Snyder, Fresno (Chmn.)	1968
Erwin P. Brauner, Visalia	1968
Carrol W. Goss, Lamont	1968
Franklin L. Murphy, Oroville	1969
Thomas Rossitto, Auburn	1969
M. Marlin Clark, Delano	1969

Committee on School Health

B. Otis Cobb, Martinez	1967
Gerald G. Jampolsky, Tiburon (Chmn.)	1967
Byron H. Demorest, Sacramento	1967
William A. Leovy, La Mesa	1968
Tom W. Robinson, Newport Beach	1968
Glenn Austin, Los Altos	1968
Ruth A. Frary, Watsonville	1968
Leslie M. Holve, Los Angeles	1969
Henry B. Bruyn, Berkeley	1969

Committee on Health Care of the Aged

Franklin F. Ham, Van Nuys	1967
Pierre Salmon, San Mateo (Chmn.)	1967
Charles E. Schoff, Jr., Sacramento	1968
Raymond D. Manchester, Visalia	1968
Leo A. Wagner, Novato	1969
Lloyd E. Gould, Napa	1969
Walter Batchelder, San Mateo	1969

Committee on Disaster Medical Care

Charles C. Henderson, San Mateo	1967
Donald E. Barker, Van Nuys	1967
Wayne P. Chesbro, Berkeley (Chmn.)	1968
Michael J. Hitchko, Eureka	1968
J. T. McNally, Stockton	1969
John Heald, San Francisco	1969

CONSULTANTS: Charles N. Earl, Arcata; Frank F. Schade, Los Angeles; Robert S. Johnson, Chico; Stephen Bruny, Fresno.

Committee on Automotive and Traffic Safety

George C. Snively, Sacramento	1967
Beverly G. Stewart, Bakersfield (Chmn.)	1967
Arnold R. Friesen, Pasadena	1968
Joseph F. Maguire, Ventura	1969
Thomas W. Lyons, La Mesa	1969
Robert L. Tour, San Francisco	1969

CONSULTANTS: Julian A. Waller, Berkeley; Julian M. Sether, Los Angeles.

Committee on Medical Aspects Of Sports and Physical Fitness

John F. Blinn, Stockton	1967
Robert K. Kerlan, Los Angeles	1967
George S. Buehler, Whittier	1968
Philip H. McFarland, Fullerton	1968
Tom W. Robinson, Newport Beach	1968
Charles G. Hutter, Jr., Hollywood (Chmn.)	1969
Arnold A. Nutting, San Rafael	1969

Committee on Environmental Health

George A. Martin, Redding	1967
Robert L. Hippen, San Diego	1967
Grace M. Talbott. San Francisco	1968
Francis M. Pottenger, Jr., Monrovia	1968
Reginald H Smart. Los Angeles	1968
Douglas Campbell, San Francisco	1969
John R. Lee, Mill Valley	1969

Committee on Blood Banks

Robert W. Purvis, Modesto	1967
Osman W. Hull, Monterey	1967
James H. Yant, Sacramento	1967
Elmer Jennings, Long Beach (Chmn.)	1968

CONSULTANT: Melvin L. Sommer, Beverly Hills

COMMISSION ON *Communications*

Frederick Ackerman, Pleasant Hill
L. Morgan Boyers, Sacramento (Secty.)
John A. E. Bullis, Los Angeles
Roberta F. Fenlon, San Francisco
Elmer F. Gooel, Beverly Hills
Walter W. Hopps, Los Angeles
George Schwartz, Beverly Hills
Malcolm C. Todd, Long Beach (Chmn.)
Richard S. Wilbur, Palo Alto
EX-OFFICIO: James C. MacLaggan, San Diego; John G. Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Scientific Board

David A. Rytand, Palo Alto	1967
William W. Belford, San Diego	1967
Jerome W. Bettman, San Francisco	1967
John B. Dillon, Los Angeles	1967
Justin J. Stein, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
R. Kirklin Ashley, San Francisco	1967
Victor Richards, San Francisco	1967
David Rubin, Los Angeles	1967
Keith P. Russell, Los Angeles	1967
Edward J. Zaik, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Seymour J. Brockman, Los Angeles	1968
S. Austin Jones, Los Angeles	1968
Hobart M. Kelly, Riverside	1968
Saul J. Robinson, San Francisco	1968
Eugene Ziskind, Los Angeles	1968
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. deC. M. Saunders, San Francisco	1968
Herbert A. Holden, San Leandro	1968
David A. Wood, San Francisco	1968
Arthur A. Clinco, Los Angeles	1968
R. Theodore Bergman, Los Angeles	1968
Leland B. Blanchard, San Jose	1969
L. S. Goerke, Los Angeles	1969
Bert L. Halter, San Francisco	1969
Ellis Jones, Pasadena	1969
Carl McCandless, San Francisco	1969
J. Blair Pace, Oceanside	1969
Donald W. Petit, Los Angeles	1969
Glenn Pope, Sacramento	1969
Robert J. Prentiss, San Diego	1969
Edward B. Shaw, San Francisco	1969
Ralph E. Worden, Los Angeles	1969

COUNCIL REPRESENTATIVES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.
EX-OFFICIO: Dwight L. Wilbur, San Francisco.

Executive Committee

William W. Belford, San Diego	1967
Victor Richards, San Francisco	1967
Justin J. Stein, Los Angeles	1967
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. Dillon, Los Angeles	1969
Donald Petit, Los Angeles	1969

COUNCIL DESIGNEES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.

Committee on Nominations

Justin J. Stein, Los Angeles (Chmn.)	1967
Edward B. Shaw, San Francisco	1969
Robert J. Prentiss, San Diego	1969

Committee on Scientific Assemblies

John B. Dillon, Los Angeles (Chmn.)	1967
Victor Richards, San Francisco	1967
David A. Rytand, Palo Alto	1967
John B. deC. M. Saunders, San Francisco	1968
Ellis Jones, Pasadena	1969
Donald Petit, Los Angeles	1969

CONSULTANT: Claron Oakley, Los Angeles

Committee on Continuing Medical Education

John B. Dillon, Los Angeles	1967
Charles E. Grayson, Sacramento	1967
Saul J. Robinson, San Francisco	1968
Donald W. Petit, Los Angeles (Chmn.)	1969
Bert L. Halter, San Francisco	1969
J. Blair Pace, Oceanside	1969

Committee on CALIFORNIA MEDICINE

David Rubin, Los Angeles	1967
R. Kirklin Ashley, San Francisco	1967

Committee on Scientific Information

Justin J. Stein, Los Angeles	1967
Edmund L. Keeney, La Jolla	1968
Arthur A. Clinco, Los Angeles	1968
Edward B. Shaw, San Francisco (Chmn.)	1969
L. S. Goerke, Los Angeles	1969

Committee on Maternal and Child Care

Russell W. Mapes, Beverly Hills	1967
Keith P. Russell, Los Angeles (Chmn.)	1967
Forrest H. Howard, Garden Grove	1967
Charles M. Blumenfeld, Sacramento	1968
Herbert A. Holden, San Leandro	1968
Hobart M. Kelley, Riverside	1968
Leland Blanchard, San Jose	1969

Committee on Cancer

John W. Cline, San Francisco	1967
Robert W. Jamplis, Palo Alto	1967
David A. Wood, San Francisco (Chmn.)	1967
John G. Walsh, Sacramento	1968
Lewis W. Guiss, Los Angeles	1969
Robert Prentiss, San Diego	1969

SUBCOMMITTEE ON CANCER EDUCATION: Sol R. Baker, Beverly Hills; Robert W. Jamplis, Palo Alto (Chmn.); Maurice Galante, San Francisco; Justin J. Stein, Los Angeles.
SUBCOMMITTEE ON TUMOR TISSUE REGISTRY: John W. Cline, San Francisco (Chmn.); Lewis W. Guiss, Los Angeles; James E. Kahler, Los Angeles; Carl M. McCandless, San Francisco; Weldon K. Bullock, Los Angeles.
SUBCOMMITTEE ON CONSULTATIVE TUMOR BOARDS: Bert L. Halter, San Francisco; Carl L. Moore, Bakersfield; Victor Richards, San Francisco; John G. Walsh, Sacramento (Chmn.).

Committee on Adverse Drug Reactions

Edward R. Bloomquist, Glendale	1967
Ralph O. Wallerstein, San Francisco	1967
John F. Murray, Fresno (Chmn.)	1968
Russell J. Tat, San Francisco	1969
William W. Thayer, Stockton	1969

CONSULTANT: John W. Brown, Berkeley; Ralph W. Weilerstein, Berkeley.
SUBCOMMITTEE ON DANGEROUS DRUGS: Edward Bloomquist, Glendale; Norman A. Gale, San Diego; Edward T. Jewett, Santa Rosa; William F. Quinn, Los Angeles (Chmn.); Helen B. Weyrauch, San Francisco.
CONSULTANT: Ralph W. Weilerstein, Berkeley.

Committee on Cardiovascular Diseases

Harold I. Griffeath, San Francisco (Chmn.); Lewis T. Bullock, Los Angeles; David J. New, Modesto; George C. Griffith, Los Angeles.
CONSULTANT: John E. Connolly, Los Angeles.

* * *

BUREAU OF *Research and Planning*

J. Brandon Bassett, Oakland
John Sheehy, Riverside
Samuel Sherman, San Francisco (Chmn.)
H. Russell Fisher, Glendale
G. Roger Ellison, Pomona
EX-OFFICIO: Malcolm S. M. Watts, San Francisco; Ralph C. Teall, Sacramento.
CONSULTANT: Gerald W. Shaw, Santa Monica.

* * *

COMMISSION ON *Professional Welfare*

Bert L. Halter, San Francisco
George K. Herzog, Jr., San Francisco (Chmn.)
Francis E. West, San Diego
James H. Yant, Sacramento (Secty.)

Committee on Physicians Group Insurance

Robert J. Wescott, El Centro	1967
Robert X. DeMangus, Glendale	1967
John B. Long, Sacramento	1967
George K. Herzog, Jr., San Francisco (Chmn.)	1968
John Hogan, San Diego	1969
Homer C. Pheasant, Los Angeles	1969

Liaison Committee to Medical Schools

Samuel R. Irvine, Beverly Hills	1967
Francis E. West, San Diego	1967
Frank J. Novak, Menlo Park	1968
Stanley Cowell, Los Angeles	1968
Bert L. Halter, San Francisco (Chmn.)	1969
Alonzo Neufeld, Los Angeles	1969
Herbert Jenkins, Sacramento	1969

EX-OFFICIO: James C. MacLaggan, San Diego; John G Morrison, San Leandro; Carl E. Anderson, Santa Rosa.

Medical Review and Advisory Committee

Eugene M. Webb, San Francisco	1967
James H. Yant, Sacramento (Chmn.)	1967
John B. Dillon, Los Angeles	1968
Carl Gnetsch, Berkeley	1968
Donald Donisthorpe, Los Angeles	1969

CONSULTANT: Don Harper Mills, Long Beach.

Liaison Committee to State Bar of California

David Rubsamen, Berkeley	1967
John V. Fiore, Westchester	1967
Francis E. West, San Diego (Chmn.)	1968
Carl Goetsch, Berkeley	1968
Robert J. McNeil, Los Angeles	1969
Henry A. Brown, San Mateo	1969

* * *

COMMISSION ON *Allied Health Professions and Services*

Roger W. Barnes, Los Angeles (Secty.)
Forest J. Grunigen, Los Angeles (Chmn.)
Frank C. Melone, Ontario
Thomas W. Ledwich, Napa
C. Gerald Scarborough, San Jose
Richard H. Schug, Long Beach

Committee on Paramedical Personnel

Frank C. Melone, Ontario (Chmn.)	1967
Gregory Bard, San Francisco	1968
Elmer R. Jennings, Long Beach	1968
Gerson R. Biskind, San Francisco	1969
Joseph Schaefer, Santa Rosa	1969
Leonard M. Asher, Los Angeles	1969
Richard H. Schug, Long Beach (Chmn.)	1969

Committee on Other Professions

George S. Buehler, Whittier	1967
Burt L. Davis, Palo Alto	1967
C. Gerald Scarborough, San Jose (Chmn.)	1968
William D. Evans, Los Angeles	1968
J. Gordon Epperson, Oakland	1969
Lewis T. Bullock, Los Angeles	1969

Liaison Committee to California Medical Assistants Association

Leon O. Burke, Sacramento	1967
John C. Brennan, Pico Rivera	1967
H. Russell Irwin, San Diego	1967
Peary Benn Berger, Inglewood	1968
Judith D. Smith, Vallejo	1969
Sanford E. Feldman, San Francisco	1969
Richard H. Schug, Long Beach (Chmn.)	1969

CONSULTANTS: Leland W. Bonar, Redding; Henry Bodner, Van Nuys.

Committee on Medicine and Religion

Albert E. Long, Daly City	1967
Roger W. Barnes, Los Angeles (Chmn.)	1967
Max Nelson, National City	1968
Frank J. Glassy, Sacramento	1969
Richard J. Lescoe, Torrance	1969

Committee on Voluntary Health Agencies

H. Mason Hohl, Beverly Hills	1967
Thomas W. Ledwich, Napa (Chmn.)	1968
Harvey May Cordua, Jr., San Diego	1969
J. Brandon Bassett, Oakland	1969

* * *

Judicial COMMISSION

Donald A. Charnock, Los Angeles	1967
Albert E. Long, Daly City	1967
Donald Cass, Hollywood	1967
Floyd O. Due, Oakland	1968
Sidney J. Shipman, San Francisco	1968
Francis E. West, San Diego (Chmn.)	1968
Douglas G. Campbell, San Francisco	1969
W. Philip Corr, Riverside (Secty.)	1969
L. E. Wilson, Anaheim	1969

* * *

COMMISSION ON *Hospital Affairs*

William L. Argo, Fresno (Secty.)
James C. Doyle, Beverly Hills
Bert L. Halter, San Francisco (Chmn.)
John C. Lungren, Long Beach
Glenn Pope, Sacramento
John T. Saidy, San Mateo
Joseph W. Telford, San Diego
Frederic N. Tyroler, Los Angeles

Medical Staff Survey Committee

Robert M. Adams, San Leandro
W. L. Argo, Fresno
Daniel W. Black, Castro Valley
Rush M. Blodget, Jr., Redding
Bert L. Halter, San Francisco (Chmn.)
J. Ralph Hughes, San Diego
Wm. Kenneth Jennings, Santa Barbara
Donald O. Lagerlof, Los Angeles
John C. Lungren, Long Beach
William A. Newsom, San Francisco
James L. Rhee, Oakland
John T. Saidy, San Mateo
Wilfred J. Snodgrass, Santa Monica
Alexander Hatoff, Oakland
Shepard F. Palitz, Bakersfield
William L. Sumption, Santa Monica
A. Jerome Hurter, Pasadena
Ernest R. Barnett, Alhambra
Richard L. Taw, Los Angeles
Richard H. McDonald, Garden Grove
Robert M. Zweig, Riverside
L. Morgan Boyers, Sacramento
James O. Farley, Sacramento
Joseph W. Telford, San Diego
Paul Deeb, Redlands
Maxwell Goldstein, Fontana
Ray Seavers, Jr., San Bernardino
William Soldmann, La Mesa
Lloyd Milburn, San Francisco
Frank Solomon, San Francisco
Sanford Feldman, San Francisco
John Bazzano, San Francisco
Kenneth Allen, San Mateo
Martin Kohn, San Bruno
William Stephenson, Santa Barbara
Merlin Hendrickson, Rialto
Andrew D. Lucine, Campbell
Fred Haven Draper, San Jose
Wayne O. Buck, San Jose
William A. Reeves, Mt. View
A. A. Thurlow, Jr., Santa Rosa
John A. Carollo, Sabastopol
William Rector, Sacramento
Gerald W. Shaw, Santa Monica
Charles L. Lear, Norwalk
John R. Heckman, Marysville
Robert Buckley, Santa Monica
Woodrow W. Burgess, Sacramento
Ira H. Degenhardt, San Rafael
Arthur E. Gladman, Oakland
Norman Graff, San Mateo
Frank V. Hoffman, Huntington Beach
Robert E. James, Belmont
Clement O. Juul, Oakland
Seymour Kolko, San Mateo
Melvin M. Lipsett, Berkeley
Jack B. Lomas, Los Angeles
Edward A. Macklin, San Francisco
D. Stuart MacRobbie, Berkeley
Felix H. Ocko, Berkeley
William R. Sheehy, Berkeley
John A. Stroud, Sacramento
R. J. Sutherland, Oakland
Rudolph B. Toller, Stockton
Albert T. Voris, Belmont
G. Cameron Cray, Palo Alto

(Continued on Page 60)

Officers of Scientific Sessions

Members who contemplate presentation of scientific papers should promptly address the secretary of the proper section at the address shown below.

Allergy

Leo N. Meleyco.......Chairman
2040 Forest Avenue, San Jose 95128

Ernest J. Saslow.......Secretary
2011-18th Street, Bakersfield 93301

Samuel Ross ....Assistant Secretary
2946 Fresno Street, Fresno 93706

Anesthesiology

Thomas W. McIntosh.......Chairman
686 East Union St., Pasadena 91101

Donald B. Dose.......Secretary
5625 Linfield Ave., San Diego 92120

Donald A. Walker.......Assistant Secretary
35 Spring Lane, Tiburon 94920

Dermatology

Robert G. Walton ....Chairman
1700 McHenry Village Way, Modesto 95350

Samuel Ayres, III.......Secretary
405 N. Bedford Rd., Beverly Hills 90210

George T. Wilson.......Assistant Secretary
Welch and Pasteur Rd., Stanford Medical Plaza
Palo Alto 94304

General Practice

J Blair Pace.......Chairman
408 Cassidy St., Oceanside 92054

Alfred F. Kandlbinder.......Secretary
302 Via Paraiso St., Monterey 93940

Allen F. Sterling.......Assistant Secretary
1285 D St., San Bernardino 92045

General Surgery

Seibert Pearson ....Chairman
2865 Atlantic Ave., Long Beach 90807

Albert G. Clark.......Secretary
450 Sutter St., San Francisco 94108

Robert T. Hood, Jr.......Assistant Secretary
2010 Wilshire Blvd., Los Angeles 90057

Industrial Medicine and Surgery

Leon R. Rudnick.......Chairman
15921 E. 14th St., San Leandro 94578

Raymond O. Oja ....Secretary
525 Golden Gate Ave., San Francisco 94102

James G. Forgerson.......Assistant Secretary
Metropolitan Life Insurance Company
600 Stockton St., San Francisco 94108

Internal Medicine

Robert L. Paver.......Chairman
384 Post St., San Francisco 94108

John L. Benton, Jr.......Secretary
1216 Wilshire Blvd., Los Angeles 90017

Edgar Rosen ....Assistant Secretary
1340 K St., Sacramento 95814

Obstetrics and Gynecology

Lester T. Hibbard.......Chairman
142 S. Windsor Blvd., Los Angeles 90004

Warren E. Jones.......Secretary
1380-45th Street, Sacramento 95819

James W. Ravenscroft.......Assistant Secretary
2330 First Ave., San Diego 92101

Ophthalmology

Victor G. Fellows.......Chairman
595 Buckingham Way, San Francisco 94117

Wendell C. Irvine ....Secretary
3875 Wilshire Blvd., Los Angeles 90005

Robert E. Hopkins.......Assistant Secretary
4255 Pacific Ave., Stockton 95207

Orthopedics

David C. Monsen.......Chairman
2300 S. Hope St., Los Angeles 90007

Arthur R. Hartwig.......Secretary
490 Post St., San Francisco 94102

Vernon L. Smythe.......Assistant Secretary
3875 Wilshire Blvd., Los Angeles 90005

Otolaryngology

G. Howard Gottschalk ....Chairman
8618 S. Sepulveda Blvd., Los Angeles 90045

Charles P. Lebo ....Secretary
490 Post St., San Francisco 94102

Irving L. White.......Assistant Secretary
911 San Antonio Dr., Long Beach 90807

Pathology

Frank R. Dutra.......Chairman
20103 Lake Chabot Rd., Castro Valley 94546

William H. Kern.......Secretary
679 S. Westlake Ave., Los Angeles 90057

Donald L. Bittner.......Assistant Secretary
Bay Area Laboratories, 2245 Post St.,
San Francisco 94115

Pediatrics

Richard L. Anderson.......Chairman
636 Harris St., Eureka 95501

Sherrod C. Swift.......Secretary
14619 Whittier Blvd., Whittier 90605

Robert J. Harvey.......Assistant Secretary
909 Hyde St., San Francisco 94109

Physical Medicine

Rene Cailliet ....Chairman
4900 Sunset Blvd., Los Angeles 90027

Anton A. Tratar.......Secretary
2360 Clay St., San Francisco 94115

Elizabeth Austin ....Assistant Secretary
Los Angeles County Hospital
1200 N. State St., Los Angeles 90033

Preventive Medicine

Henrik L. Blum.......Chairman
School of Public Health, U.C., Room 409, Earl Warren Hall, Berkeley 94720

Maxwell Rosenblatt ....Secretary
Los Angeles County Health Department,
220 N. Broadway, Los Angeles 90012

Russell B. Watson.......Assistant Secretary
Orange County Health Department, Santa Ana 92701

Psychiatry and Neurology

Werner M. Mendel.......Chairman
1934 Hospital Place, Los Angeles 90033

Ames Fischer ....Secretary
Langley Porter Inst., 401 Parnassus Ave.,
San Francisco 94122

Howard S. Barrows.......Assistant Secretary
2025 Zonal Ave., Los Angeles 90033

Radiology

Victor G. Mikity.......Chairman
1200 State St., Los Angeles 90033

Vernon R. Gee.......Secretary
2020 Court St., Redding 96001

Richard P. Storrs.......Assistant Secretary
2131 West 3rd St., Los Angeles 90057

Urology

Carl K. Pearlman.......Chairman
1518 N. Sycamore, Santa Ana 92701

James L. Goebel.......Secretary
51 Loma Linda, Ross 94957

Robert O. Pearman.......Assistant Secretary
5353 Balboa Blvd., Encino 91316

Delegates and Alternates

TO THE AMERICAN MEDICAL ASSOCIATION

DELEGATES		ALTERNATES
Francis E. West, San Diego	(1966-1967)	Vincent P. Carroll,* Laguna Beach
Samuel R. Sherman, San Francisco	(1966-1967)	Albert G. Miller, San Mateo
Henry Gibbons, III, San Francisco	(1966-1967)	A. B. Sirbu, San Francisco
John M. Rumsey, San Diego	(1966-1967)	Carl E. Anderson, Santa Rosa
Eugene F. Hoffman, Los Angeles	(1966-1967)	Joseph F. Boyle, Los Angeles
Warren L. Bostick, Los Angeles	(1966-1967)	Walter H. Brignoli, St. Helena
J. B. Price, Santa Ana	(1966-1967)	Donald C. Dodds, Oakland
Ralph C. Teall, Sacramento	(1966-1967)	Robb Smith, Orange Cove
James C. Doyle, Beverly Hills	(1966-1967)	Herman H. Stone, Riverside
Wilbur G. Rogers, Glendale	(1966-1967)	Carl M. Hadley, San Bernardino
Charles B. Hudson, Oakland	(1966-1967)	Robert L. Watson, Jr., Los Angeles
John G. Morrison, San Leandro	(1967-1968)	Frederick Ackerman, Pleasant Hills
William F. Quinn, Los Angeles	(1967-1968)	Dudley M. Cobb, Los Angeles
Robert C. Combs, San Francisco	(1967-1968)	Emmett Rixford, San Francisco
Homer C. Pheasant, Los Angeles	(1967-1968)	James C. MacLaggan, San Diego
Alfred J. Murrieta, Los Angeles	(1967-1968)	Harold Kay, Oakland
Burt L. Davis, Palo Alto	(1967-1968)	Leon P. Fox, San Jose
Arlo A. Morrison, Ventura	(1967-1968)	Henry Brown, San Mateo
James E. Feldmayer, Exeter	(1967-1968)	Charles Grayson, Sacramento
O. W. Wheeler, Riverside	(1967-1968)	Wilbur Bailey, Los Angeles
Malcolm C. Todd, Long Beach	(1967-1968)	Neil C. Hamel, Encino
J. E. Vaughan, Bakersfield	(1967-1968)	Joseph P. O'Connor, Pasadena

*(Delegates and Alternates to the AMA are elected for terms of two calendar years. Each office is scheduled for election at the Annual Session of the second year shown above.) *Council appointee.*

Other Organizations and Medical Schools

Board of Medical Examiners of the State of California

Secretary.......Shelby M. Hicks, M.D.
1021 O Street, Room A202, Sacramento 95814
San Francisco.......30 Van Ness Ave., 94102
Room 2100
Los Angeles.......107 South Broadway 90012
Sacramento.......1021 O Street, Room A547 95814

The Public Health League of California

Executive Secretary.......Ben H. Read
955 South Western Ave., Los Angeles 90006
Assistant Executive Secretary.......Al Paul Putnam
693 Sutter Street, San Francisco 94102
San Francisco.......693 Sutter Street 94102
415 474-1061
Los Angeles.......955 South Western Ave. 90006
213 731-6397
Sacramento.......1127-11th Street, 95814
916 444-7496

Department of Public Health of the State of California

Director.......Lester Breslow, M.D.
2151 Berkeley Way, Berkeley 94704
Berkeley.......2151 Berkeley Way 94704
415 843-7900
Sacramento.......1927-13th Street 95814
916 445-2264
Los Angeles.......417 South Hill Street 90013
213 620-2900

Medical Schools in California

University of California School of Medicine, Medical Center, San Francisco 94122. Dean: Stuart C. Cullen, M.D.

Stanford University School of Medicine, 300 Pasteur Drive, Palo Alto 94304. Dean: Robert J. Glaser, M.D.

University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 90033. Dean: Roger O. Egeberg, M.D.

Loma Linda University School of Medicine, Loma Linda 92354. Dean: David Burdg Hinshaw, M.D.

University of California at Los Angeles, School of Medicine, Hilgard Avenue, Los Angeles 90024. Dean: Sherman M. Mellinkoff, M.D.

University of California, California College of Medicine, 1721 Griffin Avenue, Los Angeles 90031. Dean: Warren L. Bostick, M.D.

University of California, San Diego, School of Medicine, La Jolla 92038. Acting Dean: Robert D. Tschirgi, M.D.

University of California, Davis, School of Medicine, Davis 95616. Dean: Charles John Tupper, M.D.

California Medical Education and Research Foundation

Directors: Ralph C. Teall, M.D., President; Carl E. Anderson, M.D., Vice-President; Howard Hassard, Secretary; James C. MacLaggan, M.D.; Harold Kay, M.D.; William F. Quinn, M.D.; E. B. Shaw, M.D.

Roster — CALIFORNIA COMPONENT MEDICAL SOCIETIES

Society secretaries are requested to notify California Medicine promptly when changes are indicated in their roster information

ALAMEDA-CONTRA COSTA Medical Assn., 6230 Claremont Ave., Oakland 94618. Meets quarterly.
Frederick W. Ackerman........President
2100 Monument Blvd., Pleasant Hill 93542
Herbert A. Holden........Secretary
1436 Clarke St., San Leandro 94577

BUTTE-GLENN Medical Society, P. O. Box 1008, Chico 95927. Meets Fourth Thursday.
Walter S. Mazen........President
1617 Esplanade, Chico 95926
John B. Copeland........Secretary
1423 Magnolia, Chico 95926

FORTY FIRST Medical Society, Suite 821, 1515 N. Vermont Ave., Los Angeles 90027
Paul D. Yates........President
844 Hermosa Ave., Hermosa Beach 90254
Jordan M. Phillips........Secretary
11239 S. Lakewood Blvd., Downey 90241

FRESNO County Medical Society, 1118 Divisadero St., Fresno 93721. Meets Second Tuesday, 6:30 p.m., California Hotel.
Theodore Steinberg........President
155 N. Thesta St., Fresno 93701
Jack Schiff........Secretary
167 N. Thesta St., Fresno 93701

HUMBOLDT-DEL NORTE County Medical Society. Meets Second Thursday.
Halvor J. Braafladt........President
3115 G St., Eureka 95501
Max Todd........Secretary
616-14th St., Arcata 95521

IMPERIAL County Medical Society, P. O. Box 518, Imperial 92251. Meets Second Tuesday, 8 p.m., Pioneer Memorial Hospital, Brawley.
O. G. Fais........President
1745 S. Imperial Ave., El Centro 92243
Elmer C. Werner........Secretary
1745 S. Imperial Ave., El Centro 92243

INYO-MONO County Medical Society. Meets Fourth Tuesday except December, January, February.
Milton R. Jones, Jr........President
Box 38, Lone Pine 93545
Robert W. Denton........Secretary
611 W. Line St., Bishop 93514

KERN County Medical Society, 2603 G Street, Bakersfield 93301. Meets Third Tuesday, 7:30 p.m., Society Office, 2603 G Street, except June, July, August.
Keith W. Spaulding........President
2521 "G" St., Bakersfield 93301
James R. Friend........Secretary
P. O. Box 2087, Bakersfield 93303

KINGS County Medical Society, P. O. Box 1003, Hanford 93230. Meets Second Thursday, 7:00 p.m. Place to be announced.
James Dean........President
912 N. Doury St., Hanford 93230
Willard S. Bridwell........Secretary
1507 N. 11th Ave., Hanford 93230

LASSEN-PLUMAS-MODOC-SIERRA County Medical Society. Meets on call.
James M. McCullough........President
50 N. McDow St., Susanville 96130
Charles W. Brown........Secretary
Western Pacific Hospital, Portola 96122

LOS ANGELES County Medical Assn., 1925 Wilshire Blvd., Los Angeles 90057.
Joseph F. Boyle........President
1136 W. Sixth St., Los Angeles 90017
Homer C. Pheasant........Secretary
2300 S. Hope St., Suite 300,
Los Angeles 90007

MARIN Medical Society, 1601 Second St., Suite 106, San Rafael 94901. Meets First Thursday, 7:00 p.m.
Robert Mills........President
711 D St., San Rafael 94901
William A. Galeno........Secretary
712 D St., San Rafael 94901

MENDOCINO-LAKE County Medical Society.
Frank H. Dailey........President
719 S. Dora, Ukiah 95482
Patrick R. Allanson........Secretary
765 S. Dora St., Ukiah 95482

MERCED-MARIPOSA Medical Society, P. O. Box 586, Merced 95340. Meets Fourth Thursday; meeting place changes.
Patrick J. Maloney........President
P. O. Box 586, Merced 95340
Robert J. Schiffler........Secretary
P. O. Box 586, Merced 95340

MONTEREY County Medical Society, P. O. Box 308, Salinas 93903. Meets First Thursday.
Joseph E. Turner........President
1010 Cass St., Monterey 93940
Thomas S. Elliott........Secretary
101 Maple St., Greenfield 93927

NAPA County Medical Society, P. O. Box 2158, Napa 94559. Meets Second Wednesday.
Richard W. Neil........President
1375 Main St., St. Helena 94574
Victor J. Bouillon........Secretary
3020 Beard Rd., Napa 94558

ORANGE County Medical Association, 300 S. Flower, Orange 92666. Meets First Tuesday, 7:00 p.m.
Wallace A. Gerrie........President
1901 Westcliff Dr., Newport Beach 92660
David I. Nielsen........Secretary
400 Westminster Blvd., Newport Beach 92660

PLACER-NEVADA County Medical Society. Meets Second Wednesday.
F. Harold Johnson........President
305 Oak Ridge Dr., Roseville 95678
Arthur R. Weaver........Secretary
1212 High St., Suite 106, Auburn 95603

RIVERSIDE County Medical Association, 4175 Brockton Ave., Riverside 92501. Meets Second Monday, 7:30 p.m., Spanish Arts Gallery, Mission Inn.
Roger W. Ridley........President
6969 Brockton Ave., Riverside 92506
Richard W. Trotter........Secretary
6876 Magnolia Ave., Riverside 92506

SACRAMENTO County Medical Society, 5380 Elvas Ave., Sacramento 95819. Meets Third Tuesday, 8:30 p.m. Location of meetings varies.
James W. Martin........President
2811 L St., Sacramento 95816
James O. Farley........Secretary
3428 Watt Ave., Sacramento 95821

SAN BENITO County Medical Society. Meets once a month, except July and August. Time and place to be announced.
Robert D. Quinn........President
930 Sunset Dr., Hollister 95023
Roderick L. Yip........Secretary
891 Sunset Dr., Hollister 95023

SAN BERNARDINO MEDICAL SOCIETY, 369 Court St., San Bernardino 92401. Meets First Tuesday, 7:30 p.m., Harold's Club, Fontana.
Richard W. Judd........President
1700 Waterman Ave., San Bernardino 92404
Daniel J. Rothenberger........Secretary
321 Cajon St., Redlands 92373

SAN DIEGO County Medical Society, 3427-4th Ave., San Diego 92103. Meets Second Tuesday. Location of meetings varies.
John Wanless........President
3427 Fourth Ave., San Diego 92103
W. C. Herrick........Secretary
233 A St., San Diego 92101

SAN FRANCISCO Medical Society, 250 Masonic Ave., San Francisco 94118.
Albert G. Clark........President
450 Sutter St., San Francisco 94108
Harry W. Sawyer........Secretary
909 Hyde St., San Francisco 94109

SAN JOAQUIN County Medical Society, 445 W. Acacia St., P. O. Box 230, Stockton 95201. Meets First Thursday, 8:15 p.m.
Thomas Huff........President
512 W. Harding Way, Stockton 95204
Dora A. Lee........Secretary
2420 N. California St., Stockton 95204

SAN LUIS OBISPO County Medical Society, P.O. Box 319, San Luis Obispo 93402. Meets Third Saturday, 7:00 p.m., Madonna Inn, San Luis Obispo 93402.
Robert Buck........President
P. O. Box 319, San Luis Obispo 93402
Billy W. Mounts........Secretary
P. O. Box 319, San Luis Obispo 93402

SAN MATEO County Medical Society, 122 El Camino Real, San Mateo 94401. Meets Third Tuesday.
Stanford B. Rossiter........President
Sequoia Hospital, Redwood City 94602
Eldon E. Ellis........Secretary
2900 Whipple Ave., Redwood City 94602

SANTA BARBARA County Medical Society, 300 W. Pueblo St., Santa Barbara 93105. Meets Second Monday of the month, University Club, 1332 Santa Barbara St., Santa Barbara.
T. K. Hill........President
1421 State St., Santa Barbara 93105
Casimir Domz........Secretary
317 W. Pueblo St., Santa Barbara 93105

SANTA CLARA County Medical Society, 700 Empey Way, San Jose 95128. Meets First Monday.
Gerald Besson........President
700 Empey Way, San Jose 95128
Alexander MacKenzie........Secretary
700 Empey Way, San Jose 95128

SANTA CRUZ County Medical Society. Meets every Second Month, Second Tuesday. Time, place to be announced.
W. W. Hoskins........President
30 Brennan St., Watsonville 95076
William Blade........Secretary
1510 Seabright Ave., Santa Cruz 95062

SHASTA-TRINITY County Medical Society. Meets Second Monday of the month. Place to be announced.
Joseph T. DeWitte........President
1129 Hartnell Ave., Redding 96001
Eugene A. Nutley........Secretary
2625 Edith Ave., Redding 96001

SISKIYOU County Medical Society. Meets Sunday on call.
Robert L. Anderson........President
504 Hillcrest Dr., Yreka 96097
John P. Geyman........Secretary
705 S. A St., Mt. Shasta 96067

SOLANO County Medical Society, 631 Tennessee St., Vallejo 94599.
Tuesday. 8:00 p.m., at different meeting places.
Robert L. Garrett........President
1512 Napa St., Vallejo 94590
Keith E. Vincent........Secretary
2121 Redwood St., Vallejo 94594

SONOMA County Medical Society, 1625 Franklin Ave., Santa Rosa 95404. Meets Second Thursday.
John S. Dunn........President
576 B St., Santa Rosa 95401
Elizabeth Buckley........Secretary
1140 Sonoma Ave., Santa Rosa 95405

STANISLAUS County Medical Society, 1125-13th St., Modesto 95354. Meets Third Tuesday of the month, 7 p.m., Hotel Covell, Modesto.
Howard Kuhl........President
1700 McHenry Ave., Modesto 95350
Jeanne I. Miller........Secretary
709-18th St., Modesto 95354

TEHAMA County Medical Society. Meets at call of President.
W. Scott Davis........President
P. O. Box 118, Los Molinos 96055
M. Blair McGee........Secretary
217 Corona Ave., Corning 96021

TULARE County Medical Society, 1640 West Mineral King. P. O. Box 16, Visalia 93278.
Irving M. Schor........President
P. O. Box 1151, Visalia 93277
George Tiss........Secretary
P. O. Box 7, Visalia 93277

VENTURA County Medical Society, 3291 Loma Vista Rd., Room 40, Ventura 93003. Meets Second Tuesday each month, 7:30 p.m., Los Posas Country Club, Camarillo.
Joseph F. Gstettenbauer........President
3003 Loma Vista Rd., Ventura 93003
Franklin K. Helbling........Secretary
3291 Loma Vista Rd., Room 40, Ventura 93003

YOLO County Medical Society. Meets First Wednesday.
Benjamin R. Robinson........President
650 Third St., Woodland 95695
Corbin Young........Secretary
101 Main St., Winters 95694

YUBA-SUTTER-COLUSA County Medical Society.
John R. Heckman........President
714-4th St., Marysville 95901
Norman K. Noordhoff........Secretary
320 G St., Marysville 95901

Officers — CALIFORNIA PHYSICIANS' SERVICE — 1966-67
BLUE SHIELD

Officers:

Chairman of the Board	Richard S. Wilbur
Vice-Chairman of the Board	Milo A. Youel
Secretary	Wilbur G. Rogers
Treasurer	Bert L. Halter

BOARD OF *Trustees:*

Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Roberta F. Fenlon*	San Francisco
Donald R. Fitch	Glendale
Mr. Thomas Hadfield	San Francisco
Bert L. Halter	San Francisco
Donald C. Harrington	Stockton
M. M. Haskell	Long Beach
Carl Goetsch	Berkeley
Mr. Philip S. Magruder	Flintridge
Gregory C. Murray	Los Angeles
Wilbur G. Rogers*	Glendale
Mr. Paul Schrade	Los Angeles
William H. Thompson	San Mateo
Richard S. Wilbur*	Palo Alto
Milo A. Youel	San Diego

*Appointed by CMA Council

BOARD COMMITTEES:

Executive Committee

Richard S. Wilbur, Chairman	Palo Alto
Carl Goetsch	Berkeley
Bert L. Halter	San Francisco
Mr. Philip S. Magruder	Flintridge
Wilbur G. Rogers	Glendale
William H. Thompson	San Mateo
Milo A. Youel	San Diego

Finance Committee

Bert L. Halter, Chairman	San Francisco
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Donald R. Fitch	Glendale
Mr. Philip S. Magruder	Flintridge

Medical Policy Committee

Wilbur G. Rogers, Chairman	Glendale
Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Roberta F. Fenlon	San Francisco
Carl Goetsch	Berkeley
Mr. Thomas Hadfield	San Francisco
M. M. Haskell	Long Beach
Donald C. Harrington	Stockton
Gregory C. Murray	Los Angeles

Blessed event?

Not entirely, when nausea and vomiting occur in early pregnancy. Emetrol offers prompt and safe relief. Local rather than systemic action provides emesis control on contact with the hyperactive G.I. tract.* In a study of 123 pregnant women, the drug produced measurable improvement in 79% of patients in controlling vomiting.[1]

*As shown by *in vitro* studies.
1. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. *65*:311 (Feb.) 1953.

Emetrol®
phosphorated carbohydrate solution
emesis control

RORER
WILLIAM H. RORER, INC.
Fort Washington, Pa.

DAIRY COUNCIL DIGEST

AN INTERPRETIVE REVIEW OF RECENT NUTRITION RESEARCH PUBLISHED BY
NATIONAL DAIRY COUNCIL, 111 NORTH CANAL STREET, CHICAGO, ILLINOIS 60606

Volume 38, No. 1
January-February, 1967

The Obese Patient: Problem in Management

SUMMARY

A person is generally considered to be obese if he weighs more than twenty percent above the average for his age and height. Obesity is the excessive accumulation of body fat and may be defined by caliper skinfold measurements in the physician's office. Obesity has been associated with several serious diseases as well as with increased morbidity risk. Providing the obese individual is relatively stable emotionally and is motivated to lose weight, diet, exercise, education of the patient and psychological support are the main components which determine the success of a weight reduction program. All other special techniques may be useful in particular circumstances but are not, by themselves, essential for the successful continuation of a weight control program. Childhood and adolescent obesity generally persist into adulthood. It isn't necessarily how long the problem has existed, however, but rather the nature of the problem which is important. In this respect, psychiatric help is often fundamental in successful weight reduction programs.

Obesity is a prevalent health problem, at every age and in both sexes, in the United States today. Obese children and adolescents are a major reservoir for obesity in adult life. They are more likely to remain obese as adults and to have more difficulty in losing fat and maintaining fat loss than people who become obese as adults.(1) Today, almost 3 million adolescents (10%) in the U. S. are seriously ill with overweight.(2) In adults, average body weight increases with age, increasing rapidly in men who are in their twenties and early thirties and most rapidly in women in their forties and fifties.(1) The problem of overweight and obesity and the factors which influence these conditions, as well as an evaluation of programs for weight control, have been reviewed.(3)

The association between obesity and increased mortality and morbidity has been well documented. Insurance statistics reveal that overweight is associated with a death rate much higher than that of lean persons or those of desirable body weight.(4) Diseases, such as gallbladder disease, gout, diabetes mellitus, hypertension and possibly coronary heart disease, are significantly associated with obesity.(5) Obesity increases surgical risk; is a hazard in pregnancy; prevents ambulation and self-care in arthritis, hemiplegia and fractures of the lower extremities in the aged (6); places a greater load on the heart and circulatory system (7); and increases the work of breathing.(8) Obese persons have been reported to have abnormally high values for blood cholesterol, triglycerides, and free fatty acids and for various blood and urinary steroids as well as an impaired carbohydrate tolerance. These, as well as other endocrine and metabolic changes, have been delineated in the obese individual.(1) The psychological and cosmetic effects of obesity are also important and should not be ignored. Physicians and patients alike regard weight reduction of obese individuals as being desirable for health and cosmetic reasons. However, there is no simple "cure" the physician can prescribe which will be effective for all patients.

This Digest may be reproduced in whole or part only with full citation and credit to National Dairy Council

DEFINITION

Obesity is the excessive accumulation of body fat. Obesity is not necessarily related to body weight although excessively overweight individuals generally carry their excess weight as fat. It has been suggested that a person above 10 percent of his desirable body weight (based on height and body build) be considered overweight whereas an individual exceeding 20 percent of his desirable body weight be classified as obese.(3)

CAUSE

Generally the basic cause of obesity is an intake of calories in excess of energy expenditure over a period of time. Obesity is not a disease entity as such but a symptom of some underlying difficulty which may have many causes. Inasmuch as the etiology of obesity is extremely complicated and varies greatly among individuals (9), the condition has been referred to as "the obesities."(10) Genetic (11), physiologic (12,13), psychologic (14-16) and socioeconomic influences (17,18) are other factors which may contribute to the development and perpetuation of the condition. Obesity has been classified as regulatory or metabolic in origin. In regulatory obesity it is believed that the area of the brain regulating food intake is impaired causing the individual to eat more than he requires. Recent studies, however, reveal that obese individuals not only eat less but exercise less than the nonobese.(19) Metabolic obesity, on the other hand, results from an abnormality in the metabolism of fats and carbohydrates. It is possible that in metabolic obesity there is a more efficient conversion of food energy to body fat than in nonobese individuals. Thus, even now after considerable research on various aspects of the problem in many disciplines, the disorder is poorly understood. It is not surprising that no single treatment has been found which results in successful long-term alleviation of obesity.(20)

DIAGNOSIS AND MEASUREMENT

Tables of desirable body weights for men and women published by the Metropolitan Life Insurance Company are widely used as a measure of desirable weight for height and body build.(4) These tables, developed from data obtained only on policy holders, represent the range of body weights associated with lowest mortality. Although weight ranges are given for each of three body frames (small, medium and large), no definitions of frames are provided. Hence, the user of the tables may not characterize frame size in the same way as the authors of the tables. Furthermore, individuals with a well developed musculature may be overweight by these standards without having an excess of body fat. Since excessive adiposity, rather than weight in excess of the desirable range, is the clinician's concern, there is a need for a simple method for measuring body fat on which to base a definition of obesity.(21) Beyond the measurement of height and weight, consideration of the role of constitutional factors in human obesities is commonly neglected. There may well be fundamental relationships between type of body build and obesity. Although obesity may occur in any physical type, it occurs in greatest frequency in certain types as compared to other types. (22) The type of body build may be, in part, responsible for the observed association of underweight and overweight with mortality ratios. Other anthropometric indices (in addition to height and weight) as well as anthroposcopy, skinfold thickness, strength tests and somatotyping, are extremely valuable in describing the body build and its relationship to disease and mortality.(23,24) Precise morphologic indices which reflect changes in body composition are useful to the clinician in studying the etiology and progression of obesity.

Methods for assessing fatness which are reasonably precise and adaptable to medical office practice and mass population surveys should be encouraged. The use of calipers to measure skinfold thickness is such a method and more desirable for these purposes than are height-weight tables. The measurement of skinfold thickness by the physician enables him "to make a rapid diagnosis of obesity, obtain a reasonably accurate estimate of its extent, and follow its changes."(21) Skinfold measurements have been compared with results of other methods of measuring body fat, and have been found to be a good index of subcutaneous (about 50% of the total) and total body fat in young individuals. The Committee on Nutritional Anthropometry of the National Research Council has recommended that the triceps and the subscapular skinfolds are good indexes of an individual's over-all fatness. Seltzer et al (25) reported that the triceps skinfold is the best simple predictor of body density (and hence percentage total body fat) in obese adolescent girls and Heald et al (26) indicated that it is a practical and accurate estimate of adiposity in adolescent boys. Seltzer and Mayer (21) have proposed standards for the lower limits of obesity for Caucasian American males and females, ages 5 to 50, on the basis of triceps skinfold thickness; however, for women other skinfolds may be better indexes of body fatness.

PREVENTION AND TREATMENT

The psychological and clinical effects of obesity stress the importance of prevention rather than treating a depressed overweight individual encompassed in a vicious metabolic cycle favoring fat deposition. The prevention of obesity must be directed to the child and the adolescent, for weight problems during this age group persist into adulthood and become most resistant to treatment.(27) While prevention and treatment of obesity during middle age is important, it appears that successful weight reduction is more likely to be achieved if the obesity developed before 10 or after age 16.(1) Furthermore, drastic weight reduction of the obese adult is an emotional hardship.(28)

With the realization of the complexity of the development of obesity comes appreciation of the fact that weight control programs must be individually planned. An attempt should be made to determine the specific etiologic factors involved in the individual patient. (9, 16,29,30) The medical and dietary histories, size, age, activity and occupation of the patient, as well as other personal and social factors must be taken into consideration. The patient should have ". . . at least a reasonable degree of emotional stability and some motivation meaningful to him to make weight reduction worth the struggle . . ." (31)

A recent survey (32) indicated that of the 504 physicians interviewed, over 83 percent advised patients on weight control. Furthermore, 50 percent of the questions patients asked their physician concerned caloric intake and weight control. It is obvious that planning therapy for attaining and/or maintaining desirable body weight requires a considerable amount of the physician's time as well as a cooperative effort between the patient and physician.(33,34) If the services of a clinic dietitian or nutritionist are available, much of the physician's time may be spared.

Success or failure of a weight reduction program is judged by the long-term results. A program is successful only if the patient reduces his body weight to the level the physician believes is desirable for the particular individual, and maintains his body weight at this level.(20) Given an obese patient who is relatively stable emotionally and who is motivated to lose weight, there are four essential components for long-term success. The first component is DIET. A proper diet is the mainstay of a weight reduction program.(9,29,35-37) A good reducing diet is one on which the patient does not become too hungry. The diet must provide all the necessary nutrients in amounts to meet the needs of the individual except for calories. The caloric level must be limited so as to require the body to utilize fat stores to meet some of the energy requirements, thus effecting loss of body weight (and adipose tissue). The number of calories recommended for a weight reduction program will be determined by the sex, age, size and activity of the patient and by the rate at which the physician decides weight should be lost. In general, a weight loss averaging one to two pounds a week is a desirable rate and may be accomplished on diets furnishing (for ambulatory, busy patients) 1000-1200 calories a day for women and 1400-1500 calories for men.(31, 32,37) Diets containing less than these caloric levels are not tolerated well over a long period of time.

The foods in the diet should be chosen with consideration for the patient's familial and economic status, his usual eating pattern, his tastes, and the capabilities of the person who does the marketing and cooking.(37) A diet planned on these bases is more likely to be accepted and adhered to over a long period of time than is one which is impersonally prescribed. Once again the physician should turn to the paramedical group for dietary assistance.

The diet should provide for the loss of stored fat without reducing the amount of non-fat tissue.(38) There is no evidence that a diet that differs drastically from the normal well-balanced diet in proportions of calories supplied by protein, fat and carbohydrate offers any advantages in achieving this goal. Fad reducing diets appeal to overweight individuals because they offer a quick way to lose weight without much effort. Bizarre dietary approaches to weight reduction drastically limit particular nutrients and generally promote specific foods. The health implications of fad reducing diets recently have been summarized. (39)

A diet that supplies protein, fat and carbohydrate in amounts similar to those of the normal diet, but lower in total calories, can produce satisfactory weight loss, be acceptable over a long period of time, and serve as a basis for dietary education. (40) Although many patients are less hungry and are happier on a diet high in protein and somewhat lower in carbohydrate (Young, C. M., personal communication), the four food group plan (41) is the best guide to use for planning a weight reducing diet. The four food groups--milk, meat, vegetables and fruits, breads and cereals--represent the foundation for a day's adequate meals. The groups are made up of commonly used foods. There is flexibility in the plan. Choices within the group can be made according to individual preferences. Costs can be controlled by the specific selections. Adjustments in the number of servings, and/or amounts, for individuals of different ages, sizes, and activity are easily accomplished. Finally, when desirable body weight is reached, this plan is the patient's guide for a diet for weight maintenance.

The second component for long-term success of a weight reduction program is EXERCISE. Body weight is a matter of balance between energy intake and energy expenditure. (37) Exercise, added to the diet restriction, contributes to loss of body weight and fat. (42) It is also an important part of the program because it promotes nitrogen balance and muscle tone. (12) The physician and patient should plan a program of regular moderate exercise which is appropriate for the individual's physical condition, age, health and living pattern. (43)

The third component is EDUCATION of the patient. The patient must understand that the basic cause of obesity is a faulty balance between dietary intake and energy expenditure over a considerable period of time. It must be realized that permanent adjustments in the pattern of living must be made to achieve and maintain desirable body weight. It will require effort and will power on the part of the patient to adhere to the program. (16,29) It is essential for the patient to learn the calorie content of the various foods in various sized portions. (37) The effect of food preparation on its caloric content must be understood. For example, a medium size apple has 70 calories, 1/2 cup of applesauce has 115 calories, and a piece of apple pie, 345 calories. (44) Basic nutrition education is also important, so that the patient may know why the proper selection of foods--from the standpoint of their nutrient contributions to the diet--is critical.

The patient should be prepared to accept changes in his dietary and exercise patterns as permanent. When his desirable body weight is reached, there will be minor changes in the diet, but he cannot expect to return to his former habits and retain his new weight level. The weight reduction program must re-educate the patient and provide new patterns which will result in proper balance between dietary intake and energy expenditure. (29,37)

The last, but not least, component for the long-term success of a weight reduction program is PSYCHOLOGIC SUPPORT. Obesity is a complex medical problem, not a moral issue. The attitude of the therapist should not be overly stern nor pitying. The patient should not be "scared" into dieting, nor promised unrealistic results. (12) All of his problems are not related to his obesity, although he may have convinced himself that they are. (14) Regardless of the specific therapy, the psychologic support of a genuinely interested and sympathetic physician is a critical factor in the success of the program. (16,20,29,33)

During the early stages of weight reduction, close supervision by the physician or nutritionist is important. Frequent visits permit the physician to measure body weight and fat loss, allow the patient to report problems he has encountered in adhering to the diet and exercise program and provide opportunity for patient education. (29) On the basis of his observations, the physician may make adjustment in the diet and exercise prescription to increase their acceptability and effectiveness. (37)

SPECIAL TECHNIQUES

Special techniques may be found helpful in the early weeks of a weight reduction program to make the limitation of caloric intake more acceptable for some patients. Proper diet, patient education, exercise and psychologic support are fundamental in the management of the obese patient. The following special techniques have several shortcomings and are only adjuncts to the basic concept of managing obesity.

METERED FORMULA DIETS provide exact amounts of nutritionally adequate calories and thus provide a simple rigid regimen which requires no knowledge of foods and food values (37); however, the formula per se is certainly not a panacea for obesity.(45) One of the most important goals in any long-term weight control program is educating the patient in adequate and inadequate dietary practices. Although the formula diet in itself provides no opportunity for retraining food habits, it may be used in the early weeks of a weight reduction program thus providing the physician time in which to instruct the patient concerning proper diet therapy. Constipation is a frequent side effect in patients adhering to a formula diet; diarrhea, however, may also be a problem.(33) The monotony of the formula diet makes it unacceptable to most patients for long-term use.(45)

If the manipulation of the dietary schedule does not prevent the development of excessive hunger, the use of ANOREXIGENIC AGENTS may be indicated. "When prescribing these agents, the physician should understand that he is recommending treatment which is not directed at the major problems in obesity. He must be simultaneously working to achieve understanding and application of principles of planned exercise and limitation of calorie intake in his patient which will bring about the energy deficits with which body fat is burned and body weight reduced. Anorexigenic agents may at best take the hunger pangs out of dieting."(46) Anorexigenic agents can be most effectively used after the hunger-satiety pattern on the reducing diet has been determined, so they may be given to have effect at the time of day that hunger is the greatest. Individuals vary widely in their response to appetite-depressant drugs. In general, effective duration of treatment with anorexigenic agents is from 4 to 6 weeks; however, some patients are not affected by such drugs. Amphetamine therapy is contraindicated for cardiac patients because of the adrenergic effects on the heart (increase in heart rate, potentiation of cardiac arrhythmias). In many patients side effects are observed when anorexigenic agents are used: dry mouth, restlessness, irritability, insomnia and sometimes constipation. It must be made clear to the patient that the use of anorexigenic agents does not decrease the importance of his adherence to the prescribed dietary and exercise program.(37,46,47)

STARVATION may be used as a temporary therapeutic measure in grossly obese patients who have not responded to other methods of treatment. Fasting should be undertaken only in the hospital under the close supervision of a physician. Metabolic responses to fasting should be carefully and regularly evaluated. The initial fasting period of from 7 to 14 days in the hospital provides an opportunity for daily instruction of the patient concerning diet, exercise, drug therapy and psychiatric aspects of obesity. Weight loss during this period ranges from 18 to 36 pounds. Following hospital discharge, a low-calorie diet is prescribed and the patient fasts one day a week until a "near-normal" weight is achieved. The one-day fasts are then less frequent, and finally discontinued as physical exercise and other measures serve to maintain a satisfactory state.(30) Total fasting has been found to be a useful technique in that it demonstrates to the patient that he can lose body weight, that food must be the cause of his obesity and that he has the will power to lose body weight. A sense of well being and an improvement in emotional state have been observed in fasted patients.(30,48,49) Uncontrolled labile diabetes is a contraindication to total fasting. "The fasting regimen may give rise to certain hazards unless safeguards against them are observed. These preventable hazards include (a) diabetic ketosis complicating uncontrolled labile (juvenile-type) diabetes, (b) gout, (c) hyperuricemic nephritis, (d) cardiac complications, such as atrial fibrillation precipitated in patients with chronic cardiovascular disease, by violation of the rule of physical rest during the fast, and (e) sodium depletion syndrome."(30) In obese men fasted for 10 days, body composition studies revealed that the weight lost during this period was about 65 percent due to loss of lean body tissue and only 35 percent due to loss of adipose tissue.(50) In another study, the loss of body weight due to starvation was associated with net losses of large amounts of nitrogen, phosphorus, calcium, potassium, sodium and water. The serum uric acid level increased during starvation. All of these changes were promptly reversed on refeeding the subject with a 600 calorie diet.(51)

Strict SALT RESTRICTION generally is not recommended in weight reduction programs unless clinically indicated. Obese human subjects may have a diminished glomerular filtration rate, an elevated blood titer of the posterior pituitary antidiuretic hormone and a reduced renal clearance of water. Also, a defect in renal clearance of sodium has been demonstrated. An antidiuretic effect occurs when a caloric restriction is imposed, and fluid accumulation takes place during weight reduction. Mild salt restriction, not severe enough to make the food unpalatable, tends to minimize the sodium and fluid retention problem.(52) ". . . cutting down on the salt intake decreases the tendency to excessive fluid retention seen in many reducing obese subjects, particularly middle-aged and older women and sedentary individuals. It may also have some effectiveness in the prevention of hypertension."(37)

In addition to formula diets, the use of anorexigenic agents and total fasting, other special techniques have been utilized in weight control programs. Desiccated thyroid preparations, triiodothyronine, diuretics, bulk-producing agents such as cellulose and methylcellulose, frequent small meals and even extreme measures such as surgery (to bypass the small intestine) have experienced very limited success in weight control programs.

BIBLIOGRAPHY

1. Mayer, J. and S. M. Fox (eds). Obesity and Health, U. S. Department of Health, Education, and Welfare, Public Health Service, November 1, 1966, 77 pp.
2. Spargo, J. A., F. Heald, and P. S. Peckos. Nutr. Today 1:2, 1966.
3. Anonymous. Weight Control Source Book, National Dairy Council, Chicago, 1966, 19 pp.
4. Overweight: Its Prevention and Significance. A series of articles reprinted from Statistical Bulletin. Metropolitan Life Insurance Company, 1960.
5. Olson, R. E. J. New York Med. Coll. 1:46, 1959.
6. Mayer, J. Postgrad. Med. 32:394, 1962.
7. Best, C. H. and N. B. Taylor (eds). The Physiological Basis of Medical Practice, 7th Ed., Baltimore: The Williams and Wilkins Co., 1961, p. 865-868.
8. Sharp, J. T., J. P. Henry, S. K. Sweaney, W. R. Meadows, and R. J. Pietra. J. Clin. Invest. 43:728, 1964.
9. Young, C. M. J. Amer. Diet. Assoc. 45:134, 1964.
10. Hilleboe, H. E. Health News 39:3, 1962.
11. Mayer, J. Postgrad Med. 37:A-103, 1965.
12. Mayer, J. Nutr. Abstr. Rev. 25:871, 1955.
13. Mayer, J., L. F. Monello, and C. C. Seltzer. Postgrad. Med. 37:A-97, 1965.
14. Bruch, H. Borden's Rev. Nutr. Res. 19:57, 1958.
15. Bruch, H. The Importance of Overweight, New York: W. W. Norton and Co., 1957, p. 267-297.
16. Brosin, H. W. J. Amer. Med. Assoc. 155:1238, 1954.
17. Goldblatt, P. B., M. E. Moore, and A. J. Stunkard. J. Amer. Med. Assoc. 192:1039, 1965.
18. Moore, M. E., A. J. Stunkard, and L. Srole. J. Amer. Med. Assoc. 181:962, 1962.
19. Stefanik, P. A., F. P. Heald, and J. Mayer. Amer. J. Clin. Nutr. 7:55, 1959.
20. Feinstein, A. R. J. Chronic Dis. 11:349, 1960.
21. Seltzer, C. C. and J. Mayer. Postgrad. Med. 38:A-101, 1965.
22. Seltzer, C. C. and J. Mayer. J. Amer. Med. Assoc. 189:677, 1964.
23. Seltzer, C. C. New Eng. J. Med. 274:254, 1966.
24. Seltzer, C. C. and J. Mayer. Postgrad. Med. 40:A-107 and A-145, 1966.
25. Seltzer, C. C., R. F. Goldman, and J. Mayer. Pediat. 36:212, 1965.
26. Heald, F. P., E. E. Hunt, Jr., R. Schwartz, C. D. Cook, O. Elliot, and B. Vajda. Pediat. 31:226, 1963.
27. Abraham, S. and M. Nordsieck. Pub. Health Reports 75:263, 1960.
28. Kubland, H. D. Time 89:46, 1967.
29. Young, C. M. J. Amer. Med. Assoc. 186:903, 1963.
30. Duncan, G. G., M. A. Hunscher, F. C. Christofori, T. G. Duncan, and G. L. Schless. Postgrad. Med. 38:523, 1965.
31. Young, C. M. Amer. J. Clin. Nutr. 8:896, 1960.
32. Anonymous. Physician's Attitudes and Practices Concerning Heart Disease, Weight Control and Diet, National Dairy Council, Chicago, 1966, 12 pp.
33. Young, C. M. Amer. J. Clin. Nutr. 8:523, 1960.
34. Roberts, H. J. Amer. J. Clin. Nutr. 8:817, 1960.
35. Stare, F. J. World-Wide Abstr. Gen. Med. 6:8, 1963.
36. Strang, J. M. "Obesity" In: Diseases of Metabolism, 5th Ed., G. G. Duncan (ed), Philadelphia: W. B. Saunders Co., 1964, p. 693-804.
37. Mayer, J. Postgrad. Med. 38:A-133, 1965.
38. Keeton, R. W. and D. Dickson. Arch. Int. Med. 51:890, 1933.
39. Anonymous. Dairy Council Digest 37:7, 1966.
40. The Regulation of Dietary Fat, Council on Foods and Nutrition, J. Amer. Med. Assoc. 181:411, 1962.
41. Food For Fitness--A Dairy Food Guide. Leaflet 424, U. S. Department of Agriculture, 1964.
42. Buskirk, E. R., R. H. Thompson, L. Lutwak, and G. D. Whedon. Ann. N. Y. Acad. Sci. 110:918, 1963.
43. Mayer, J. Postgrad. Med. 25:325, 1959.
44. Nutritive Value of Foods. Home and Garden, Bulletin 72, U. S. Department of Agriculture, 1964.
45. Formula Diets and Weight Control, Council on Foods and Nutrition, J. Amer. Med. Assoc. 176:439, 1961.
46. Shank, R. E. Nutr. Rev. 19:289, 1961.
47. Combined Staff Clinic. Amer. J. Med. 19:111, 1955.
48. Bloom, W. J. Amer. Med. Assoc. 186: Suppl. 43, 1963.
49. Spencer, H. J. Amer. Med. Assoc. 186: Suppl. 43, 1963.
50. Benoit, F. L., R. L. Martin, and R. H. Watten. Ann. Int. Med. 63:604, 1965.
51. Spencer, H., I. Lewin, J. Samachson, and J. Laszlo. Amer. J. Med. 40:27, 1966.
52. Gordon, E. S., M. Goldberg, and G. J. Chosy. J. Amer. Med. Assoc. 186:156, 1963.

Because of the scope and importance of this subject, WORTH NOTING has been omitted from this issue of Dairy Council Digest.

BOOKS RECEIVED

Books received by CALIFORNIA MEDICINE *are acknowledged in this column. Selections will be made for more extensive review in the interest of readers as space permits.*

ABORTION: LEGAL AND ILLEGAL—A Dialogue Between Attorneys and Psychiatrists—Edited by Jerome M. Kummer, M.D., Associate Clinical Professor of Psychiatry, University of California, Los Angeles, School of Medicine. Published by Jerome M. Kummer, M.D., Post Office Drawer 769, Santa Monica 90406, 1967. 63 pages, $2.95.

CIBA FOUNDATION SYMPOSIUM—Ethics in Medical Progress: With Special Reference to Transplantation—Edited by G. E. W. Wolstenholme, O.B.E., M.A., and Maeve O'Connor, B.A. Little, Brown and Company, Boston, Mass. 02106, 1966. 257 pages, $11.75.

CIBA FOUNDATION SYMPOSIUM—Principles of Biomolecular Organization—Edited by G. E. W. Wolstenholme, O.B.E., M.A., and Maeve O'Connor, B.A. Little, Brown and Company, Boston, Mass. 02106, 1966. 491 pages, $15.00.

COMPREHENSIVE MENTAL HEALTH SERVICES FOR THE DEAF—By John D. Rainier, M.D., and Kenneth Z. Altshuler, M.D. Published by Department of Medical Genetics, New State Psychiatric Institute, Columbia University, 722 West 168th Street, New York, N.Y. 10032 1966. 191 pages. Price: Distributed to interested agencies and individuals upon request; mailing charge $1.00.

DIFFERENTIAL DIAGNOSIS—A Memory Stimulus System for Pocket, Punchcard and Computer—By Stanley B. Berkowitz, M.D., Diplomate, American Board of Surgery, Adjunct in Surgery, Mt. Zion Hospital and Medical Center, San Francisco. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1967. 333 pages, $11.50.

DIFFERENTIAL DIAGNOSIS — Second Edition — By William C. Matousek, M.D., Chief of Staff, Veterans Administration Center, Wood (Milwaukee), Wisconsin. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago, Illinois 60601, 1967. 351 pages, $8.50.

DOCUMENTARY HISTORY OF PSYCHIATRY — A Source Book on Historical Principles—By Charles E. Goshen, M.D. Philosophical Library, Inc., 15 East 40th Street, New York, N.Y. 10016, 1967. 904 pages, $20.00.

JAUNDICE DURING PREGNANCY—With special emphasis on recurrent Jaundice during Pregnancy and its differential diagnosis—By Urs Peter Haemmerli. Springer-Verlag New York, Inc., 175 Fifth Avenue, New York, N.Y. 10010, 1967. 111 pages, $3.80.

MEDICAL DEPARTMENT, UNITED STATES ARMY—RADIOLOGY IN WORLD WAR II—Prepared and published under the direction of Lieutenant General Leonard D. Heaton, The Surgeon General, United States Army. Editor in Chief, Colonel Arnold Lorentz Ahnfeldt, MC, USA; Editor for Radiology, Kenneth D. A. Allen, M.D.; Associate Editors, Elizabeth M. McFetridge, M.A., and Mindell W. Stein, B.Sc. Office of the Surgeon General, Department of the Army, Washington, D.C., 1966. For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C., 20402, at $8.25 per copy. 1,087 pages.

THE MEDICAL STAFF IN THE MODERN HOSPITAL—Edited by C. Wesley Eisele, M.D., Associate Professor of Medicine, Associate Dean for Postgraduate Medical Education, University of Colorado School of Medicine. The Blakiston Division, McGraw-Hill Book Company, 330 West 42nd Street, New York, N.Y. 10036, 1967. 523 pages, $15.95.

MEDICINE AT THE PARIS HOSPITAL, 1794-1848—By Erwin H. Ackerknecht, M.D. The Johns Hopkins Press, Baltimore, Maryland 21218, 1967. 242 pages, $8.95.

OBSTETRIC ANALGESIA AND ANESTHESIA—By Charles E. Flowers, Jr., M.D., F.A.A.O.G., F.A.C.O.G., F.A.C.S., F.I.C.A., Professor and Chairman, Department of Obstetrics and Gynecology, Baylor University College of Medicine, Texas Medical Center, Houston; Chairman, Committee on Obstetric Analgesia and Anesthesia, American College of Obstetricians and Gynecologists, 1965-1966. Hoeber Medical Division, Harper & Row, Publishers, New York, Evanston, and London, 1967. 240 pages, $8.00.

OSLER'S TEXTBOOK REVISITED—Reprint of selected sections with commentaries—Edited by A. McGehee Harvey, Professor of Medicine and Chairman, Department of Medicine, and Victor A. McKusick, Professor of Medicine, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland. Appleton-Century-Crofts, New York (Division of Meredith Publishing Company), 1967. 361 pages, $6.75 (Paperback).

RENAL CARCINOMA—By James L. Bennington, M.D., Director of Pathology, King County Hospital, Seattle; and Assistant Professor of Pathology, University of Washington, School of Medicine; and Robert M. Kradjian, M.D., Staff Surgeon, Kaiser Foundation Hospital, Oakland, California; Consulting Surgeon, Highland Alameda Hospital, Oakland. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105. 1967. 263 pages, illustrated, $15.00.

TOWARD IMPROVED LEARNING — A Collection of Significant Reprints for the Medical Educator—Compiled by the Public Health Service Audiovisual Facility. U.S. Department of Health, Education, and Welfare, Public Health Service, Bureau of Disease Prevention and Environmental Control, National Communicable Disease Center, Atlanta, Georgia 30333, 1967. 417 pages, no price.

Your public relations problem has been our prime consideration in collection procedures during two generations of ethical service to the Medical Profession.

✶

THE DOCTORS BUSINESS BUREAU

Since 1916

821 MARKET ST., SAN FRANCISCO 94103
GArfield 1-0460

For topical treatment of DENUDED and PAINFUL SKIN LESIONS

Anti-Pyrexol antiseptic ointment reduces pain, minimizes scarring, aids healing of burns, sunburn, scalds, lesions, wounds, and local inflammation of skin and mucous membrane. Sold through surgical supply houses. 1, 5, 10 and 50 lb. tins. Time tested—professionally since 1921. Active ingredients: Oils of spearmint, bay, wintergreen (syn.), salicylic acid, lanolin, zinc oxide, phenol .44%, ortho-hydroxyphenyl-mercuric chloride .056%, petrolatum, paraffin.

Anti-Pyrexol Benzocaine. Acutely anesthetic. Contains Benzocaine 3%. 1, 5 and 10 lb. tins.

EASY SPREADING
Anti-Pyrexol
KIP, INC.—LOS ANGELES 21

STACEY'S for any Medical Book in Print
Retail Stores in Palo Alto and San Francisco

NEW BOOK

YEAR BOOK OF MEDICINE, 1966-67. Edited by Paul B. Beeson, M.D., et al. 826 pages. Illustrated. 1966. Year Book. $9.50.

Stacey's
326-9955
2575 HANOVER STREET
PALO ALTO, CALIFORNIA 94304

Please send me on approval:__________

Name__________
Street__________
City__________ State__________

in alcoholism:

B and C vitamins aid therapy. Therapeutic amounts of B and C vitamins can be important in the management of the alcoholic patient. In alcoholism, as in many chronic illnesses, STRESSCAPS vitamins aid therapy.

Stresscaps®

Stress Formula Vitamins Lederle

FOR PROFESSIONAL RECOMMENDATION ONLY

Each capsule contains:

Vitamin B_1 (as Thiamine Mononitrate)	10 mg
Vitamin B_2 (Riboflavin)	10 mg
Vitamin B_6 (Pyridoxine HCl)	2 mg
Vitamin B_{12} Crystalline	4 mcgm
Vitamin C (Ascorbic Acid)	300 mg
Niacinamide	100 mg
Calcium Pantothenate	20 mg

Recommended intake: Adults, 1 capsule daily, for the treatment of vitamin deficiencies. Supplied in decorative "reminder" jars of 30 and 100; bottles of 500.

LEDERLE LABORATORIES, A Division of American Cyanamid Company, Pearl River, New York

691—6—3942

STRESSCAPS

AMA Journal's Fourth Annual Book Issue

The physician who writes as well as heals has long been a feature of the literary world.

Doctors have often recorded their own experiences and observations, and as literate men with wide-ranging interests, they have often excelled at setting down the real or imaginary experiences of others. The list of physician-novelists, for instance, is a long one.

To recognize the doctors' interest and achievement in literature, the *Journal of the American Medical Association* has founded a tradition of its own—an annual issue devoted to the relations of medicine and letters, in the broad sense. The *Journal's* fourth annual Book Issue was published April 3.

Among its 15 articles, this year's issue features a study of Civil War books by physicians, a review of Sigmund Freud's hundreds of letters to friends, best-selling author-physician Frank G. Slaughter's account of his decision to be a writer instead of surgeon, and a survey of a kind of book that was unusually popular in early day America, the do-it-yourself home medical adviser. There also are scholarly articles on books, plays, and libraries, and some lighthearted satire and parodies of medical life.

Editor of the Book Issue is Lester King, M.D., a senior editor of the *Journal.*

Physicians of the Civil War faced enormous medical problems, but made significant contributions of medical history, notes Paul E. Steiner, M.D., Ph.D., in the lead article entitled, "Patriotic Gore."

From the writings of physicians, Dr. Steiner notes much can be learned about the war, and about diseases and their effects upon large, close-living groups. The records of Civil War physicians help give a clearer understanding of how disease and disability impaired some military leaders and decimated their armies.

The Civil War emphasized the importance of prompt medical care, but physicians continued to be scarce during the 19th Century in this huge, lightly populated country. Physicians had been few and far between since America's earliest days, and their scarcity helped the popularity of the "doctor book," the home medical adviser.

It's impossible to trace the origin of these books, but the 17th, 18th, and 19th Centuries witnessed increasing numbers of such publications, said Lester King, M.D.

"Today, books of this type emphasize the professional activity of the physician," Dr. King said. They provide the layman with background material to explain what the doctors are doing, or describe what to do until the doctor comes. For some conditions, they may even explain how the layman might treat himself while calling a physician.

In the 19th Century, this was not necessarily the case, Dr. King said. The quality and quantity of physicians varied widely, and so did the quantity and quality of medical books.

When the do-it-yourself volumes are examined, "the reader must keep in mind the profound differences in cultural, economic, and social levels in various parts of the country at that time," Dr. King said. The home medical advisers reflect social as well as medical history.

The medical adviser books present an opportunity to study the sociology of medicine, Dr. King pointed out. They reflect what the public, and to some extent, the medical community, thought important in the classification, description, and cure of disease.

There were few doctors in the American colonies, partly because conditions here weren't the best, and partly because the European physician or surgeon usually was a well-educated man from the upper levels of society—he didn't want to practice among immigrants, writes John Duffy, Ph.D., of the Department of the History of Medicine, Tulane University, New Orleans.

The vacuum in medical care was filled by an assortment of practitioners, many of them quacks. For many years there were only a few American medical schools. The result was public distrust and contempt for many physicians, a condition from which medicine was a long time recovering.

The 19th Century was equally as bad. An expanding, thinly populated country was not a good place to organize well controlled medical education and practice. Quacks and quack medical schools flourished.

"Chemistry, physics, biology and the other sciences were making remarkable strides, but medicine was still preoccupied with theorizing," Dr. Duffy said. "Among the more thoughtful physicians, old theories were denounced and new ones hotly debated, but the average practitioner continued to bleed and purge, blister and vomit, much as his predecessors had done for generations.

"To the public, it seemed that the current medical theories, instead of determining medical practice, were designed to justify the existing forms of therapy."

By the early 20th Century, the American medical profession had established a strong, effective organization; the level of medical education had moved up sharply, and the lag between medicine and the other scientific and technological areas had disappeared.

"The position of the American physician had never been so high. To appreciate the discoveries in physics and chemistry requires a high degree of sophistication, but the seeming miracles performed

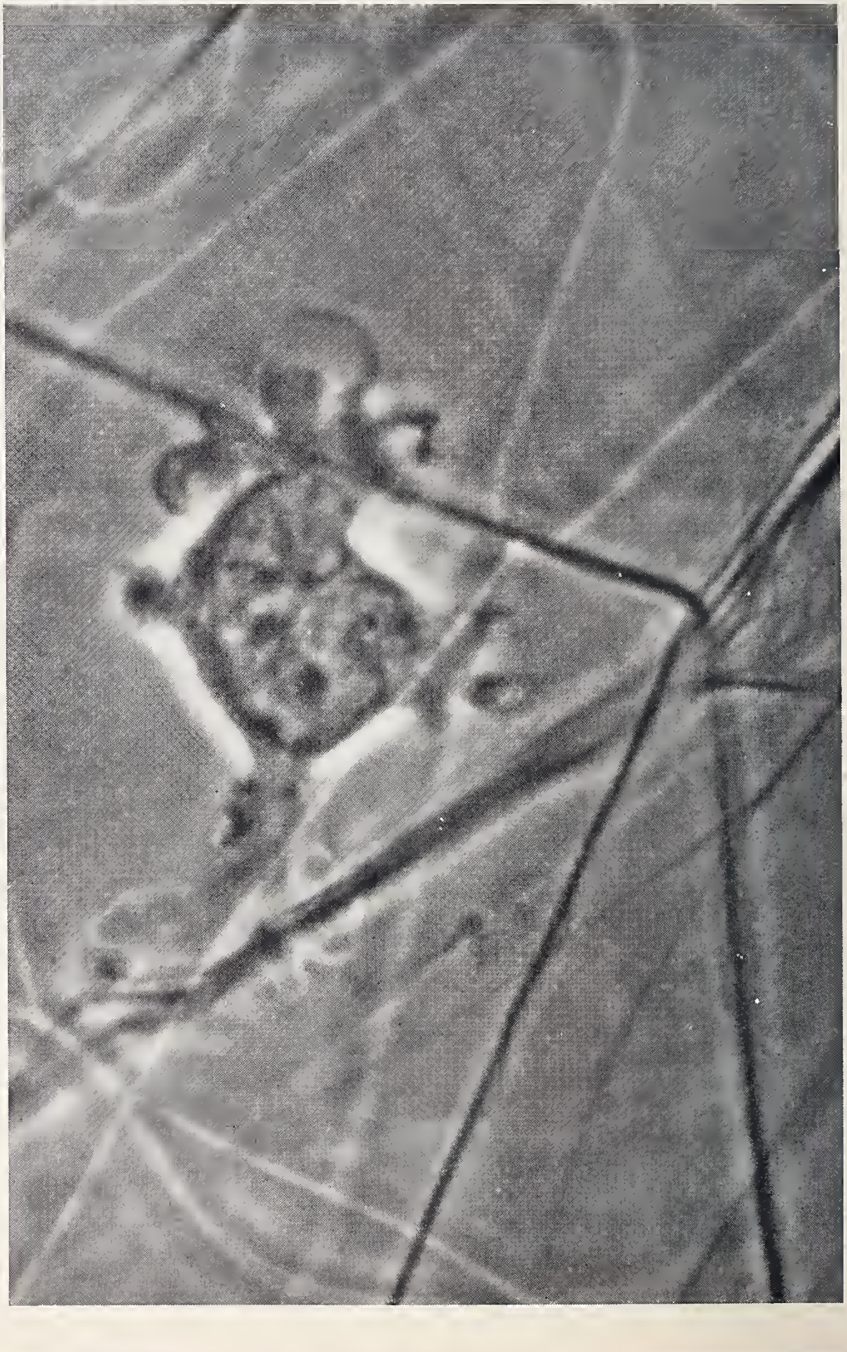
(Continued on Page 43)

INFLAMMATION:
A cellular fight for life

A SYNTEX REPORT based on recently developed hypotheses about topical corticosteroids, including the cellular theories of inflammation by Thomas F. Dougherty, Ph.D., University of Utah.

You are looking at a fibroblast fighting for life. This cell—one of the most common found in connective tissue—has literally been poisoned by cytotoxins released from other cells that have ruptured. Soon, if the abnormal activity of this fibroblast does not cease, it, too, will rupture and die—one more casualty in the inflammatory wave of destruction precipitated by injury.

Until a short time ago no one had ever witnessed such a scene at the cellular level. Now, through advanced cinemicrographic techniques, it is possible to view and photograph the inflammatory process as produced experimentally in living animal tissue. This method permits new insight into the mechanism of inflammation and the role of corticosteroids in therapeutic management. Equally important, these techniques shed new light on factors that may make one corticosteroid more effective than another—factors that can be correlated with other chemical, biologic, and clinical parameters.



in many types of otitis externa—of varying etiology—as part of the treatment regimen

comprehensive

Coly▲Mycin® S Otic

with Neomycin and Hydrocortisone

Each ml. provides:

Colistin base activity (as the sulfate) 3.0 mg.
Neomycin base activity (as the sulfate) 3.3 mg.
for complementary broad-spectrum bactericidal action

Hydrocortisone acetate
10.0 mg. (1%)
for anti-inflammatory/antipruritic benefits

Thonzonium bromide
0.5 mg. (0.05%)
for more rapid dispersion and penetration

It also contains polysorbate 80, acetic acid and sodium acetate in a buffered (pH 5) aqueous vehicle and thimerosal 0.002% as a preservative.
Buffered to pH 5 to reduce the abnormal alkaline pH which may be favorable to bacterial growth, and to help restore the normal acid pH of the ear canal.[8,9]
combats the bacterial cause
relieves the dermatologic manifestations
Recommended Dosage: Therapeutic—4 drops t.i.d. Prophylactic—4 drops before and after swimming.
Contraindication: A history of sensitivity to any of the components, or in tubercular, fungal and most viral lesions; especially herpes simplex, vaccinia and varicella.
Precautions: If sensitivity or irritation occurs, medication should be discontinued promptly. Overgrowth of resistant organisms is possible. Use with care in cases with perforated eardrum or in long-standing otitis media because of the possibility of ototoxicity caused by neomycin. Increased skin sensitivity to neomycin has been noted in current medical literature.
Adverse Reactions: A low incidence of mild burning or painful sensation in the ear has been reported. Such local effects do not usually require discontinuance of medication. Sensitivity reactions were reported in a few instances.
Full information is available on request.
References: 1. McLaurin, J. W.: J.A.M.A. *154*: 207, 1954. 2. Sulzberger, M. B., *et al.*: Dermatology: Diagnosis and Treatment, ed. 2, Chicago, The Year Book Publishers, Inc., 1961, p. 595. 3. Stewart, J. P.: J. Laryng. & Otol. *65*:24, 1951. 4. Elia, J. C.: Southwestern Med. *42*:405, 1961. 5. McLaurin, J. W.; Raggio, T. P., and Simmons, M.: Laryngoscope *75*:1699, 1965. 6. Mann, P. H.: Current Therap. Res. 6:101, 1964. 7. Saltzman, M.: Clin. Med. *70*:559, 1963. 8. Ettenger, M. S.: Pennsylvania M. J.: 66:35, 1963. 9. Pullen, F. W. II: Eye Ear Nose & Throat Month. *42*: 38, 1963. 10. Jones, E. H., and McLain, P. G.: Eye Ear Nose & Throat Month. *43*:52, 1964.

WARNER-CHILCOTT
CO-GP-72 Morris Plains, N.J.

AMA Journal's Fourth Annual Book Issue

(Continued from Page 23)

by medicine and surgery in the 20th Century were apparent to all," Dr. Duffy said.

Dr. Duffy had a closing warning, however. "Since mankind often reacts better to adversity than to success, the new status of the American physician carries with it a heavy burden of responsibility. Economic success, a frequent corollary of high status, often engenders complacency and in many subtle ways influences moral judgments.

"For the past 100 years, American physicians have demonstrated an ability to live up to their professional obligations. Their status in American society today requires an even greater obligation to live up to their responsibilities."

Today's Health Guide Into Third Printing

Today's Health Guide, the American Medical Association's fast-selling manual of health information, has gone into its third printing, and is now available in book stores.

Still priced at $5.95, the 640-page book is a compendium of information designed to help the family make the best and most economical use of health services. More than 250,000 copies have been sold.

Today's Health Guide has 90 chapters, each dealing with an important aspect of health in the family. It is illustrated by hundreds of easy-to-understand drawings, including "trans-vision," or full color "see-through" drawings, of the organs and systems of the human body.

This is not an old-fashioned "doctor book." A non-profit project of the AMA, the health guide is designed to furnish the latest information at a time when medical knowledge is increasing rapidly.

The guide is the product of many doctors and allied scientists. More than 200 people had a role in its preparation—practicing physicians and specialists, dentists, veterinarians, clergymen, chemists, physicists, nurses, educators, engineers, safety experts, writers, reviewers, and members of the AMA staff.

Possessing and reading a book won't enable a person to bring modern medical procedures into the home, AMA spokesmen stress. The book helps the homemaker create an atmosphere, a "climate" in the home in which health will be favored, disease discouraged, and life prolonged and enriched.

Today's Health Guide is available through your bookseller or by mail order from the American Medical Association, 535 North Dearborn St., Chicago, Illinois 60610, for $5.95. Check or money order should accompany each order.

now he can cope...

thanks to

Butisol SODIUM®

(SODIUM BUTABARBITAL)

the anxiety-allaying agent for everyday situational stress

When stress is situational—environmental pressure, worry over illness—the treatment often calls for an anxiety-allaying agent which has a prompt and predictable calming action and is remarkably well tolerated. BUTISOL SODIUM (sodium butabarbital) meets this therapeutic need. After 30 years of clinical use . . . still a first choice among many physicians for dependability, safety, and economy in mild to moderate anxiety.

Cautions and side effects: Contraindicated in patients with porphyria or in those sensitive to barbiturates. To be used with caution in moderate to severe hepatic disease. Drowsiness at daytime sedative dose levels, skin rashes, "hangover" and systemic disturbances are seldom seen. (Warning: May be habit forming.)

Usual Adult Dosage: As a daytime sedative, 15 mg. (¼ gr.) to 30 mg. (½ gr.) t.i.d. or q.i.d.

Available: Tablets, 15 mg. (¼ gr.), 30 mg. (½ gr.), 50 mg. (¾ gr.), 100 mg. (1½ gr.); Elixir, 30 mg. per 5 cc. (alcohol 7%). BUTICAPS® [Capsules BUTISOL SODIUM (sodium butabarbital)] 15 mg. (¼ gr.), 30 mg. (½ gr.), 50 mg. (¾ gr.), 100 mg. (1½ gr.).

McNEIL

McNeil Laboratories, Inc., Fort Washington, Pa.

California
Medicine

A Description of the Role of the University in Modern Society Together with Encouragement to the Medical School to Turn Its Flirtation with the University into a Full Blown Romance

CHARLES E. ODEGAARD, PRESIDENT,
UNIVERSITY OF WASHINGTON, *Seattle*

President Odegaard here presents some stimulating thoughts for medical educators and for other members of the medical profession. Although a historian by profession, he has considerable familiarity with medical matters from his membership in the Citizens Commission on Graduate Medical Education (Millis Commission), the President's National Advisory Commission on Health Manpower and the Natonal Advisory Health Council.

ONLY AN EIGHTEENTH century title reminiscent of the English novelist Fielding could do justice to the gargantuan task I am here assuming; namely, to describe the role of the American university in modern society to an audience which has a special interest in the relation of the university to medical education. I will have to skitter over my subject like a water bug on the surface of a pond, but I hope that I may provide some suggestions for the subsequent discussion.

To turn first to the American university, no book on this subject in recent years has been more used and abused than Clark Kerr's *The Uses of the University*. I am sure that the author himself must often be surprised by the meanings which some have found in his words. Without entering into these debates let me call attention to one affirmation that is not much debated, the recognition that the American university is a multipurpose, multi-faceted institution—especially in relation to what it used to be in America and, for that matter, in relation to what a university now generally is in Europe. Dr. Kerr will probably regret to his dying day that he coined the term *multiversity* to suggest this undisputed feature of the American university. But multiversity, the American university surely is. It would be the obvious thing to turn immediately to the array of human activities and natural phenomena which are the object of attention of that collection of faculty members who constitute an American university. But before doing that, I want to suggest to you that in a still more profound sense the American university has embraced a multiplicity of purposes which indeed help to explain why it has come to embrace so many areas of interest and instruction.

Let me suggest this range of interests by turning to an analysis of educational objectives which comes from Sir Michael Sadler. While studying English schools at the turn of the twentieth century, he undertook a comparative survey of educational institutions on the European continent and in the United States. Writing in 1902, he said that representatives of different countries tended to ask somewhat different questions about students and that these questions reflected different perceptions of the objectives of education in their respective countries. He said that the German asks of the student, what does he know? The American asks, what can he do? The Englishman asks, what kind of fellow is he? And the Frenchman asks, what examinations has he passed?

I have no wish to defend to the death the complete appropriateness of Sadler's categories for the several nations. I shall assume that there is some measure of truth in his generalization, and go on to

---

Presented at the Boerhaave Conference, University of Leiden, 15 to 17 December 1966. Printed with permission of the University of Leiden.

Submitted 28 February 1967.

Reprint requests to: Office of the President, University of Washington, Seattle 98105.

use them in my own fashion by saying that the American university has entered into the middle and late twentieth century in a manner to suggest that its faculty, at least collectively, has come to ask itself about its student three out of four of these questions. We do not ask the Frenchman's question, what examinations has he passed, since we have made little effort to arrive at widely used standard examinations. We have not exemplified passion for a highly organized, consistent and public examination system. Instead we muddle along with a high diffusion of responsibility for setting standards of accomplishment among a multitude of institutions which are coordinated only to the degree that we succeed in setting similar standards among some institutions through the indirect influence of private professional associations. It is no wonder then that foreigners have so hard a time keeping track of our fluid prestige system for universities and university graduates. But I am not here to elucidate that subject, so I shall pass on to another question.

The Humanistic Thread

Interest in the English question, what kind of fellow is he, was expressed in our oldest university tradition, that which goes back to the colonial foundations like Harvard and Yale. These English colonial institutions and the many colleges founded in imitation of them in the nineteenth century were American derivatives of Cambridge and Oxford in their sleepier seventeenth and eighteenth century days. Their curriculum was largely based on the ancient classics, and the newer science was generally neglected in their academic halls until the later nineteenth century. Their pedagogical emphasis was humanistic, if not always humane, but at their best they produced literate frontiersmen who escaped the trap of parochialism and who acquired a breadth of view of man and society which made them apostles of a universal version of the principles of the American Revolution which has proved to be exportable even to this day to the ends of the earth.

This humanistic thread, this interest in the quality of being human, this concern with what manner of man will the student become, has passed through periods of lowered visibility in our institutions only to reassert itself periodically in the twentieth century through the guilty conscience of the university faculty; this uneasiness is often expressed implicitly in a peculiarly American preoccupation with the subject of "teaching," by which is meant especially the human quality of the teaching of undergraduates. In the 1930's it appeared in the effort to recapture so-called "liberal education." It arose again in the struggle in the post World War II years to restructure the curriculum in the name of "general education," as exemplified by the Harvard "red book." Latterly at Berkeley, in the Muscatine Report, a troubled faculty is responding to student dissidents, a portion of whose complaints stem from alleged university neglect of teaching about human values and problems of human action; and there are currently echoes of the same concern on many other American university campuses.

In sum, the teaching of morals or about morals in action may be an untidy matter in intellectual communities now heavily influenced by modern positivism, but commitment to this humanistic objective in teaching continues to haunt the American university scene. Thus this English inheritance, this interest in the quality of being, perseveres to join with other motivations to complicate the objectives of the modern American university.

Pure Knowledge and Applied Knowledge

The German commitment to the objective search for knowledge exemplified by the aims and methodology of modern scientific research took root in a number of what were to become influential American institutions in the later nineteenth century and flowered in faculty competence to offer respected, research-based graduate Ph.D. programs in a substantial number of American universities in the twentieth century. I need not underline the zeal with which American universities have embraced the pursuit of new knowledge and made the advancement of objective science a cardinal aim of the institution.

But even while the American university was grafting to its original English humanistic root the stem of Germanic pure science and objective scholarship, it was also adopting a pragmatic attitude, an apparently typically American practical concern with how to do things which has led to an effort to apply knowledge to an ever widening range of human activities.

Until recently, to many European university men, the willingness of the American university to use its energy for practical ends, to mix pure knowledge with applied knowledge or technology, has seemed *infra dig* and incomprehensible. Let me set aside for the moment the question of *dignitas* of the American university to turn to a brief explanation of how an elite institution like the earlier college was led to stoop to such extremes of practicality. The American university, not without soul searching that still goes on, simply responded in the later nineteenth century to some peculiar but obviously pressing needs in its own American setting. Permit me to explain.

In the nineteenth century vast areas of America previously but thinly populated by an uncivilized indigenous people were subjected to human settlement by preeminently European immigrants and their offspring. These areas varied greatly in climatic and geological and geographical conditions, and the physical circumstances faced by the incoming population were often very different from those they had left behind in their homes in the old country. In the long settled areas of their original homeland the peculiarities of the European terrain, its possibilities for human use as well as its hazards, had been subjected to observation by generations of men. The trial and error experienced by their forefathers, usually without more sophisticated analysis by university men, had suggested workable methods of use and control, and had built up a viable tradition by which an acceptable mastery of the practical circumstances of life could be passed on easily, almost unconsciously in place after place by inheritance from father to son. But the terrain of the United States had been very sparsely populated by man; much of it was a wilderness to man, and even where the indigenous population had settlements, their methods were primitive to immigrants molded culturally by a European background to have quite different expectations of life. An enormous task of conversion of desert, prairie, woodland and forest to civilized human use confronted these American settlers. They rarely inherited appropriate engineering works accomplished by earlier generations. Instead they faced *de novo* vast needs in clearing, drainage, terracing, bridging, and road building. The land often could offer great possibilities in return for a man's labor; but without rapidly acquired knowledge of its peculiarities and skill in its manipulation, it also offered the threat of impoverishment, of waste of effort, even of starvation and death itself.

The townsmen and city folk of nineteenth century America likewise found themselves in need of new knowledge and new skills by no means readily inherited from a previous generation or from the old country. They were far removed geographically from the crafts and industry of Europe. Labor in the United States, relatively speaking, was scarce. To provide the many tools and the abundant stock of materials needed for building new towns and cities in a wilderness, a premium had to be placed on increased skill in production. There was a pressing practical demand for knowledge made useful for industry. To prosper in such a new land men in city, town and countryside needed an abundance of relevant knowledge often not brought directly from the old country and skill in procedures and methods often foreign to their previous experience. The possibility of applying organized knowledge to these challenges was too obvious to escape the notice of imaginative men.

This demand for practical help expressed itself in many ways, but it was symbolized dramatically in the Morrill Act of 1862 which made a grant of Federal lands available to each state to finance instruction in agricultural and mechanical arts either in new institutions or in previously established institutions bent to this new purpose. Support was provided later for costs of investigations into practical applications in agriculture and engineering and for the establishment of so-called extension educational activities which would bring directly to individuals in rural areas, to farmers on their own doorstep, information concerning useful procedures in the conduct of everyday business on their farm. The results of this pragmatic approach to knowledge in agriculture, and similarly in engineering, were so obviously useful as to cause this motive to be applied gradually to many other kinds of human activity in a necessarily innovative society.

This practical aim of the university, the "how to do it" motivation, was introduced prominently into the university when the American people found themselves confronted by the frontier of the wilderness; but this same motivation persists as the frontier has ceased to be that of the wilderness and has become that of swift technological and sociological change equally demanding of many new skills and new applications of knowledge. Now that Europe similarly confronts rapid technological and sociological change, Europeans would appear to be finding it useful in their context to encourage a more practical attitude on the part of their universities. Hence in recent years the *dignitas* of American universities seems to be suffering less by comparison with their European counterparts.

Motivations in American Universities

Be that as it may, the modern American university is multipurpose in the sense that it is motivated by three kinds of concerns: Concern with being, knowing, and doing. These three motivations are not equally distributed in each member of the faculty, but they will all be voiced within the faculty collectively. From person to person, from time to time, and from place to place within the university one concern will be in the ascendant over the others but not to the exclusion of the others. There is tension among these basic goals and continuing argument over them among their protagonists within the faculty. But these aims endure, and they continue to add variety to the ways in which the university periodically evaluates and weighs its stance toward whatever human activity or interest it takes within its orbit.

The American university impelled by these three different kinds of motivation has constantly added subjects to the basic disciplines in the arts and sciences, widening the range of phenomena in nature and society brought within the analysis of the disciplines. It has also dignified by its attention an ever enlarging list of professions. To university instruction for the long established learned professions of the ministers, doctors and lawyers, it has added university preparation for engineering, mining, agriculture, forestry, fisheries, oceanography, business administration, industrial relations, dentistry, pharmacy, nursing, social work, school teaching, school administration, librarianship, public administration, urban planning, architecture, art, music, drama, and so on. The catalog of any major university will reveal the enormous variety of programs of instruction offered. In this sense too the American university has become a multi-purpose institution.

Yet another feature of the university reveals its multi-faceted operations. The age spread of persons affected by the university's instruction is becoming almost all inclusive. The earlier years of men from the cradle to roughly 18 years of age are influenced indirectly largely through the university's professional instruction of their teachers in the elementary and secondary schools; but thereafter from roughly 18 years of age to the grave individuals may never fully escape the grip of the university. In an ever growing number of instances a student beginning at 18 may spend at the university in continuous formally organized study up to a dozen years by passing through a sequence of instruction for undergraduate and graduate or advanced professional degrees or certificates. In recent years more and more adults who have completed what might be called the education preparatory to their adult roles are coming back to the university periodically for refreshment in a host of types of specially organized instructional programs in what is now called continuing education.

In yet another sense the American university is a multiversity. I refer to the mounting tendency to house all of the higher learning and responsibility for all the professions within one all-encompassing, comprehensive institution called the university. While there are a few institutes of technology, specially devoted to science and engineering, of which the Massachusetts and California Institutes of Technology are the most distinguished examples, the larger number of candidates for science and engineering are to be found within the comprehensive universities. Indeed, I doubt that M.I.T. or Cal Tech would be refounded today; they are with some agony, and yet in some instances with remarkable results, reaching out for the humanities and social sciences and becoming indeed something more than institutes of technology, something approximating the broader character of a university. The free-standing separate teachers college or institute is even more a thing of the past as all universities have come to include teacher education within their compass and as the former teachers colleges have assumed broader responsibilities. Similarly, except for a few vestigial and unimportant remains, all law schools are within the orbit of a university. The same can be said for medical schools, but on that subject I will have more to say in a moment. The last arrivals within the university compound are the creative or performing arts. Universities having accepted responsibility for architecture at least 50 years ago, are now embracing instruction in the actual creation of art (painting, sculpture, design), music, drama, even dance. The separate schools, academies, or conservatories of art, music, and drama are withering on the vine; and it is a certain number of universities with the courage—or folly if you like—to ignore the scorn of academic conservatives which are now becoming the principal centers of education for the perpetuation and furtherance of our aesthetic capacity to perform in the arts.

What I have been saying is that the American university moved by its triple motivation for being, knowing and doing, is embracing a host of interests of concern to many kinds of men and women; it is helping men do many kinds of things in human society through its zeal to learn much through research and its willingness to teach many of many ages and many walks of life. It has therefore emerged in the post World War II period as an institution serving not a few needs of a narrow elite, but rather many needs of an enormous range of the American people from high to low estate, from exalted to humble positions, who are now included within its instructional programs.

The Public's Attitude Toward Universities

The "ivory towers" of the university, even if they still survive physically here and there amid trees on grassy lawns in small towns, thanks to automobiles, trains, jet airplanes, air mail, telephones, television, computer hookups, and innumerable conversations and conferences are linked in a partnership with business, industry, the professions, welfare and cultural institutions, and government. To all these varied institutions the university contributes ideas, provides sharpened skills, and offers evaluative criticism even when the latter is not requested. It is no wonder then that the university is now seen by the public as one of the cardinal institutions of American life, an ever present help in time of need, the source of the brainpower needed to solve our problems, the creator of our—and it is implied—better future. If members of university faculties or I were to say such things, it might be reasonable to accuse us of megalomania. But this kind of statement about the university is what more and more non-academic Americans are saying about their universities. Indeed, it is the very magnitude of their expectations of the university which makes many American academics and certainly university presidents worried about, and indeed the more critical of, their own university enterprise for fear that it may not live up to the challenge of these great expectations.

There are, of course, problems for the university in confronting such hopes and in endeavoring to do so much for so many: Availability of academic and supporting manpower, adequacy of physical facilities and supplies, sufficiency of funding, methods of financial support, organization and administrative structure, coherence, determination of priorities, maintenance of autonomy for flexibility and criticism of its own society, rate of growth and size, achievement of comprehension of common interests among the many specialists who make up the faculty of a complex institution.

Medical Education

These problems, however, are not the special focus of concern of medical educators. Let us turn them and the role of the university in society to the particular case of the relationship of medical education to the university itself. The burden of these remarks will be that the medical school is not as close to other faculties of the university and to the total university institution as appearances might suggest, or as it ought to be.

The earliest instruction in medicine in the United States, as you might expect in a frontier society, was provided through a feeble informal apprenticeship system. For some energetic aspirants in the eighteenth century such preceptorial instruction was supplemented by visits to Leiden, Paris, London and Edinburgh in which they could encounter clinical instruction in hospitals and formal lectures and demonstrations in a university setting. Against these precedents there developed in the late eighteenth century what could be called university medical schools based on linkages between hospitals and certain colonial colleges, such as those established at Pennsylvania, Harvard, Columbia, and Dartmouth. But these imported European university medical traditions quickly died, and for most of the nineteenth century the very large number of medical schools which developed in the United States either had no university connection or had a purely titular connection with a university and were generally devoid of university financial assistance, curricular control, shared academic values, or any sense of participation in the academic standards established elsewhere in the university.

The Flexner Report

In the later nineteenth century, however, reforming voices began to be heard, the most dramatic manifestation of which was the establishment in 1893 of the Johns Hopkins University School of Medicine. But the influential articulation of a new case for university control of medical schools and for a new kind of university medical curriculum was provided by Abraham Flexner in 1910 in his famous report for the Carnegie Foundation for the Advancement of Teaching entitled *Medical Education in the United States and Canada.* If I turn now to Flexner, it is because his recommendations prevailed with the help of foundation financial grants to a number of model-setting universities; and developments in medical education in the United States from his time are still based with only minor modifications on Flexner's view of medical education or on extrapolations therefrom.

Let us dispense quickly with a certain number of Flexner's recommendations which were broadly adopted and which are not now arguable.

He advocated that all medical schools should be controlled by universities. Many of the non-university schools prevalent in 1910 expired quickly. Today there are 88 medical schools established and in full operation, and the number of them is increasing every year. Only five from the pre-Flexner period remain without university control, though their curriculum now comports with the university medical schools' curriculum.

To Flexner, university control implied university assumption of a large measure of financial responsibility for the more costly program of medical education he advocated. The previous financial dependence of medical education mainly or only on the fees paid by the students themselves has long since passed out of the picture along with the proprietary, profit-seeking character of many of the earlier schools. Universities are now subsidizing the heavy cost of medical education, far above the level of fees received from students, with state appropriations and federal grants, with private endowments and gifts. Medical schools have indeed achieved the honor of being the costliest part of a university's educational obligations. The slightest suggestion today that there might be any financial profit in medical school operations would throw university presidents into gales of hysterical laughter.

According to Flexner, medical educators were to be professors, members of a faculty appointed as others were appointed to the university faculty and salaried like them by the university. Except for some special privileges in some institutions for some clinical medical professors, usually to permit them to have even higher personal incomes, and except for some uncompensated voluntary faculty drawn from neighboring practitioners, medical faculty members are now not only salaried but characteristically as a group are now the highest paid members of an American university faculty.

Flexner proposed as the minimum—and one could say normal—requirement for admission of students to the medical school the successful completion of two years of undergraduate study beyond high school, based in turn on university established criteria for the admission of high school graduates to the university itself. Today most students are admitted to medical schools after the completion of the four-year baccalaureate degree and none are normally admitted with less than three years of undergraduate study.

Flexner proposed that the M.D. program proper should itself be a four-year program superimposed upon two years or more of pre-medical undergraduate instruction. Only after the completion of this minimal six-year course to the Doctor of Medicine degree could a man be regarded as fit for the practice of medicine. Deferring for a moment the question of the content of his educational experience, it is interesting to note that Flexner dismissed post-graduate, post-M.D. education with only a few words about its being sometimes merely remedial, or a refresher course in practical skills or, it might be hoped, a further exploration under the aegis of a university clinical professor by such individuals as might wish to pursue some special matter beyond the usual M.D. educational experience. We could surely agree that it was quite enough for Flexner in 1910 to devote attention to the cure of the sickly M.D. program without worrying much about post-graduate medical education. But today the post-graduate years in internships and residencies for most prospective physicians are absorbing as many years as, or more years of instruction than, the four-year M.D. program. Even so, contemporary post-graduate medicine characteristically is still an extrapolation from, a specialized extension from, the base erected in the M.D. program. Its general nature is noted in the recently published report entitled *The Graduate Education of Physicians* by the Citizens Commission on Graduate Medical Education appointed by the American Medical Association.

Let us turn to the content Flexner proposed for the M.D. course of study. It is worthwhile rereading him to detect the still prevailing assumptions of American medical education. As everyone knows he proposed that in his four-year medical curriculum the last two years should be devoted to instruction under professors of clinical medicine. In addition to lectures and typical classroom or laboratory demonstrations the budding physicians were to be taken into university controlled hospitals and outpatient clinics, there to be brought into association with actual patients and patient care under the general supervision and preceptorial instruction of a professor physician. This reminds one of the innovations of Leiden's Boerhaave. But listen to Flexner describe the method to be used. Speaking of the historical development of such clinical teaching he says:

> "It was first didactic: The student was told what he would find and what he should do when he found it. It was next demonstrative: Things were pointed out in the amphitheater or the wards, those who got the front seats seeing them more or less well. Latterly it has become scientific: The student brings his own faculties into play at close range,—gathering his own data, making his own construction, proposing his own course, and taking the consequences when the instructor who has worked through exactly the same process calls him to account: The instructor, no longer a fountain pouring forth a full stream of knowledge, nor a showman exhibiting marvelous sights, but by turns an aid or an antagonist in a strenuous contest with disease."

Here Flexner is speaking of science as a method, a technique applied to clinical analysis and treatment.

But of course he wanted to bring science in another sense into medicine. Under his recommended two years of instruction in clinical medicine in hospitals he would lay a foundation in sciences basic to medicine by providing two years of systematic laboratory instruction as he says, in the following subjects:

> *"First year:* Anatomy, including histology and embryology; physiology, including bio-chemistry.
>
> *"Second year:* Pharmacology, pathology, bacteriology, physical diagnosis."

Such is still in essence the basic science portion of the American M.D. curriculum.

Flexner justified this interpretation of the sciences basic to medicine in the following way: After noting that there were three characteristic stages in the evolution of medical teaching, the first being the era of dogma highlighted by the traditions based on Hippocrates and Galen, and the second being the era of the empiric initiated by the introduction of anatomy in the sixteenth century, he came down to his own time to affirm:

> "The third era is dominated by the knowledge that medicine is part and parcel of modern science. The human body belongs to the animal world. It is put together of tissues and organs, in their structure, origin, and development not essentially unlike what the biologist is otherwise familiar with; it grows, reproduces itself, decays, according to general laws. It is liable to attack by hostile physical and biological agencies; now struck with a weapon, again ravaged by parasites. The normal course of bodily activity is a matter of observation and experience, the best methods of combating interference must be learned in much the same way. Gratuitous speculation is at every stage foreign to the scientific attitude of mind."

In a related vein he quotes Huxley as saying:

> "There can be no doubt that the future of pathology and of therapeutics, and *therefore of practical medicine,* depends upon the extent to which those who occupy themselves with these subjects are trained in the methods and impregnated with the fundamental truths of biology." Flexner himself then adds:
>
> "Now the medical sciences proper—anatomy, physiology, pathology, pharmacology—already crowd the two years of the curriculum that can be assigned to them; and in so doing, take for granted the more fundamental sciences—biology, physics, and chemistry—for which there is thus no adequate opportunity within the medical school proper."

It is because the medical student needs a useful preparation for studying the "medical sciences proper," or basic sciences as we say, that he must have behind him at least two years of undergraduate pre-medical experience in which he can acquire the necessary grounding in biology, physics, and chemistry.

At last I reach the essential point of this pilgrimage. Flexner's view of medicine (and the view that still commands the scene in American medical education) is that medicine is part and parcel—and I interpolate, *of that part*—of modern science which views man as a human body that, as he says, "belongs to the animal world. It is put together of tissues and organs, in their structure, origin, and development not essentially unlike what the biologist is otherwise familiar with, it grows, reproduces itself, decays, according to general laws. It is liable to attack by hostile physical and biological agencies, now struck with a weapon, again ravaged by parasites."

What marvelous results have come in our day from this strictly biological view of man as the object of medicine! I will not attempt to suggest even one of the triumphs of modern medicine; they are legion. The chances are that all of us are still alive now because of one or more of them.

The Need for Social Insight

But even a mouse may look—and squeak—at a king. Is this anything like a sufficient view of man whose health is in the care of a physician?

I call as principal witness Flexner himself who near the end of a chapter entitled "The Proper Basis of a Medical Education" says:

> "So far we have spoken explicitly of the fundamental sciences only. They furnish, indeed, the essential instrumental basis of medical education. But the instrumental minimum can hardly serve as the permanent professional minimum. It is even instrumentally inadequate. The practitioner deals with facts of two categories. Chemistry, physics, biology enable him to apprehend one set; he needs a different apperceptive and appreciative apparatus to deal with other, more subtle elements. Specific preparation is in this direction much more difficult; one must rely for the requisite insight and sympathy on a varied and enlarging cultural experience. Such enlargement of the physician's horizon is otherwise important, for scientific progress has greatly modified his ethical responsibility. His relation was formerly to his patient—at most to his patient's family; and it was almost altogether remedial. The patient had something the matter with him; the doctor was called in to cure it. Payment of a fee ended the transaction. But the physician's function is fast becoming social and preventive, rather than individual and curative. Upon him society relies to ascertain, and through measures essentially educational to enforce, the conditions that prevent disease and make positively for physical and moral well-being. It goes without saying that this type of doctor is first of all an educated man."

So the practitioner of medicine deals with facts of two categories. One set he is to master by a carefully organized and planned pre-medical and medical curriculum elaborately described by Flexner. The other set, much more difficult according to Flexner, he is to become master of somehow by being "first of all an educated man." And there Flexner let the matter of the physician's acquiring competence to deal with the second set of facts drop. And to all practical purposes so have our medical educators to the present.

Let us not be churlish. We are enormously in debt to Abraham Flexner for what he did do in 1910 and to those who have built our modern medical schools in the image then portrayed by him. But I submit we need some new Abraham Flexners to undertake the analysis of that "more difficult" second set of facts relevant to the profession of the physician. By attention to them educational planning may achieve something for the better care of patients as it has obviously achieved great results from its consideration of the first set of facts.

People come into hospitals and outpatient clinics and exist in their homes and in places of work and of other activity not only with bodies similar to those of animals but with memories; hopes; fears; aspirations, senses of achievement and of failure; loves, hates; and capacities for self-expression, social interaction, cultural behavior, and artistic play; and we know that all these human qualities can and do affect those same tissues and organs with which the biologist is familiar; and vice versa. Man belongs not only to the animal world observed by the biologist. He is, as the philosopher has observed, a social animal, and as the novelist has described, a creature of affect. The physician who is educated to see his patient as only a collection of interrelated tissues and organs is not seeing his patient whole; and, except as he may be aided fortuitously by untutored intuition, he will not be able to deal with his patient's health in all its aspects.

Even as the physician should be prepared to deal with the biological, social, and emotional aspects of his patient's life if he is to help preserve or restore the latter's health, in yet another sense he is in need of deep social insight. Under the emerging circumstances of the present, if the physician is to retain his customary role, he must see himself also as a participant and a social administrator in an ever more complex social structure designed to provide health care in a comprehensive and continuing manner for an ever larger proportion of the population. He must be not only a practitioner of applied biology; he must also be a practitioner of applied social science if patients are to be guided by him through the interrelated specializations and institutions of a modern health care industry. These two needs, seeing the individual patient whole and guiding the sociological and technological development of appropriate health care facilities, are very demanding. Where can the physician turn for help?

Bridges to the Other Sciences

The university, especially the comprehensive American university, is filled with faculty members from many disciplines and professions who are already possessed of bits and pieces of knowledge and skills which could be applied to the facts of that more difficult second category which must be controlled by the physician. And they could be inspired to seek still more by the prodding of persons imaginatively concerned with problems of health. But it will require the leadership of the physician and the medical educator, themselves acting in a sense as brokers for the sick, to exploit for medicine in this larger sense what can be harvested from other parts of the university, from sciences other than the traditional basic medical sciences, from social or behavioral sciences, yes, even from the humanities, and from other fields of professional application, engineering, business and public administration, social work, and nursing. Stimulation of discussion of health care and of disease in the broadest sense to include among other things "diseases of civilization" as René Dubos calls them, could bring into contributing roles for medicine far more kinds of scholars and scientists than are now regarded as related to medicine and health care.

The truth of the matter is that men of our medical schools have come into our universities in a real but in a still limited sense. Tied for obvious and historic reasons to their hospital laboratories, their clinical faculties have often been physically separated from the rest of the university sometimes even in cities a hundred miles or more from the rest of the university; usually the basic science faculties in the Flexner sense have likewise been pulled into proximity to the clinical environment and away from other sciences. But even when clinical and basic medical science professors have been geographically close to the rest of the university, the narrow definition of medicine's interest has usually diminished contact with other faculties even in closely related disciplines. While separatistic tendencies exist in all faculties in an American university, medicine has been one of the most isolationist of all in many universities.

Psychiatry might conceivably have been a bridge from medicine to other disciplines within the university but in practice it has had its own problems in attaining respectability in the eyes of the biologically oriented medical fraternity, and it has so far been able to serve only as a limited link to other parts of the university. Some additional centers of energy within medicine are needed to make congenial the task of exploring the utility for medicine of the university in a larger sense. Happily for my thesis I can report that in the last decade especially there has begun an opening of communication between medical faculties, both clinical and "basic science" faculties, with other segments of the university community on some university campuses. Some of our leaders in medical research at this very time are beginning to investigate the possibilities of engineering for so-called bio-engineering; and they are also asking questions about the status of social science, usually called behavioral science, to see if intellectual intercourse with these breeds might be honorable and useful.

But more is needed than tentative half-hearted fishing outside the present medical education compound. In my judgment it is time to revise the basic Flexnerian definition of medicine itself to include a conscious and planned concern for that mysteriously "educated man" who then presumably acquires a carefully designed medical education to enable him to carry out the less difficult half of his profession. It is time to work on that "education" which will provide a larger view of man and of health care responsibilities.

To conclude, I am not trying to chastise medical educators and physicians. It is rather that I wish to make a vigorous appeal to a remarkably conscientious profession which has already done so much for mankind. I urge it to undertake that next big step. In essence medical educators, acting in conformity with a narrow definition of the physician's responsibility, have so far used a very narrow segment of the intellectual resources of a university. Admittedly what they have done has been to the enormous advantage of our health. But I am convinced that there is yet more treasure in the university to be mined for the improvement of man's health.

Past dalliance between medical schools and the university has produced pleasurable results. If Fielding's view of man suggested by my title could be joined with the biologist's view of man and the medical school should embrace Alma Mater more fully and more vigorously, who knows what productive results for the human race may flow therefrom!

This then is my plea to my colleagues in medical education in the United States. I have let you listen to it. I must leave it to this audience to determine the relevance of this plea to your own situation.

Marijuana:

Social Benefit or Social Detriment?

EDWARD R. BLOOMQUIST, M.D., *Glendale*

THE CURRENT INTEREST in legalizing the use of marijuana as a social catalyst and as a tool with which parlor psychologists can probe their restless minds is of sufficient magnitude that physicians can no longer ignore nor avoid taking a stand on the marijuana controversy.

The urgency of taking an intelligent, informed, united position on this subject is augmented by two growing problems: The entrance of sociologically important persons into the field of drug abuse and the insistence of a growing number of educators and other influential persons that marijuana is a harmless drug that is less of a problem than is alcohol or tobacco, and thus that its use should be included in our group of socially accepted activities.

These attitudes have caused a growing number of individuals to acquire criminal records because of their defiance of existing narcotics laws. As a result some 450 new arrests for marijuana violations involving persons with no previous drug abuse records are currently being recorded by enforcement officers each month in California.

The solution offered by proponents of the use of marijuana—that if there were no laws restricting the use of marijuana there would be no enforcement problem—is specious and dangerous. The solution they offer is the legalization of the use of marijuana on the grounds that current laws are, among other things, discriminatory and a violation of the constitutional rights of persons who prefer to smoke marijuana instead of tobacco or instead of indulging in alcohol.

Since drug abuse is a medical problem as well as one involving enforcement agencies, physicians may soon find themselves in the position of having to advise their patients, as well as younger members of their families (for the use of this drug has begun to cut across all social and economic levels) as to the propriety or impropriety of indulging. Few physicians, however, are sufficiently informed on this subject to act as mentors when an inquiry is made. For this reason it seems wise to summarize some of the contemporary thought concerning the use of this drug, to review its main pharmacophysiologic features and to examine some of the attitudes of those who think it ought to be used at will.

Chemical Features

The active principle is obtained from the amber-colored resin of the flowering tops and leaves of the female hemp plant. The resin contains most if not all of the active principle, apparently tetrahydrocannabinol.

Psychotoxic activity produced by the plant seems to be due to the composite effect of its individual component principles. Hence, various samples of the crude material will vary considerably in potency and effect. This creates a most unpredictable situation should one desire to determine a "typical" clinical reaction which could be relied upon to reasonably repeat itself in a given user on future occasions.

Appearance

Marijuana, the leaves of the hemp plant *Cannabis Indica,* cannot be confused with tobacco. It is green, rather than brown. It smells like alfalfa

---

From the Department of Anesthesiology, Los Angeles County General Hospital, Los Angeles.

This article was submitted by the Committee on Dangerous Drugs of the California Medical Association in furtherance of a House of Delegates resolution that an educational campaign be conducted to alert the medical profession to the problems of drug dependence and drug abuse.

The author is Associate Clinical Professor of Surgery (Anesthesiology), University of Southern California School of Medicine.

Submitted 8 August 1966.

Reprint requests to: 1910 Niodrara Drive, Glendale 91208.

when burned. It appears on the illicit market in this country as a bulk compound or as cigarettes which are made by rolling the weed in double wheat straw paper and tucking in the ends to prevent loss of the prickly, loosely packed weed. The "joints," as these cigarettes are called, burn hotter than do cigarettes made of tobacco and the burning tip is brighter. The tip is easily extinguished unless a continuing effort is made to keep it alive.

Classification

Marijuana is a true hallucinogen which possesses elements of both stimulation and depression. It is not medically considered to be a narcotic, although the law declares that it is, and thus abuse of the drug may land the user in jail.

The problem of classification is one of nomenclature, in that in the United States any drug covered by the Harrison Narcotic Act of 1914 is considered to be a narcotic and violations are handled on this basis.

Recently the World Health Organization suggested a new nomenclature for abuse patterns of dangerous drugs. The terms *addiction* and *habituation* were replaced by the term *drug dependence,* with this dependence and accompanying features being considered part of an *abuse syndrome*. Physical dependence with its associated tolerance and withdrawal symptoms is recognized as being relatively unimportant except as it reinforces the principal problem—psychological dependence. Perhaps in time this nomenclature will be universally accepted and it would no longer be necessary to rely on inadequate, ill defined terms which long ago lost their specificity.

Pharmacophysiological Actions

In proper classification the hallucinogen marijuana presents a drug abuse pattern that includes psychological dependence, some tolerance and certain undesirable side effects. In man, the significant actions of the drug are manifested in changes referable to the central nervous system. The depression of blood pressure and respiration, the hypoglycemia which increases the user's appetite, the engorgement of ciliary vessels, pupillary dilatation, oropharyngitis, chronic bronchitis and asthma which may result from continued use are of secondary importance to the central effects.

Central Nervous System Effects

Shortly after inhaling the smoke the user notices a feeling of "inner joy" that is out of proportion to the apparent motivation. This is described as being "high." If the user is alone he may "trip off" and be quiet and drowsy or he may sit and watch the passing parade of technicolor illusions which occur as toxicity increases. In company he may be talkative and hilarious. His awareness, touch and perception are considerably altered, particularly as they relate to time and space.

Coordination is altered, although the user may fail to recognize this and complex intellectual capacities are impaired, particularly those which govern speed and accuracy. The individual's basic personality is not appreciably changed but his behavioral reactions may be modified. Self-confidence, often unwarranted, is one of the usual reactions. The user loses his inhibitions in varying degrees. During this early period he may feel relaxed and at ease. If a negative influence is insinuated, however, he may become anxious, slightly paranoid and apprehensive. This experience is known as a "bring down," a "downer," or a "bummer."

As he continues going up (or down) his judgment and memory are impaired. He becomes easily irritated and may, if the dose is high enough, become confused, disoriented, afraid and filled with a foreboding he may die. Behavior is impulsive and mood reactions are variable. Not infrequently the user experiences phantasmagoria—the sensation that figures are rushing toward him at tremendous speed, increasing in size as they approach. Conversely, they may depart from him, decreasing in size. As one user described this, "It's like a zoom lens on a TV camera."

With increasing doses, hallucinations may appear. If they are pleasant the user "trips" on them and remains high. If they are unpleasant he may become so upset and afraid that if the proper ingredients for the experience are present, a true though usually transient psychiatric reaction may develop.

The total effects of a marijuana "trip" last from three to five hours, after which the user feels slight lethargy and hunger. Experience will vary, however, since marijuana is an unpredictable drug which is usually employed by unpredictable persons.

Adverse Psychological Reactions

Most marijuana trips are reputed to be seemingly innocuous affairs which can be indulged in with less total destructive effect than one would experience from an alcoholic binge. For this reason many persons do not recognize the true dangers of the drug. Too many voice the sentiment of the high school student who argued, "I know it won't hurt me; I read about it in the library and I know." The problem is that the interested student often reads sources that are in accordance with his own beliefs and avoids information that would tend to destroy a cherished illusion.

The psychological effects of marijuana are as varied as the range of human personality and as complex as the multiple factors which influence the user each time he smokes the leaves. If calm and in a good mood when he takes the drug, he may sing and laugh and joke and enjoy himself harmlessly. Under other circumstances he may nod and dream. But he may enter a motor vehicle and with "teashades" (dark sunglasses worn because of the dilated pupils) over his handicapped eyes and with impaired reflexes he may plow through a crowd of pedestrians. He may meditate on the beauties of art, nature and music or he may inexplicably step out of an eighteenth story window to pick up a butterfly on the lawn below.

"Man," an adolescent user told this author, "when I'm up on weed I'm really livin.' I float up and up and up until I'm miles above the earth. Then, Baby, I begin to come apart. My fingers leave my hands, my hands leave my wrists, my arms and legs leave my body and I just flooooooat all over the universe." Depersonalization of this type is common with users if they inhale sufficient quantities of the weed's active ingredients.

Sexual Influence

For years opinion has varied concerning what effect the smoking of marijuana has on one's sexual response. This drug is not an aphrodisiac but it does influence sexual response by releasing restraints and thus permitting greater freedom of participation by the user. By distorting time and space the drug makes the experience of orgasm seem more prolonged and more intense. As the senses are far more acute than under normal circumstances, the experience seems more sensuous when it occurs when one is under the influence of the drug.

The desire for sex and stimulation to the act, however, must originate with the individual. Marijuana is no cure-all for impotence. But it is an efficient releaser of normal inhibitions. Whether or not such a release is advantageous is a matter of opinion, for these inhibitions serve to protect the individual and society from serious sexual misadventures.

Clinical Application

Neither Cannabis nor its active principles are currently considered to have valid clinical application despite the fact that in many countries the drug has been considered at one time or another as a cure for almost all illnesses.

Social Application

With the entrance of "intellectuals" into the drug user community, a caste system seems to have evolved. The castes differ to some extent in both their approach to and in their use of marijuana. The lower caste, usually composed of poorly educated, socially disadvantaged persons, simply "trip" on the effects of the drug; that is, they take the drug to experience the bizarre effect for the effect itself. In general a "trip" is a psychological excursion during which the user enjoys (or endures) certain emotional effects.

Since the intellectual challenge normally fails to appeal to the lower caste user he will, if he responds to the effect of the drug, try to remain high as much of the time as possible. If he decides to progress to a stronger drug for escapist purposes, he will often turn to heroin because of its sedative effect, rather than to more potent psychedelic substances.

The upper caste is composed of intellectuals—students, certain misguided faculty, professional men and the "arty" group—who take this drug either alone or as a group experience in an attempt to find an inner meaning to an existence which has become prosaic, empty, confused or meaningless.

This upper or intellectual caste has further been divided into three subcategories: The dabblers, the users and the heads. The dabblers are the counterparts of the "chippers" in the opiate field. They play with the drug, use it sporadically, think it is something daring and the contemporary thing to do. The "respectable" weedhead is usually found among this group.

The user is frequently a weekender. He indulges in his drug much as some of his friends with a different bent might enjoy a weekend beerblast. He likes the effects of the drug, considers the effect a mystical experience, smokes it for social as well as psychological reasons and, as a matter of ego satisfaction, tends to exaggerate the effects he derives. This group is important, for it is here that the "marijuana cult" has its beginning. It is here that the "kooks and radicals" as well as the otherwise seemingly reasonably well balanced users appear. And it is here that the potential for going on to more dangerous drugs makes its first significant entry.

Heads, *per se,* constitute only a small segment of the upper caste users at present; but they are important because they tend to be leaders of the marijuana cult and susceptible individuals are influenced by them. The head is in essence solipsistic. His world rotates around his drug experiences. He abhors authority whether it be parental, educational or legal. To him the whole respectable world is an enemy.

He frequently drops out of school, finding it difficult to communicate with or associate with persons who are not attuned to his philosophy. Regrettably, he often makes his home near educational centers where, because drugs are in demand, he can make a living selling drugs ("pushing") while at the same time attacking organized society. LSD and other psychedelics are the next step in his program, but hard narcotics are not unknown to him.

Among the intellectual drug abusers there is a serious tendency to use marijuana and other psychedelic drugs (substances which alter perception and produce mental distortion) for purposes of "maintaining" and for mental exploration. To "maintain," in this parlance, is to defer the enjoyment of the pleasurable effects of the drug and to utilize the experience to better understand one's inner self and to rid one's self of psychological problems ("hangups") which prevent one from arriving at the desired state of being master of himself ("cool").

The maintainers are usually intelligent, capable, socially useful persons who insist there is nothing dangerous about the use of the drug. Many feel they have found a useful psychological tool which will amplify their personality and individual understanding.

In talking to college students, Goldstein found it possible to make some rough generalizations concerning users in this group although there was no general conformation to a stereotype. The maintainer tends to be either apolitical or liberal. His drug experimentation tends to be a part of an overall pattern of rebellion against current social values which he tends to reject. Academically he may be an honor student—or a dropout. He is convinced there is no significant danger associated with the use of marijuana. The majority of student users, however, seemed to sense that progression to stronger psychedelic drugs may be associated with serious consequences. But in equating marijuana with other social indulgences the general feeling was that the only bad thing about the drug is that its use could land the user in jail.

These students gave various reasons for persisting in the use of the drug: "It broadened the perspective I had on myself." "I'm not so fooled by what people around me think." "I've got a firmer grip." "Really, I'm much calmer now."

"Pot takes the pressure off in a very real way," an honor student told Goldstein. "There's a sense of total, timeless ease."

Another student, however, brought the social factor into play: "Pot gave me a real sense of purpose, an assurance, a kind of concept of the straight and narrow, although that might sound ironic. The whole thing is the kids you smoke it with. You really start to share bits of yourself with the smoke. And after a while you've got friends, and an ideology, and even girls—you'd be surprised how many girls equate pot with seduction."

While the maintainers are intellectually inclined and are likely to be dabblers, the trippers represent another form of expression. This lower caste majority is frequently at odds with itself and with society. Its members use the drug, consciously or unconsciously, as a continuing weapon in their personal vendetta against a society with which they cannot cope. Sometimes, released by the effect of the drug, they do things that are startling and damaging.

Although it is inaccurate and unfair to try to categorize all members of this group in one stereotype, Goodman and Gilman have managed to accumulate certain striking characteristics of this level of user and with them paint a disturbing picture. The marijuana user, they state, "is usually 20 to 30 years of age, idle and lacking in initiative, with a history of repeated frustrations and deprivations, sexually maladjusted (often homosexual) who seeks distraction, escape and sometimes conviviality by smoking the drug. He almost uniformly has major personality defects and is often psychopathic."

How Marijuana Is Used

Smoking of marijuana requires a special technique. Usually several smokers will meet in a room (teapad) or in a car. They may, if avid enough, hole themselves up in a closet or a very small room so they can rebreathe as much smoke as possible.

The user takes a joint, drags on it, holds the smoke deep in his lungs, exhales slowly and passes the joint along to the next user. As the smoke fills the room, each user begins to feel the "buzz." With increasing rapidity the cigarettes are passed around the circle of smokers.

There is a specific point at which the user can assume he is high. At this point he seems to be freed from the restrictions of time and space. He can "free associate" with unbelievable ability. What occurs from this point on depends upon the mood of the crowd and the compliance of the individual users.

Surprisingly the user can walk on the street without appearing to the casual observer to be in any but a normal state. He talks with his usual ability, although sometimes hesitantly and with a slight slur. On the surface he seems quite coherent. But underneath he is probably tripping on the beauty of the sky, the noises of traffic or the smell of some nearby flowering bush or tree. If he is placed under stress or required to coordinate he may fail miserably in performance.

Use Pattern

In America, and for that matter in the world, the use of marijuana as an intoxicant is second only to that of alcohol. In this country it is usually obtained by contact with friends who are users. Not infrequently more affluent users will serve reefers to their friends as a social gesture much as one might offer a friend a cocktail.

New recruits know what they are doing when they smoke marijuana, since it takes an effort to acquire the drug's effect. Many youngsters are introduced into the use of this drug under social circumstances. They may begin because they are curious or because they want to go along with the crowd. Many will never return to the use of the drug but others, pressured by the wrong friends or because they psychologically respond to the effects of the drug, will continue to escape reality through smoking it.

Attempts to Legalize Marijuana

In recent years several societies have been formed to advocate the individual rights of those who wish to smoke marijuana. They ignore the example set by India, which after years of accepting bhang as a part of the religious life of the country, outlawed its use in 1959 because of the negative effects of the drug. In Africa, nations which once accepted marijuana have now begun to worry about its overall effect. In recent riots in Nigeria thousands of hoodlums, apparently under the influence of the drug, assaulted each other and the police, in screaming indifference to their own safety. As a result the military government there has decreed the death penalty for unauthorized sale or production.

The trend toward social acceptance and legalization of marijuana really began in America in 1945 when the mayor of New York became concerned about the effects of marijuana on juveniles and appointed a special commission to review the situation. The commission confined its observations to the behavior patterns and remarks of 77 prisoners who, it is possible to assume, told the commission what it wanted to hear. From this limited survey the commission drew conclusions that marijuana was not significantly associated with either delinquency or crime, not a cause of insanity, not a sexual stimulant. It also concluded that the effects of the drug were no more adverse than are the effects of the social use of alcohol.

Although roundly denounced by enforcement officials and by the American Medical Association, the 220-page report immediately became the Magna Charta of the weedheads. Its far-reaching effects are being felt today in California where advocates of marijuana are trying to place a bill on the state ballot that would legalize its use. Even if the bill should pass, federal law will still prohibit use of the drug, but psychologically the effect of a permissive law in California might be useful in argument for a change in federal law.

Drug Behavior Patterns

The variety of drugs that can alter man's perceptivity and behavioral patterns is almost limitless. Some do so with only a few adverse side effects and are thus useful drugs. Others, marijuana among them, are so unpredictable that the adverse reactions outweigh the beneficial effects. With a drug such as marijuana, with potency varying from batch to batch and reaction varying from user to user—and from time to time in an individual—the fact of unpredictability must be given full weight in any consideration of legalizing its general use.

The Tobacco Argument

One argument solemnly offered for the legalization of marijuana is that the smoking of tobacco is more injurious, is capable of producing both physical and psychological dependence and yet is accepted by society. Proponents of marijuana state that since the drug is less of a problem in these respects, prohibiting its use while sanctioning the use of tobacco is discriminatory. This line of thought ignores some great differences between the smoking of tobacco and the smoking of marijuana. Tobacco can be smoked while the smoker goes about other activities and with minimal danger to others in the immediate environment. Getting the effects of marijuana requires an intense effort which limits the smoker's ability to do anything else. Tobacco has little effect on the emotional reactions of the smoker. Marijuana specifically affects the emotional and mental responses to the point of producing feelings of omnipotence. The harmful effects of tobacco smoking are slow in making their appearance and they primarily affect the user rather than his associates. The effects of marijuana are quick to appear and may result in erratic behavior on the part of the user that can endanger those around him.

As for the association of tobacco smoking and marijuana smoking, very few smokers of tobacco progress to the use of marijuana, and then only because of an almost unassociated relationship. For that matter, very few users of marijuana will progress to the use of heroin. But it is significant that the large majority of heroin users begin the habit following their introduction to the abuse of marijuana.

The Alcohol Argument

Recently a noted sociologist[5] and proponent of legalizing marijuana stated: "The controversy with respect to marijuana is solely concerning the relative prevalence or frequency of such results in comparison with similar consequences following from the use of alcoholic beverages. All empirical investigations indicate that alcohol constitutes a far greater social danger than does marijuana."

Again this is the old argument that "I have just as much right to my poison as you do to yours." One should note in this respect, however, that there are in this country five to six million physically and mentally sick persons who have reached that condition because of their aberrant use of or reaction to alcohol. There is every reason to believe that marijuana has the same potential to produce an equal if not greater number of socially disturbed people. Noting this and in the light of the tremendous costs of the permissive use of alcohol to the people of this nation, one can only marvel at the seeming lack of social responsibility on the part of marijuana's proponents.

The argument that marijuana is cheaper, quicker and not physically addicting and thus can provide competition which would drive distilleries out of business and bankrupt the alcoholic beverage industry is another example of the unsupported, specious thinking of the marijuana proponents. It has been amply proved in countries such as India where marijuana (bhang) was socially sanctioned and alcohol disapproved, that those who prefer one drug will pursue it regardless of the availability of the other. India constantly had problems with enforcement of its alcohol laws despite the fact that bhang was legally available to anyone who wished to use it.

Marijuana and Crimes and Violence

Violent behavior has long been associated with the abuse of marijuana. There is little question that this occurs, although the majority of users may not exhibit this complication, for the behavior pattern of the smoker will vary with his own personality, the strength of the drug he is using and the circumstances in which he finds himself.

Enforcement officers so often encounter this manifestation of the use of marijuana that they consider it a matter of course. The sociologist who sees the user in the office, where the user is trying to make a good impression, may conclude that violence and crime are never associated with the use of marijuana.

Sociologists may make their case simple by stating that violence rarely or never occurs; enforcement officers, on the other hand can document such occurrences with case histories.

Concomitant Drug Abuse

Does the use of marijuana lead to the abuse of even more dangerous drugs? In many instances it may. There are two reasons for this and they vary with the caste that is employing the drug.

It is probable the minority of marijuana smokers who, once the effects of hemp become prosaic, turn to something stronger. But how small is this minority? Is it as small as the number of those who drink alcoholic beverages and find they cannot control themselves when under the effects? It is currently estimated that some 10 per cent of students in large urban universities employ marijuana in their personal or social activities. These users have in common the search for "inner truth" and "inner peace." More often than not they have become bored, curious or in desperate need of finding something "different" to experience.

The association of an already emotionally disturbed youth with marijuana abusers poses a real threat to the nation. This threat is better appreciated when enforcement statistics are considered which suggest that one out of nine American youths will become involved in serious delinquent behavior in the coming year.

When disturbed lower caste youth comes into contact with circles in which marijuana is used, two things happen. First, a significant number of them can be enticed to try something "better." Secondly, this group of "eligibles" become easy marks for heroin users who need money to support their own abuse patterns and thus want new recruits who will become steady cash customers. For it is well known that narcotic abuse is spread, not by wholesale pushers, but by the "little guys," the small-time users who are also peddlers.

The threat to youths of the better-educated circles is even greater. Here, the tendency is to experiment with "mind drugs" because of curiosity or a seeming need to explore and solve personal problems. Marijuana, because of its mild hallucinogenic effects, is a perfect introductory drug to the use of LSD, a drug capable to taking its users to weird "highs" and, on occasion, permanent "trips." The potential for personal and social harm resulting from the abuse of psychedelic drugs is frightening. This hazard is augmented by continuing efforts on the part of older adults who, disturbed themselves, tend to minimize the danger and encourage the use of psychedelic drugs in undisciplined environments for their "spiritual" and "intellectual" value.

Social Harm

There seems to be little lasting physiological harm occurring from the use of marijuana. Stressing this and seemingly ignoring the adverse psychological effects of the drug, proponents of marijuana advocate its legalization.

It is true the casual user, the curious one-timer and the investigator may take the drug without deleterious effects. But the chronic user develops a psychological dependence which, in view of today's knowledge, is the prime detrimental factor. This dependence soon causes him to lose control of his use of the drug because the psychological factors which drew him to try it in the first place now precipitate a pattern of chronic, compulsive abuse. At this point the user is just as "hooked" as are the persons we used to call addicts.

As the abuse pattern grows, the chronic user develops inertia, lethargy and indifference. Even if he does not have psychotic or pseudopsychotic episodes or begin a criminal or violent existence, he becomes a blight to society. He "indulges" in self-neglect. And even though he may give the excuse that he uses the drug because it enlarges his understanding of himself, it is the drug experience, not his personal development, which becomes the principal interest.

Motivation outside the drug experience becomes insignificant. Whatever antisocial tendencies are in his personality make-up are augmented by his continual abuse of the drug. In the end, society has on its hands another "drug bum" who expects to sponge a living while offering nothing in return.

The Current Problem

In effect the problem today is, Do we or don't we want individuals smoking marijuana? Some sociologists argue that "since the smoking of marijuana will undoubtedly continue regardless of legislation against it, it can also be argued that it would be better to accept the inevitable than to wage war for a lost cause." And some also argue that we should treat the marijuana problem the same as we have the alcohol problem. The difficulty, however, is that we have never really managed the alcohol problem; the abusive use of alcohol has created the major drug problem in the United States today.

Certainly there is wisdom in the comment of the President's Ad Hoc Panel on Drug Abuse: "It is the opinion of the Panel that the hazards of marijuana *per se* have been exaggerated and that long criminal sentences imposed *on an occasional user or possessor* of the drug are in poor social perspective" (italics supplied). But before transcending the meaning of this recommendation and permitting all who might wish to use marijuana to do so, it would be well to recall the warning of this country's sixteenth President who held that destruction of our nation could not come from abroad, but "If destruction be our lot, we ourselves must be its author and finisher. As a nation of free men, we must live through all times, or die by suicide.

Although President Lincoln had no reference to drug abuse when he made this statement, he could not have painted a clearer picture of a national menace where drug abuse is concerned.

BIBLIOGRAPHY

1. Associated Press: Nigeria crackdown, death penalty set for drug peddlers, Los Angeles Times, 12 May 1966.

2. Cohan, Sidney V.: Uncanny Power of the Hallucinogens, The Drug Takers, Time-Life Books, New York, 1965, p. 102.

3. Goldstein, Richard: Drugs on campus, Saturday Evening Post, 239:11, 21 May 1966.

4. Goodman, Louis S., and Gilman, Alfred: The Pharmacological Basis of Therapeutics, Ed. 2, MacMillan Co., New York, 1955, pp. 172-174.

5. Lincoln, Abraham: Address before the young men's lyceum, Springfield, Illinois, 27 January 1838.

6. Lindesmith, Alfred R.: The Addict and The Law, Indiana University Press, Indiana, 1965, p. 234.

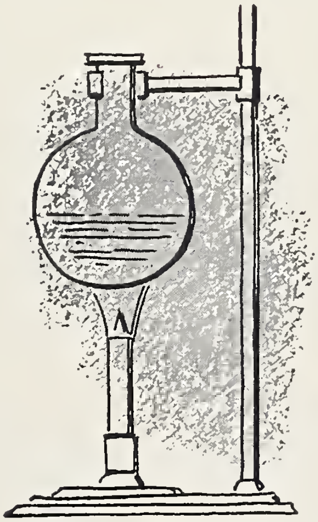
7. Munch, James C.: Narcotic addiction problems, marijuana and crime, I.N.O.A.E., 6th Annual Conference Report, 1966, p. 55.

8. Seevers, Maurice H.: A view of marijuana in perspective, Michigan Daily, 10 November 1965.

9. Williams, John B.: Narcotics, Wm. C. Brown Co., Iowa, 1963, p. 172.

10. Wilson, Earl: The crazy dreamers, Colliers Magazine, 124:27, 4 July 1949.

11. White House Conference on Narcotic and Drug Abuse Proceedings, U.S. Government Printing Office, 1962, p. 286.



Chronic Obstructive Pulmonary Emphysema

Is Exercise Beneficial?

LASZLO AMBRUS, M.D., SAM H. THAL, M.D., SELIG B. WEINSTEIN, M.D., AND
JUANITA WARNECKE, R.P.T., *Martinez*

■ *Following a six-week program of training in a series of exercises, a significant number of patients with chronic obstructive pulmonary emphysema showed decided improvement in functional activity. Subjective improvement also was noted and kept the patient motivation high.*

Preliminary observations indicated that the improvement could be maintained long after the end of the training period.

CHRONIC OBSTRUCTIVE PULMONARY emphysema (COPE) is an increasingly important medical problem. It is estimated that in the last 10 years the number of patients with this disease has quadrupled.[1] U.S. Public Health Service surveys in 1960 estimated more than one million persons had emphysema.[8,9] Deaths from the disease in 1963 totaled 17,595, an increase of 600 per cent from 1953.[1]

The treatment is controversial and various therapeutic regimens have been tried. Many investigators consider the stress imposed by exercise to be deleterious for patients with COPE. For this reason, limitation of physical activity is recommended. Noehren[13] listed 23 therapeutic measures but did not mention graded exercises. Yet clinical observations, such as Barach,[3,4] Pierce,[14] Lyons[11] and Miller,[12] indicate that patients with COPE benefit from physical exercise.

This presentation is concerned with evaluating the efficiency of a specific physical treatment program combined with the usual medical treatment.

Material and Methods

Forty-three male patients with chronic obstructive pulmonary emphysema, all of them in hospital, were selected for a program of graded exercise training. Pulmonary function studies, blood gas analysis and various functional activities and patients' attitude (Table 1) were measured at the beginning and at the end of a six-week period. Three dropouts occurred, two due to severe illness and one to emergency operation. The patients were treated five days weekly in groups of four to five, in air-conditioned rooms. None had previously followed a program of regular exercises. The grade of dyspnea and tests of ventilatory functions of the remaining 40 patients at the beginning of the training and at the end of six weeks are shown on Table 2.

The combined exercise program (Table 3) consisted of breathing exercise, relaxation exercise, postural exercise and general conditioning exercise.

Breathing Exercises. Breathing exercises consisted of training in diaphragmatic breathing—inhalation through nose and exhalation through pursed lips, with exhalation controlled to take twice as long as inhalation. A metronome was used for training and indoctrination. These exercises

---

From the Physical Medicine and Rehabilitation Service, the Pulmonary Function Laboratory, the Pulmonary Disease Section and the Physical Therapy Section of the Veterans Administration Hospital, Martinez.

Submitted 1 August 1966.

Reprint requests to: 1050 W. Pacific Coast Highway, Harbor City 90710 (Dr. Ambrus).

were done in sitting, standing and supine positions. In the supine position, with the head lowered 15°, the patient placed one hand on the thoracic cage and the other on the abdomen to gain tactile sensation of diaphragmatic movements. Later, in the supine position, weight (sandbags 2 to 30 pounds, as tolerated) was placed on the abdomen to accentuate the rise on inhalation and the fall on exhalation. Exercises used by the British Asthma Research Council were followed in general.[2]

TABLE 1.—*Measurements of Ventilatory Function and Activities*

Test of Ventilatory Function	Forced expiration volume, liters (FEV) Maximum voluntary ventilation L/Min. (MVV)
Arterial Blood Studies	Blood pH, units O_2 saturation per cent O_2 tension, mm mercury CO_2 tension, mm mercury
Functional Activities Muscle Strength Relaxing Ability	Walking on level—distance and time Stair climbing—number of flights and time Exerow activity tolerated, time Walking on Treadmill 15° incline, distance and time Hand grip measurements, pounds General strength Relaxing ability
Breathing Exercises	Number of blows required to transfer 1 liter of water Longest single blowing time Chest expansion changes: a. Nipple line b. Rib angle c. Navel line
Occupational Therapy Activity	Activity tolerance Attendance Motivation

TABLE 2.—*Grade of Dyspnea (Functional Activity)*

Grade	*Initial*	*Six Weeks*
I	0	4
II	3	15
III	18	12
IV	12	9
V	7	0

No grade change in 10 patients. One grade up in 26 patients. Two grade up in four patients.

Test of Ventilatory Function—Forced Expiration Volume (FEV) *and Maximal Voluntary Ventilation* (MVV)

Grade	*Initial*	*Six Weeks*
I	0	3
II	7	4
III	7	15
IV	19	14
V	7	4

No grade change in 20 patients. One grade up in 17 patients. One grade down in three patients.

In the afternoon blow bottle exercises were used in sitting and standing positions for a total of 20 minutes.

Physical Relaxation Exercises. Physical relaxation exercises were practiced in sitting, standing and recumbent positions. The patients were made aware of their contracted and relaxed muscles in sitting and recumbent states.

Postural Exercises. The postural exercises were performed once daily for 10 minutes in standing and sitting positions. (Before each training session, the therapist stressed posture.)

Progressive Resistive Exercises. Progressive resistive exercises consisted of upper extremity, lower extremity and trunk exercises in the gymnasium twice daily. Wall pulleys, treadmills, rowing machines and exercise tables, shoulder wheels and weights were mingled for a change of program. Progressive ambulation and stair climbing were practiced daily.

Before the program of exercises was begun, medical history, physical examination and laboratory tests (including complete blood cell count, urinalysis, x-ray film of chest and electrocardiogram) were completed. After examination, the patients were graded in accord with the respiratory system classification of the Committee of Rating of Mental and Physical Impairment[6] (with slight modifications). Table 4 shows the grades of ventilatory function and dyspnea. (The grade of dyspnea does not correspond at all times with the grade of ventilatory functions shown in Table 2.)

None of the patients was acutely ill during the

TABLE 3.—*Exercise Schedule*

9:15-10:00	Postural drainage, breathing, relaxation and conditioning exercises
10:00-11:00	Rest, recreation, room activities
11:00-11:30	Relaxation, breathing, posture exercises
11:30-12:00	General conditioning exercises in the gymnasium with treadmill, pulley, rowing machine, exercise table, etc
12:00- 2:00	Lunch, rest
2:00- 2:30	Breathing, relaxation, ambulation exercises
2:30- 3:00	Breathing (blow bottles) ambulation, stair climbing exercises
3:00- 4:00	Occupational therapy program (plastic—bench work, floor looms)
4:00- 5:00	Rest, supper

TABLE 4.—*Data on Ventilatory Function and Functional Activity Related to Grade*

Test of Ventilatory Function	*Grade*	*Dyspnea Functional Activity*
Not less than 85 per cent of the predicted normal	I	Unrestricted
70-85 per cent of the predicted normal	II	Can do moderate activity
55-70 per cent of the predicted normal	III	Can do light work, not involving hurrying, climbing, heavy lifting
55-35 per cent of the predicted normal	IV	Present on level walking of 100 yards, or after few minutes
Less than 35 per cent of the predicted normal	V	Present on slight exertion, dressing, talking, even at rest

training and observation period. The medical treatment (consisting mainly of bronchodilator therapy, intermittent positive pressure breathing) was continued during the observation period. Avoidance of respiratory irritants (smoking) was stressed.

Thirty-three patients had a history of heavy cigarette smoking (more than one package daily). Seven said they did not smoke. Sixteen said they had a heavy intake of alcohol, 19 that they drank "socially" and five denied drinking.

Previous pulmonary disease was discovered in 36 patients (tuberculosis, bronchitis, asthma). Eight patients had previous histories and findings of right heart failure, and 20 had gastrointestinal complaints and findings.

Four patients were between 35 and 44 years of age, another four between 45 and 54, seven 55 to 64, twenty-two 65 to 74 and three over 75. The youngest patient was 39 and the oldest 78. There were 36 Caucasians, three Negroes and one Oriental.

All patients received and used the Gordon-Barach Belt for abdominal support.

After the period in hospital the patients were urged to continue their training program of the exercises they had learned, and blow bottles were issued to patients for home use.

Results

Of the two variables in ventilatory function that were tested, forced expiration volume (FEV) and maximal voluntary ventilation (MVV), the FEV showed an increase in the mean value, significant at better than 0.01 probability level. The mean value changes in MVV were statistically not significant. However, the oxygen tension measured showed a differential at the 0.05 probability level. Blood pH, oxygen saturation and carbon dioxide tension did not show significant changes. Table 5 shows the six variables measured, the mean values before and after training and the differences between the values.

Functional Activities. Greatest improvement was registered in the tests of functional activities. All the activities, except speed of stair climbing, showed improvement at the probability of 0.01 level. All of the tests for increased endurance were highly significant with Student's "T" test values of 3 to 7. Speed of stair climbing would appear to be a measure of other factors besides endurance.

Breathing Exercises. Significant improvement was noted in both the number of blows required (Table 1) and in the longest single sustained blow, as was to be expected with training. However, the

TABLE 5.—*Test of Ventilatory Function and Arterial Gas Studies*

*Variables**		*Mean of First Variable*	*Mean of Second Variable*	*N*	*T*
First	*Second*				
1	2	224.97	243.00	39	2.99†
3	4	63.14	68.38	39	3.01†
5	6	40.00	43.12	39	1.96 N.S.
7	8	42.74	45.79	39	1.75 N.S.
9	10	7.37	7.37	34	0.04 N.S.
11	12	92.50	94.29	34	1.81 N.S.
13	14	78.29	82.26	34	2.12‡
15	16	47.05	45.97	34	0.87 N.S.

N=Number of paired measures. T=Student's "T" test.

N.S.=Means non-significant difference between means.

*Variable factors before (first) and after (second) six weeks of training.

†=Means are significantly different at the .01 probability level.

‡=Means are significantly different at the .05 probability level.

1 vs 2—Forced expiration volume liters.

3 vs 4—Forced expiration volume liters: Per cent of predicted normal.

5 vs 6—Maximal voluntary ventilation (L/Min).

7 vs 8—Maximal voluntary ventilation (L/Min): Per cent of predicted normal.

9 vs 10—Blood pH, units.

11 vs 12—Arterial O_2 saturation per cent.

13 vs 14—Arterial O_2 tension, pO_2, mm, Hg.

15 vs 16—Arterial CO_2 tension, pCO_2, mm, Hg.

Information in this table prepared by the Western Research Support Center, Veterans Administration, Sepulveda, California.

expansion changes at navel level indicate improvement in diaphragmatic excursion. No changes were noted in measurement at the nipple or rib angle.

Table 6 shows the functional activities and breathing exercises measured initially and after training.

Occupational Therapy Activities. Four patients refused to enroll in programed activities, but participated in recreational activities (billiards, model assembly). Motivation was high to adequate in 32 patients, poor in four. Ninety per cent of the patients had good to excellent attendance. The activity tolerance, as measured by the finished task, showed an increase. Ninety per cent of the patients progressed to 60 minutes of activity in sitting or standing positions. The patients became less anxious and restless as indicated in their ability to relax, as follows:

	Poor	*Fair*	*Good*
At start of program	20	14	6
Six weeks later	3	21	16

Of the 40 patients studied, 11 used the intermittent positive pressure breathing aid before entering the hospital, 24 during the stay in hospital and four after the training period. There was less reliance on IPPB apparatus after training.

None of the eight patients who had previous history and findings of right heart failure had cardiac difficulty during the training, in spite of a doubling of activity.

A more efficient respiratory pattern was developed during exercise, and this change could reduce the energy cost of breathing.[10]

Certain weight changes occurred during training. On the regular hospital diet, 15 underweight patients gained an average of 3.6 pounds, 15 normal weight patients gained an average of 4.5 pounds and 10 overweight patients lost 5.6 pounds on a regular hospital diet.

Thirty patients showed grade changes in dyspnea and 20 had changes in ventilatory function as shown on Table 2.

After the six-week program, the patients were interviewed. The subjective improvement reported by the patients was one of the most important factors in the motivation of the patients. This interview indicated that the patients felt better and enjoyed the exercise program and the increased physical activity. They could breathe easier, they worried less about their conditions, they felt their food tasted better and their outlook became more optimistic.

Preliminary observations indicated that 80 per cent of the patients maintained or even improved their functional activity levels when observed three, six, nine and twelve months after the six-week training period.

Discussion

Barry[5] reported similar improvement in work capacity in post-coronary patients who were trained in a way similar to that described in the present report. Direct supervision helped maintain the patients' motivation[2,5,6] and prevented over-indulgence of exercises.

Physical fitness may be defined as the ability to maintain certain cardiorespiratory functions as

TABLE 6.—*Variables in Function Before and After Exercise Training*

Variables First	*Variables* Second	*Mean of First Variable*	*Mean of Second Variable*	*N*	*T*
1	2	224.43	425.21	37	3.05†
3	4	1.83	2.86	37	5.29†
5	6	59.56	67.02	37	1.60 N.S.
7	8	161.64	233.82	34	4.63†
9	10	77.91	148.22	36	7.51†
11	12	100.38	164.19	36	6.63†
13	14	62.51	72.81	37	4.56†
15	16	70.18	78.00	37	3.68†
17	18	2.59	1.40	37	−4.68†
19	20	21.10	38.59	37	7.63†
21	22	0.53	1.54	34	4.55†
23	24	2.58	2.33	39	−2.51*

First initial measurements. Second after six weeks of training.
N=Number of paired measures. T=Student's "T" test.
N.S.=Means non-significant difference between means.
*=Means are significantly different at the .05 probability level.
†=Means are significantly different at the .01 probability level.
1 vs 2—Time in seconds—walking on level.
3 vs 4—Number of standard flights—stair climbing ability (1 flight=20—7 inch risers and 1 landing mid-flight).
5 vs 6—Number in seconds—stair climbing.
7 vs 8—Time in seconds Exerow can be tolerated.
9 vs 10—Treadmill, 15 per cent grade-peds walked.
11 vs 12—Treadmill time in seconds.
13 vs 14—Left hand grip (pounds).
15 vs 16—Right hand grip (pounds).
17 vs 18—Number of blows to transfer 1 liter of water.
19 vs 20—Longest single blow in seconds.
21 vs 22—Expansion changes measured at navel line (inches).
23 vs 24—Emphysema grade changes.
Information in this table prepared by the Western Research Support Center, Veterans Administration, Sepulveda, California.

closely as possible to the resting state during exertion and to recover promptly from any disturbed function[5] induced by exercise. Walking reflects physical fitness to some degree when the maximum speed at which the patient walks for a specific period and the length of time a patient walks at a particular speed are considered.[7] Physical fitness of the COPE patients, as measured by functional activities, was improved and efficiency in performing physical work was increased by carrying out a combined exercise program. In addition, patients showed increased capacity for work as demonstrated by their ability to walk farther and climb more steps with increased speed, to tolerate rowing machine and occupational therapy activities and the like. Speeds that could not be tolerated before training were sustained after training. All these observations, including the objective measurements and the subjective improvement reported by the patients, indicate that a prescribed exercise program is beneficial and a worthwhile supplement to medical therapy. Graded exercise has a definite place in the routine care of chronic obstructive pulmonary emphysema.

REFERENCES

1. A.M.A. News, 27 June 1966.
2. Asthma Research Council, 28 Nortfolk Place, London W.2: Exercises for asthma and emphysema, third revision, 1962.
3. Barach, A. L.: Management of pulmonary emphysema, Modern Medicine, 29:18-91, August 1961.
4. Barach, A. L.: The value of oxygen inhalation; Exercise and trained breathing in the treatment of pulmonary emphysema, J.A.M.A., 185:30-32, 7 July 1963.
5. Barry, A. J., Daly, J. W., Pruett, E. D. R., Steinmetz, E.: Effect of physical training in patients who have had myocardial infarction, Am. J. of Card., 17:1-9, January 1966.
6. Committee on Rating of Mental and Physical Impairment. The respiratory system, J.A.M.A., 194:928-929, 22 November 1965.
7. Darling, R. C.: The significance of physical fitness Arch Phys. Med., 28:140, 1947.
8. Farber, S. M., and Wilson, R. J. L.: Pulmonary emphysema, Clinical Symposia (Ciba), 10:171, November-December 1958.
9. Haas, A., and Luczak, A.: The importance of rehabilitation in the treatment of chronic pulmonary emphysema, Arch. Phys. Med., 42:734-739, November 1961.
10. Henry, R. M., and Berg, W. E.: Physiological and performance changes in athletic conditioning, J. Appl. Physiol., 3:103, 1950.
11. Lyons, A. H.: Treatment of emphysema, J.A.M.A., 194:1234-1236, 13 December 1965.
12. Miller, W. F.: Physical therapeutic measures in the treatment of chronic bronchopulmonary disorders, Amer. J. Med., 24:929-939, 1 June 1958.
13. Noehren, T. H.: Pulmonary emphysema, J.A.M.A., 182:889-893, 1 December 1962.
14. Pierce, A. K., Taylor, H. F., Archer, R. K., and Miller, W. F.: The response to exercise training in patients with emphysema, Arch. Int. Med., 113:78-86, January 1964.

Aortography in the Diagnosis of Cancer

WILLIAM WEIDNER, M.D., *Torrance*, GABRIEL WILSON, M.D., AND
JOHN RILEY, M.D., *Los Angeles*

■ *The role of angiography in the diagnosis of cancer includes the direct visualization of the tumor, the demonstration of its size, extent and possible metastasis. The criteria which are used to judge the prognosis of a tumor include the size, angiographic appearance, relationship to adjacent organs and tissues, growth or extension into draining veins and the presence or absence of metastasis. Chemotherapeutic agents can be instilled through the same catheters that are used for angiography and these catheters may be left in place from five to eight days.*

ADVANCES IN ANGIOGRAPHIC techniques offer the means by which a neoplasm can be visualized directly by opacification of the vessels supplying blood to it. Radiologists no longer have to rely upon indirect changes seen on conventional roentgenograms but rather can demonstrate the neoplasm itself. The role of aortography is not only to establish the diagnosis but also to provide information for the subsequent management of the patient.[2]

Angiography of the Tumor

Not only the presence of a tumor but also its extent, the nature of its vascular supply and possible metastasis can be confirmed at angiography. At times, the angiographic findings of the primary or metastatic tumor may be so specific as to suggest the histologic type.[2,3] When an investigation is undertaken, the angiographer is obligated to delineate the lesion if present, to outline its vascular supply and to determine if metastasis has occurred.

The techniques which are employed are usually modifications of the Seldinger method of percutaneous catheter angiography. In patients under the age of 55, the femoral arterial route is usually employed; for patients over this arbitrary age limit, the axillary artery is used. Frequently, aortography is performed as a preliminary scanning procedure to be followed by selective angiography after the tumor is demonstrated and the vascular supply partially outlined.

Visualization of the complex anatomical structure of a tumor is enhanced by selective angiography. The vascular patterns within the tumor are seen more clearly as the contrast material is delivered selectively and overlapping vessels arising from the aorta are not filled. Serial filming is essential for the arterial, capillary and venous phases of the tumor. Direct angiographic signs of a neoplasm are tumor neovascularization and tumor stain secondary to opacification of minute tumor vessels. Indirect signs are stretching and displacement of vessels due to the mass of the tumor tissue.[6]

The following criteria may be used for judging the prognosis of a tumor angiographically: The size, angiographic appearance, relationship to adjacent organs and tissues, growth or extension into draining veins and presence or absence of metastasis.

Associate Professor of Radiology (Weidner) and Assistant Professors of Radiology (Wilson and Riley) UCLA Center for Health Sciences, Los Angeles.

Submitted 5 July 1966.

Reprint requests to: Department of Radiology, Torrance Memorial Hospital, 1000 W. Carson Street, Torrance 90509 (Dr. Weidner).

Size

Generally speaking, the larger a tumor, the worse the prognosis, although there are exceptions to this rule.

Angiographic appearance

The vascularity of the tumor and the appearance of the vessels within it correlate roughly with the degree of malignancy. Irregular vessels, a poor capillary phase, pooling and laking of the contrast material in the vessels and early filling of veins are characteristic of the more malignant tumors.

Relationships to adjacent organs and tissues

If the growth of the tumor has extended directly into adjacent organs or tissues, the prognosis is much more unfavorable.

CASE 1.—A 61-year-old Hungarian man had an exploration of the left kidney in Budapest in September 1963. An inoperable tumor of the inferior pole of the kidney was found and x-ray therapy was given. The patient did well until June 1965, when hematuria and left flank pain developed. When an intravenous pyelogram was done, it was observed that there was no function of the left kidney but calcification was present in the lower pole.

Selective left renal angiography was done and a large vascular tumor mass was present on the lower half of the kidney. The vessels were irregularly dilated, there was a poor capillary phase and there was pooling and laking of contrast material in the vessels (Figure 1). The inferior margin of the kidney was indistinct in the capillary phase of the angiogram (Figure 2). This was interpreted as indicative of extension into the perirenal tissues.

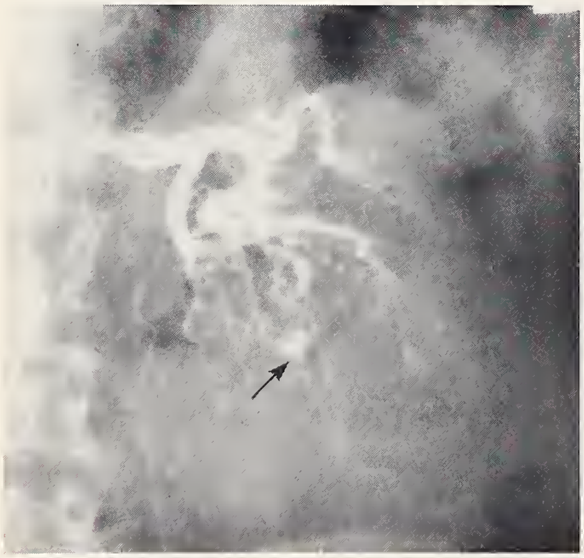


Figure 1. (Case 1)—Arterial phase, selective left renal arteriogram. There is a large neoplastic mass involving the entire lower half of the kidney. Neovascularization of the tumor is present and there is an early tumor stain (arrow). The vessels to the inferior pole are stretched and displaced by the mass of tumor tissue.

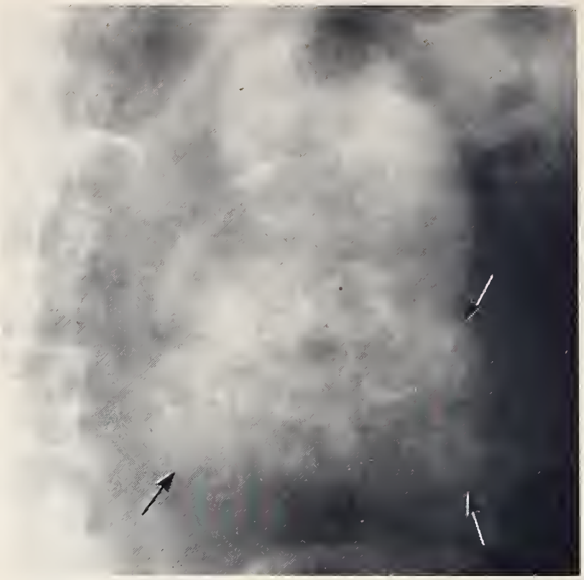


Figure 2. (Case 1)—Capillary phase, selective left renal arteriogram. The inferior margin of the left kidney is indistinct and the tumor stain extends into the perirenal tissues (arrows). This was confirmed at operation.

At operation an adenocarcinoma of the kidney was found. This tumor extended through the renal capsule and involved the retroperitoneal tissues. Three months later the patient died with pulmonary and cerebral metastatic lesions.

Extension of kidney tumors into the perirenal soft-tissues can occasionally be demonstrated by selective lumbar arteriography (Figure 3).

Extension into draining veins

CASE 2.—For five months a 26-year-old man had swelling of the left testicle, which gradually doubled in size. Left orchiectomy and left groin dissection were carried out. The tumor proved to be teratocarcinoma with areas of embryonal carcinoma and foci of chorial carcinomatous elements. He was referred to UCLA for further therapy and, as part of the work-up, inferior vena cavography was done. This revealed a polypoid filling defect in the inferior vena cava below the level of the renal veins and complete obstruction of the inferior vena cava just above the renal veins (Figure 4). On the basis of venous invasion by the tumor, it was elected to treat him with chemotherapy but he had no response and died within two months.

Occasionally, it is not possible to see the venous drainage of a tumor when arteriography is performed. If this occurs, selective catheterization of

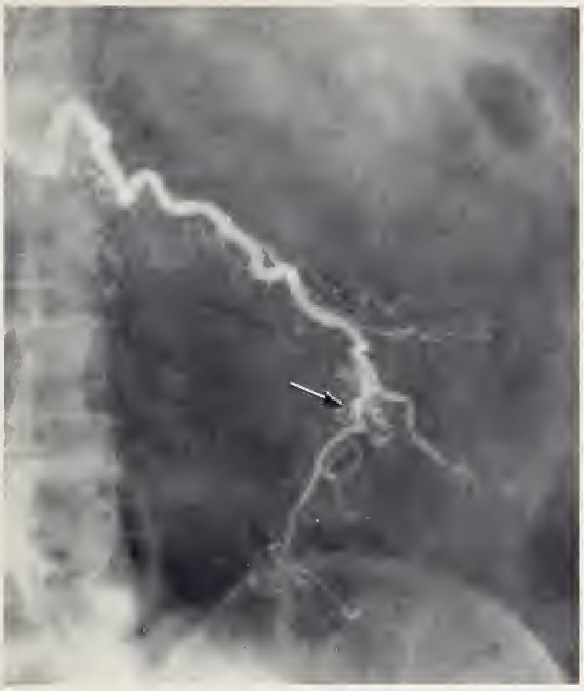


Figure 3.—Selective lumbar arteriogram in a 53-year-old man with adenocarcinoma of the left kidney. There is pronounced hypertrophy of the lumbar artery and an early tumor stain is seen in the perirenal tissues (arrow) indicating extension beyond the kidney.

the major veins draining the lesion can be performed in order to determine whether they are free of tumor.

Presence or absence of metastasis

Tumors which have metastasized generally have a much poorer prognosis.

The management of a tumor is directly related to its location, extent and whether metastasis has occurred. Selective angiography can demonstrate infiltration of a tumor into adjacent tissues. This can drastically affect therapeutic management. For example, angiography has been used to determine the stage of tumors of the bladder before treatment.[4,8]

Hepatic arteriography is particularly valuable in the evaluation of metastatic disease. A number of malignant lesions of the gastrointestinal tract extend by metastasis only in the liver. Advances in surgical technique have extended the indications for hepatic operations, and resections are now performed for primary and metastatic tumors in the liver. Mapping the blood supply helps the surgeon plan his procedure and in addition outlines the possibilities for perfusion chemotherapy.[2,3]

The following case demonstrates the use of angiography to outline the extent of a tumor in the liver before operation.

CASE 3.—A 47-year-old man with a history of alcoholism began to have signs of cirrhosis and hepatic failure six or seven years before admission. He stopped drinking. Two months before admission, discomfort in the right upper quadrant of the abdomen developed. Laparotomy was carried out and cholecystectomy was contemplated until it was found that a large hepatic tumor seemed to occupy the entire right hepatic lobe. Biopsy was done and the lesion was identified as a cholangiocarcinoma. The patient was referred for examination to determine if the left lobe was involved with tumor. A liver scan demonstrated only involvement of the right lobe. Angiography showed extensive tumor throughout the entire right lobe of the liver but the left lobe appeared to be free of the lesion (Figure 5). Right hepatic lobectomy and resection of part of the medial aspect of the left lobe were carried out. Hepatic coma developed and the patient died.

Angiographically, this tumor is interesting because it has a vascular cholangiocarcinoma and could not be differentiated from a hepatoma on the basis of angiographic appearance. Cholangiomas previously have been reported as relatively avascular tumors.[5]

Using clinical symptoms and signs, primary tumors of the liver cannot consistently be differentiated from metastatic lesions or cirrhosis. Hepatic angiography is a method by which hepatomas can be differentiated from metastatic disease. They are both supplied by the hepatic artery but there are differences in the angiographic appearance. Hepatomas are characterized by dilatation

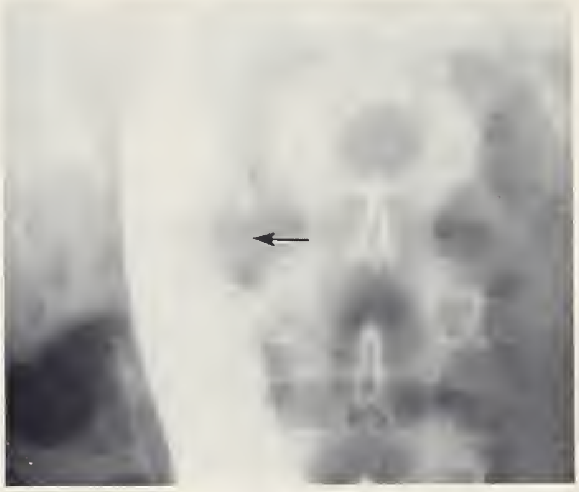


Figure 4. (Case 2)—Inferior vena cavogram. There is a polypoid filling defect due to a tumor implant in the inferior vena cava below the level of the renal veins (arrow). The inferior vena cava is obstructed by tumor implants just above the renal veins.

of the hepatic artery, displacement of dilated intrahepatic arteries, tortuous tumor vessels, prolongation of the circulation time in the tumor and occasionally arteriovenous shunting. Metastatic lesions may have a rich blood supply in the periphery but usually are relatively avascular.[5]

Perfusion Therapy

Chemotherapeutic agents have a limitation in their application because they influence normal cellular metabolism as well as cancer cells. This limits the dosage. If the agent can be delivered selectively to the vascular bed of a tumor, the concentration of the drug can be greatly increased, enhancing its effect on the tumor and reducing the systemic effects. For example, the liver has a relatively isolated blood supply and tumors in the liver can be perfused by placing a catheter in the hepatic artery.[1] An added advantage of hepatic artery infusion is the fact that normal liver cells detoxify the compound and reduce its systemic effects.[7]

Various techniques have been employed for intra-arterial perfusion chemotherapy in cancer. Extracorporeal perfusion has proved to be disappointing in its results. There now is considerable interest in continuous intra-arterial perfusion through an indwelling arterial catheter. These catheters may be placed surgically in the vessel supplying a tumor or may be inserted percutaneously and directed into the vessel under fluoroscopic control. The percutaneous method has the advantage of obviating the need for a surgical procedure, but at times it may be difficult or impossible to direct a catheter into a distorted or displaced vessel supplying a tumor.

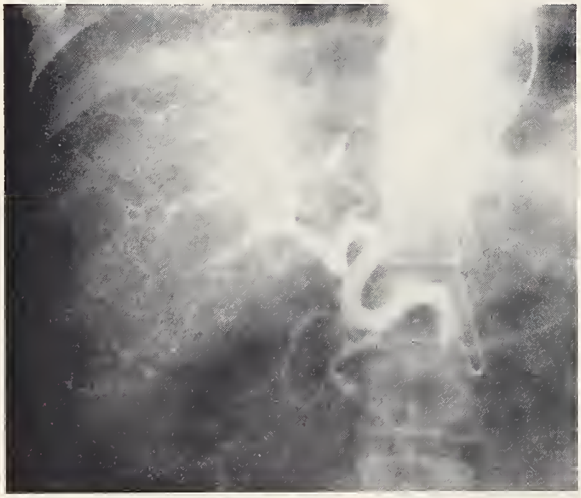


Figure 5. (Case 3)—Selective hepatic angiography. There is a large vascular cholangioma occupying the entire right lobe of the liver. The intrahepatic arteries are dilated and tortuous. The entire tumor is vascularized and the vessels are irregular.

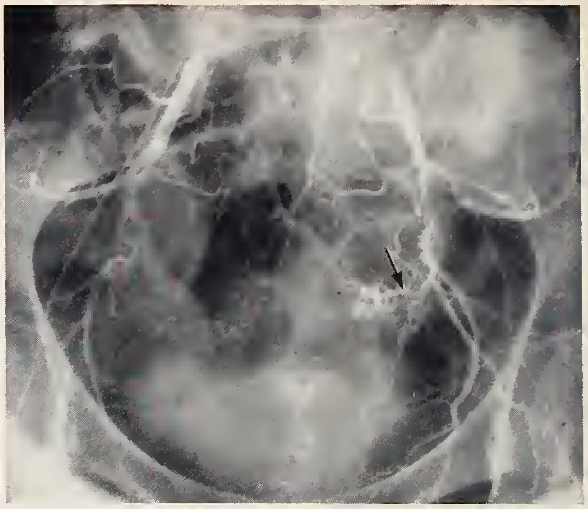


Figure 6. (Case 4)—Bilateral selective hypogastric arteriography. There is hypertrophy and tortuosity of the left uterine artery (arrow) and hypertrophy of the vaginal arteries supplying the recurrent tumor.

Before infusion procedures arteriography should be performed to delineate the vessel supplying the tumor. Unsuspected anomalies, displacements or occasionally thrombosis of the vessel may drastically alter perfusion planning.

Initially preformed polyethylene catheters were used. They were placed in the hepatic, hypogastric, internal carotid and external carotid arteries for perfusion therapy of primary and metastatic tumors of the liver, pelvis, head and neck. After they were inserted, heparin was administered and the catheters were left in place for several days. Occasionally, a thrombus formed on the polyethylene catheter. Teflon catheters were then substituted and the incidence of thrombi was reduced. With heparinization, the catheters may be left in place for five to eight days. When the catheter is introduced via the axillary artery, the patient has more mobility during perfusion therapy than when the femoral artery is used.

CASE 4.—A 42-year-old woman had a transitional cell carcinoma of the vagina with metastasis to the pelvis and periaortic nodes. Radiation therapy was given but a vesicovaginal fistula and a rectovaginal fistula developed and the primary tumor recurred. Percutaneous pelvic arteriography was performed to determine the extent of recurrent tumor (Figure 6). The catheters in the hypogastric arteries were left in place for eight days while she received 50 mg of methotrexate daily with 6 mg citro varum factor. At the conclusion of chemotherapy, the catheters were removed.

Later, when an intravenous pyelogram was done the left renal collecting system was not visualized and a right cutaneous ureterotomy and ligation of the left ureter were done. The patient had considerable relief of pelvic pain following this procedure and was discharged ambulatory and using a Lapides bag without leakage. She died six months later with extensive metastasis.

REFERENCES

1. Ariel, I. M., and Pack, G. T.: Intra-arterial chemotherapy for cancer metastatic to liver, Archives of Surgery, 91:851-862, November 1965.

2. Baum, S., Kuroda, K., Roy, R. H.: The value of special angiographic techniques in the management of patients with abdominal neoplasms, The Radiologic Clinics of North America, 3:583-599, December 1965.

3. Baum, S., Roy, R., Finkelstein, A. K., Blakemore, W. S.: Clinical application of selective celiac and superior mesenteric arteriography, Radiology, 84:279-295, February 1965.

4. Boijsen, E., and Nilsson, J.: Angiography in the diagnosis of tumors of the urinary bladder, Acta Radiologica, 57:241-258, July 1962.

5. Boijsen, E., and Abrams, H. L.: Roentgenologic diagnosis of primary carcinoma of the liver, Acta Radiologica, 3:257-277, May 1965.

6. Nebesar, R. A., Pollard, J. J., Stone, D. L.: Angiographic diagnosis of malignant disease of the liver, Radiology, 86:284-292, February 1966.

7. Sullivan, R. D., Norcross, J. W., Watkins, Jr., E.: Chemotherapy of metastatic liver cancer by prolonged hepatic artery infusion, New Engl. J. Med., 270:321-327.

8. Taylor, D. A., Macken, K. L., Vennema, R. J., Bachman, A. L.: A preliminary report of a new method for the staging of bladder carcinoma using a triple contrast technique, Brit. J. Radiol., 38:667-672, September 1965.

Occult Glucose Intolerance: Incidence In a General Population

RONALD L. SEARCY, PH.D., *Nutley, New Jersey,* AND EDMUND M. Y. LOW, M.D., *Los Angeles*

■ *A survey of two-hour postprandial glycemic responses to an oral dose of 100 gm of glucose was conducted in 456 appparently healthy adult members of a small Southern California community. Serum glucose levels of 32 per cent of the men and 36 per cent of the women exceeded 140 mg per 100 ml at the two-hour sampling period.*

In both sexes, there was a tendency for hyperglycemia to become more conspicuous with age.

Under the age of 50, a much higher percentage of women than men had seemingly excessive serum glucose values.

These findings may suggest a need for modifying criteria currently used for interpreting glycemic responses to oral glucose challenges.

AS EARLY AS 1920, Spence[14] called attention to a decline in glucose tolerance that was associated with aging. A number of subsequent studies confirmed this observation in elderly non-diabetic subjects.* Recent evaluations showing the prevalence of postprandial hyperglycemia in rest home[15] and community[6] populations have focused attention on the possible implications of glucose intolerance, particularly in elderly persons. This renewed interest prompted us to conduct a survey of glycemic responses of members of a suburban community to ingestion of 100 gm of glucose dissolved in fruit-flavored gelatin. It is the purpose of this report to present the data derived from this study which, in view of the high frequency of apparent glucose intolerance, suggest a need for modifying certain criteria employed for interpreting glycemic responses.

Methods and Materials

The populace of a relatively small city in Southern California was invited to participate in a series of diabetic detection clinics to be held in a local hospital. Persons with suspected or diagnosed diabetes mellitus were excluded from the study.

All volunteers were instructed to fast for at least four hours before attending the clinics, where they were asked to consume 225 ml of chilled cherry-flavored gelatin containing 100 gm of glucose.* They were encouraged to ingest the semi-solid mixture within five minutes. Blood was collected two hours thereafter, and the serum was separated from the samples within 30 minutes. The specimens were assayed for "true" glucose by an automated procedure involving use of glucose oxidase.[11]

From the Departments of Pathology, Los Angeles County General Hospital (Unit 2), Los Angeles, and University of California-California College of Medicine, Los Angeles.

This work was supported in part by funds from the Research Division of Uni-Tech Chemical Manufacturing Company, Sun Valley, California.

Submitted 8 September 1966.

Reprint requests to: Research Division, Hoffman-La Roche Inc., Nutley, New Jersey 07110 (Dr. Searcy).

*Reference Nos. 1-5, 7, 9, 12, 13.

*Gel-a-dex, a product of Uni-Tech Chemical Manufacturing Company, Sun Valley, California, was used.

After the blood collection, a majority of the participants were interviewed individually. Each was asked a uniform series of questions that were designed specifically to disclose a familial history of diabetes mellitus.

Results

Glycemia Before Glucose Challenge. At one clinic session, blood specimens were collected from 88 volunteers before ingestion of the test meal. Analysis of the specimens showed that 84 of the subjects had base line serum glucose levels ranging from 40 to 100 mg per 100 ml. The two-hour postprandial glucose levels were compared with the base line values (Chart 1). Of the 42 subjects with initial glucose levels between 40 and 60 mg per 100 ml, only 15 had postprandial glycemia higher than 100 mg per 100 ml. By contrast, 24 out of a group of 42 volunteers with initial serum glucose levels ranging from 60 to 100 mg per 100 ml had postprandial glycemia in excess of 110 mg per 100 ml.

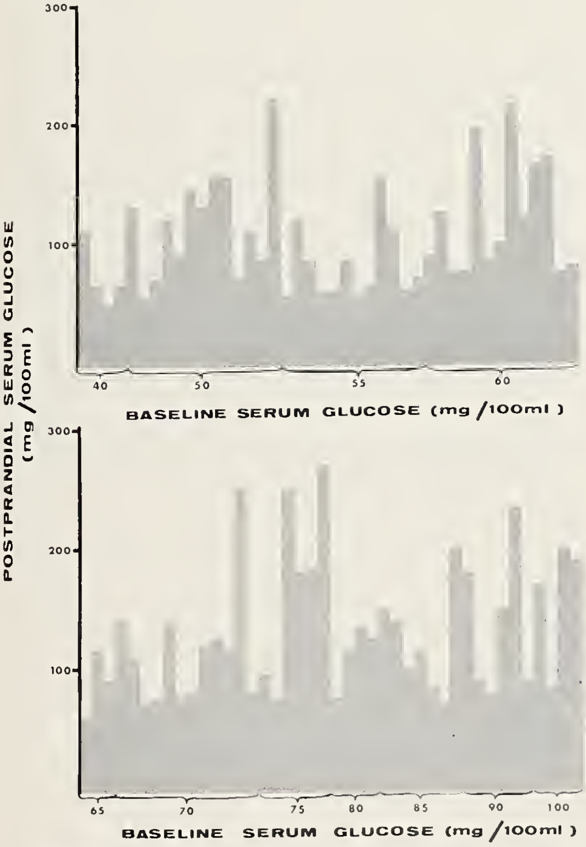


Chart 1.—Upper chart shows the two-hour glycemic responses of subjects exhibiting serum glucose levels between 40 and 60 mg per 100 ml; lower chart gives similar data for those with values between 65 and 100 mg per 100 ml.

The postprandial glucose levels in the subjects with the lower fasting levels averaged 102.9±45.2 mg per 100 ml (range 50 to 220 mg per 100 ml). A mean of 129.2±55.6 mg per 100 ml (range 70 to 270 mg per 100 ml) was obtained for the group of subjects with higher base line glycemia. Statistical analysis of the data disclosed that the difference in glycemic responses of the two groups was significant ($P = < 0.02$).

Of the 88 persons tested at this session of the clinic, four had base line serum glucose values of 120, 125, 135 and 225 mg per 100 ml; after challenge with the test meal, their serum glucose levels rose to, respectively, 150, 160, 450 and 400 mg per 100 ml.

Glycemia After Glucose Challenge. Postprandial glucose values were obtained for 150 men and 306 women (Chart 2). In the entire study group, approximately 35 per cent (159 of 456) had two-hour postprandial glucose values higher than 140 mg per 100 ml. When categorized according to sex, about 32 per cent of the men (48 of 150) and 36 per cent of the women (111 of 306) had postprandial serum glucose values above 140 mg per 100 ml.

In both sexes there was a general trend for the frequency of hyperglycemic responses to increase with age. This tendency was, however, much more conspicuous in the women than in the men (Table 1). For example, only 45 per cent of the women below the age of 50 had postprandial serum glucose levels below 110 mg per 100 ml, whereas slightly more than 70 per cent of the men in the same age group had normal degrees of glycemia. Quite similar percentages of seemingly abnormal glucose levels were noted in both men and women over the age of 50.

Familial History. A total of 251 women and 117 men were questioned to determine consanguineous incidence of diabetes mellitus. Thirty-nine per cent of the women and 31 per cent of the

TABLE 1.—*Data on Results of Ingestion of 100 gm of Glucose in Gelatin*

Sex	*Number of Subjects*	*Age (Years)*	*Two-Hour Postprandial Serum Glucose Levels* *<110 mg/100 ml*	 *>110 mg/100 ml*
Male	57	<50	40 (71)*	17 (29)
	93	>50	37 (40)	56 (60)
Female	110	<50	52 (45)	58 (55)
	196	>50	77 (39)	119 (61)

---

*Parenthetical numbers indicate the per cent of the total number of subjects in each category.

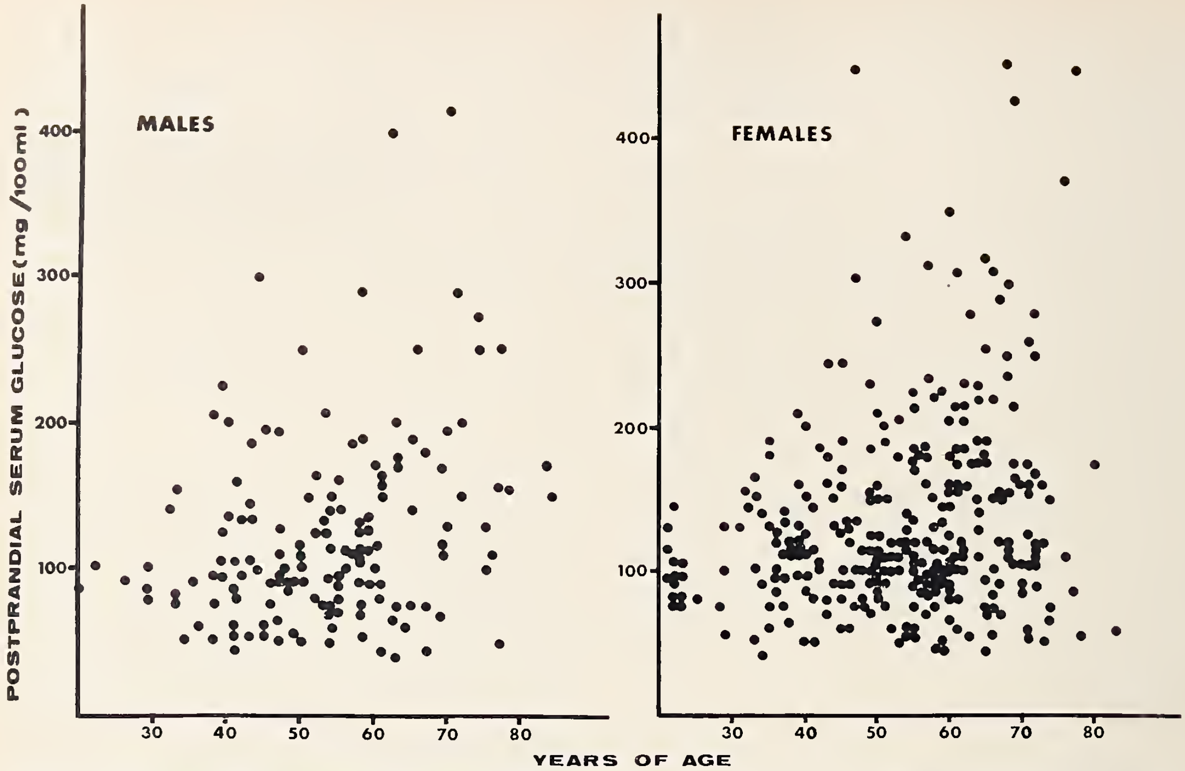


Chart 2.—Postprandial serum glucose levels as related to age. Chart at left is for 150 apparently healthy males, and the chart at the right is for 306 apparently healthy females.

men had blood relatives with diabetes mellitus (Table 2). In both sexes the mothers were found to be the relative most often reported to have had the disease.

Discussion

Examination of the serum glucose levels before the test meal was administered suggested that most of the participants at that time were in the post-absorptive state. (Perhaps they were at the other sessions, as well.) Of particular interest is the fact that there appears to be a significant relationship between the base line glucose level and the glycemic response to the oral glucose challenge. Although 84 subjects had initial serum glucose concentrations that would be considered within normal limits, those with levels above 60 mg per 100 ml showed a higher frequency of hyperglycemia. Therefore, the post-absorptive serum glucose value may be a very subtle indicator of an individual's capacity to respond to a glucose challenge. As base line values approach the upper limits of normal, there is a greater likelihood of a high postprandial response to oral glucose.

Since the test was first introduced by Jacobson[8] more than half a century ago, glucose loading has been an accepted method for assessing carbohydrate tolerance. There has been some disagreement, however, as to the interpretation of the results of oral glucose tolerance tests. A group of authorities[17] have concluded, for example, that two-hour postprandial serum glucose values exceeding 120 mg per 100 ml should be regarded as suggestive evidence of diabetes mellitus. Other investigators[10] have considered values as low as 100 mg per 100 ml at the two-hour interval after

TABLE 2.—*Family History of Diabetes Mellitus (From Interviews)*

Sex	*Number of Subjects Interviewed*	*Number of Subjects with Familial History of Diabetes Mellitus*	*Relationship of Diabetic Member**								
			F	*M*	*B*	*S*	*Gm*	*Gf*	*A*	*U*	*C*
Male	117	36	5	12	4	3	4	0	6	6	2
Female	251	99	16	26	15	18	20	3	24	16	9

*F=Father, M=Mother, B=Brother, S=Sister, Gm=Grandmother, Gf=Grandfather, A=Aunt, U=Uncle, C=Cousin.

glucose ingestion as a presumptive sign of the disease. Our findings support the suggestion[16] that either of these limits would place an alarmingly high proportion of the general population under suspicion of having diabetes mellitus. In our survey, over 30 per cent of the participants had two-hour postprandial serum glucose values above 140 mg per 100 ml. This may be construed as evidence that there is a need for modifying the criteria currently advocated for interpretation of glucose tolerance tests.

There appears to be a conspicuous tendency for apparently healthy women under the age of 50 to have higher degrees of postprandial glycemia after glucose challenge than men of corresponding age. The factors responsible for this sex-linked difference in glucose tolerance remain obscure. It is tempting to speculate that prevalence of glucose intolerance in the premenopausal woman may be related to hormonal mechanisms. Pregnancy during the childbearing years could possibly burden the endocrine pancreas excessively and render the female less capable of efficiently utilizing an oral glucose load.

In healthy men and women, aging is associated with a greater frequency of what might be interpreted as abnormal glycemic responses. Incidences of impaired oral glucose tolerances ranging from 53 to 100 per cent have been reported in seemingly healthy elderly subjects.[5,9] This apparent defect in responding to a glucose challenge may be attributed to several factors. Abnormal results may reflect an impairment in the intestinal absorption of the administered glucose or a diminished capacity of the pancreas to secrete adequate amounts of insulin. Streeten and coworkers[15] recently concluded that the hyperglycemic tendency in elderly subjects is not due to either of these defects, but rather to the presence of inordinate levels of circulating insulin antagonist.

REFERENCES

1. Brandt, R. L.: Decreased carbohydrate tolerance in elderly patients, Geriatrics, 15:315, 1960.

2. Chesrow, E. J., and Bleyer, J. M.: The glucose tolerance test of the aged, Geriatrics, 9:276, 1954.

3. Chesrow, E. J., and Bleyer, J. M.: Diabetes case finding among 1,000 patients over 60, Geriatrics, 10:479, 1955.

4. Deren, M. D.: Dextrose tolerance in the aged, J. Lab. Clin. Med., 22:1138, 1937.

5. Gottfried, S. P., Pelz, K. S., and Clifford, R. C.: Carbohydrate metabolism in healthy old men and women over 70 years of age, Amer. J. Med. Sci., 242:475, 1961.

6. Hayner, N. S., Kjelsberg, M. O., Epstein, F. H., and Francis, T., Jr.: Carbohydrate tolerance and diabetes in a total community, Tecumseh, Michigan. I. Effects of age, sex, and test conditions on one-hour glucose tolerance in adults, Diabetes, 14:413, 1965.

7. Horvath, S. M., Wisotsky, R., and Carwin, W.: The oral glucose tolerance test in old men, J. Geront., 2:25, 1947.

8. Jacobson, T. B.: Utersuchungen über den einfluss des Chloralyhydrats auf experimentelle Hyperglykämieforemen, Biochem Z, 51:443, 1913.

9. Marshall, F. W.: Sugar content of the blood in elderly people, Quart. J. Med., 24:257, 1930-31.

10. Mosenthal, H. O., and Barry, E.: Criteria for and interpretation of normal glucose tolerance tests, Ann. Intern. Med., 33:1, 175, 1950.

11. Saifer, A., and Gerstenfeld, S.: The photometric microdetermination of blood glucose with glucose oxidase, J. Lab. and Clin. Med., 51:448, 1958.

12. Schneeberg, N. G., and Finestone, I.: Effect of age on the intravenous glucose tolerance test, J. Geront., 7:54, 1952.

13. Smith, L. E.: Glucose tolerance in the aged, J. Geront., 3:66, 1948.

14. Spence, J. C.: Blood sugar tolerance and age, Quart. J. Med., 14:314, 1920-21.

15. Streeten, D. H. P., Gerstein, M. M., Marmor, B. M., and Doisy, R. J.: Reduced glucose tolerance in elderly human subjects, Diabetes, 14:579, 1965.

16. West, K. M.: Laboratory diagnosis of diabetes, a reappraisal, Arch. Int. Med., 117:187, 1966.

17. Wilkerson, H. L. C.: Diabetes Mellitus: Diagnosis and Treatment, N.Y., Am. Diabetes Assoc., p. 33, 1964.

The Obese Person as an Adolescent

DALE C. GARELL, M.D., *Los Angeles*

■ *Obesity constitutes a major health problem in the United States. Hypertension, atherosclerosis, coronary artery disease, diabetes and gout are often associated with obesity and may be a direct result of persistent obesity in adult life.*

Obesity frequently has its beginnings in childhood and adolescence. Unfortunately, obesity which develops in early life is a progressive problem. Eighty per cent of overweight children and adolescents will continue to be overweight as adults.[12] *Furthermore, adults with a history of obesity in childhood are the most resistant to treatment.*

Recent studies have shown there is more than one body constitutional type among obese adolescents. Obese adolescents tend to eat less than nonobese controls. While obesity may be found to have many different causative factors, efforts to control this disease may be most successful in the area of primary prevention.

IN ORDER TO GAIN perspective about obesity in the adolescent age, it is important to be aware of several basic facts about normal adolescence.

Adolescence is the final and second most rapid period of growth. It is also a time when male and female sex hormones begin to exert their effect upon human growth and development. Age as a time clock, however, is not a good index of biological growth and development. Several other measurements are important in determining this growth. They include anthropometry, the measurement of various parameters of body growth, assessment of height and weight, skeletal maturation as measured by bone age on x-ray examination and an estimate of sexual maturation as developed by Greulich.[18]

A frequent misconception is that menarche heralds the beginning of adolescent growth in the female. In fact, it occurs almost near the end of longitudinal growth and sexual maturation. There is good evidence to suggest that adolescent females, in most instances, begin their growth spurt before the ninth birthday. In males the spurt of growth begins approximately two years later. Recent evidence by Cheek[4] suggests that the onset of adolescent growth may occur even earlier. Lean body mass begins to increase in the female at six years of age and in the male at about the ninth year. In some cases, however, adolescent growth in quite normal persons may be delayed until the fourteenth or fifteenth year; in these instances, normal growth may not be completed until the beginning or middle of the third decade of life.

Adolescents vary physiologically in their individual rate of growth. There are often striking differences in height, weight and body build among persons who are approximately the same age. There may also be variations in growth within any one individual.

From The Division of Adolescent Medicine, Childrens Hospital of Los Angeles, Los Angeles.

Presented at USC School of Medicine Symposium on Obesity, 10 March 1966, Los Angeles.

Submitted 13 April 1966.

Reprint requests to: Director, Division of Adolescent Medicine, Childrens Hospital of Los Angeles, 4614 Sunset Boulevard, Los Angeles 90027.

Finally, growth during adolescence includes psychological maturation. The developmental tasks of adolescence—the mastery of self-identity, the resolution of problems of independence and acceptance of the rules of society, the development of sexual identity, the ability to conceptualize and the attainment of vocational goals—are important factors in psychological growth during adolescence.

Natural History of Obesity

Obesity constitutes a major clinical problem in clinics for adolescents throughout the United States.[7] Studies by Johnson[11] indicate that approximately 12.5 per cent of adolescent girls and 9.5 per cent of adolescent boys are overweight. In a study recently completed by Rauh in Cincinnati[13] a similar prevalence of approximately 10 per cent in the adolescent age population was noted.

Genetic and environmental factors indicate an 8 to 10 per cent incidence of obesity in children with no family history of this disorder. If one parent is overweight, there is a 40 per cent chance of obesity; if both parents are overweight, 80 per cent.[11]

Work by Heald[8] and others[1,20] showed that there is no apparent difference in birth weight between the obese and non-obese. However, Heald[8] reported that obese adolescent girls were already heavier than the average at one year of age. Juvenile obesity seems to have its peak onset at 0 to 4 years of age and again at 7 to 11 years. There is some suggestion that obese adolescents grow at a more rapid rate, although it is difficult to determine whether this represents a more rapid growth or a more rapid maturation. Studies of menarche in obese and non-obese girls have shown no significant differences.

Seltzer and Mayer,[15] studying the somatotypes and anthropometric measurements, showed that there are differences in somatotypes in obese subjects in addition to obesity as measured by triceps skin folds. Obese adolescent girls, apart from being more endomorphic, appear to be more mesomorphic and decidedly less ectomorphic than non-obese controls. Ectomorphs are rarely obese.

Current Research

Eighty per cent of adipose tissue is composed of triglycerides, which serve primarily as a reservoir for fuel. Insulin has been found to play a key role in lipogenesis, the formation of triglyceride and its storage in adipose tissue. In lipolysis, 11 individual hormones, including growth hormone, act upon triglyceride to release fatty acids. Insulin and growth hormone are antagonistic to one another: Insulin promotes lipogenesis and blocks the release of free fatty acids, while growth hormone inhibits lipogenesis and enhances lipolysis. Three possible metabolic pathways may lead to the development of obesity in an individual: One may either synthesize triglycerides faster than normal or hydrolyze free fatty acids or inhibit mobilization of triglycerides.

In Table 1, current metabolic factors associated with obesity in adults are compared with similar factors in adolescents. Very little physiological and biochemical information is available on obese children and adolescents. It is not clear whether the metabolic changes observed in obese patients are a result of obesity or actually precede its development.

Forbes[6] recently studied lean body mass in 15 boys and 12 girls from 8 to 18½ years of age by means of total potassium data obtained by a whole-body scintillation count. His findings suggest that there are two types of obesity in childhood and adolescence. One-third of the obese patients studied were characterized by an increase in lean body mass as well as in fat, by a tendency to tallness, by advanced bone age and by a history of obesity since infancy. The remaining two-thirds showed no increase in lean body mass and normal growth characteristics, including height and bone age. In the latter patients obesity developed during childhood and adolescence. Further work may indicate a genetic defect in the smaller group, while in the larger group obesity may represent a progressive metabolic disorder.

TABLE 1.—*Metabolic Factors in Obesity, Adults as Compared with Adolescents*

	Obese Adults	*Obese Adolescents*
Fasting Free Fatty Acid	Elevated	Elevated
Response to Epinephrine	None (19 Per Cent)	Normal (Lipid Mobilization)
Hyperinsulinism	Present	Unknown
Response to Exogenous Insulin	Resistant	Resistant
Response to Fasting	No Elevation In HGH	Unknown
Elevated Cortisol	Present	Unknown
Resistance to Ketosis	Present	Present

Nutrition of Adolescents

Studies by Johnson[11] have shown that, contrary to common clinical impressions, obese adolescent girls eat significantly less than non-obese controls. Hueneman and associates[10] reported the dietary histories of 950 teen-agers in the Berkeley school system. By means of a longitudinal study, she has followed the same students from the ninth to the twelfth grade, making anthropometric measurements and obtaining careful dietary histories. The results are only preliminary at the present time, but they confirm previous prevalence data for obesity, as well as for a lower intake of calories in the very obese adolescent. The mean caloric intake for all ninth grade boys is 2,900 per day; for girls, 2,000. In contrast, obese boys eat 2,360 calories and obese girls 1,530. In the three-year period there were no significant changes in the percentage of obese and non-obese subjects, although body fat increases in both boys and girls. Finally, characteristic of the adolescents' concept of body size and shape, it was noted that 50 per cent of the boys and 65 per cent of the girls in the total study sample said they were trying to gain or lose weight or to change their body proportions.

Physical Activity

Whether because of physiological or psychological factors or both, physical activity is much less in obese adolescents than in the non-obese.[2] Studies have shown differences in attitudes toward physical activity: Obese patients were unaware of the degree of their inactivity. Psychological studies also revealed difficulties in separations from their families.[3] However, these attitudes toward inactivity in obese subjects are difficult to distinguish from other psychological components of obesity. Wenzel has suggested a possible physiological factor contributing to inactivity. In a study of obese youngsters in an adolescent clinic, a significantly lower serum iron was found than in normal non-obese controls.[19] Hemoglobin determinations were normal for both groups studied.

Atherosclerosis

While atherosclerosis and other long-term complications of obesity have been of little previous concern to pediatricians, Reisman[14] has pointed out the importance of these diseases in children and adolescents. Holman[9] described the development of fatty streaks in the aortas of infants under three years of age. During adolescence these streaks are converted into fibrous plaques which may subsequently be the sites of such complications as lipid accretion, hemorrhage and vascular stenosis in middle age or earlier and may give rise finally to infarction, cerebral vascular accident and peripheral vascular occlusion disease. Reisman also has called pediatricians' attention to autopsy studies showing that 50 per cent of American soldiers who died in Korea had grossly visible atheroma of the aortic wall; and in 10 per cent the lumen of at least one coronary artery was reduced to less than 50 per cent of the normal size.[5] The average age of the autopsy subjects was 22 years. From this it appears that heart disease is a progressive entity. This disease, particularly primary prevention, should be a shared responsibility of physicians who care for children and adolescents. Research in this area is long overdue.

Treatment

Finally, the treatment of obesity in adolescents should be mentioned briefly. In a comprehensive review of the literature of the last 30 years, Stunkard and McLaren-Hume[17] observed that most attempts to control obesity are ineffectual. This would seem also to be true in clinical experience with adolescents. The key lies not in weight loss but in prolonged maintenance of weight reduction. Recently, Shutter and Garell[16] studied the use of an anorexic drug, a dextro-amphetamine-barbital combination, in 78 children and adolescents. Significant factors contributing to initial weight loss in a 12-week period included the anorexic drug, the first six-week period of treatment, and the degree of motivation to lose weight. While 94 per cent of the subjects who completed the 12-week period lost weight at first, only two-thirds of them were maintaining the loss when examined six to twelve months after the completion of this study. These findings support the observation that outpatient treatment may have initial value but weight loss is not often sustained.

REFERENCES

1. Bruch, H.: Obesity in childhood. 1. Physical growth and development of obese children, Am. J. Dis. Child., 58:457, 1939.

2. Bullen, B., Reed, R. B., and Mayer, J.: Physical activity of obese and non-obese adolescent girls appraised by motion picture sampling, Am. J. Clin Nutr., 14:211, 1964.

3. Bullen, B., et al.: Attitudes towards physical activity, food and family in obese and non-obese adolescent girls, Am. J. Clin. Nutrition, 12:1, 1963.

4. Cheek, D.: Personal communication. In publication, Bull. Johns Hopkins, April 1966.

5. Enos, W. F., Beyer, J. C. and Holmes, R. H.: Pathogenesis of coronary disease in American soldiers killed in Korea, J.A.M.A., 158:912, 1955.

6. Forbes, G.: Lean body mass and fat in obese children, Pediatrics, 34:308, 1964.

7. Garell, D. C.: Adolescent medicine, Am. J. Dis. Child., 109:314, 1965.

8. Heald, F. P., and Hollander, R. J.: The relationship between obesity in adolescence and early growth, J. Ped., 67:35, 1965.

9. Holman, R. L., McGill, H. C., Jr., Strong, J. P., and Geer, J. C.: The natural history of atherosclerosis, Am. J. Path., 34:209, 1958.

10. Hueneman, R. L., Hampton, M. C., Shapiro, L. R., and Behnke, P.: Adolescent food practices associated with obesity, Fed. Proc., 25:4, part I, January-February 1960.

11. Johnson, M., Burke, B., and Moyer, J.: Relative importance of inactivity and overeating in the energy balance of obese high school girls, Am. J. Clin. Nutr., 4:37, 1956.

12. Lloyd, J. K., Wolff, O. H., and Whelan, W. S.: Childhood obesity. A long-term study of height and weight, Brit. Med. J., 5245:145, 1961.

13. Rauh, Joseph: Personal communication.

14. Reisman, M.: Atherosclerosis and pediatrics, J. Peds., 66:1, part I, January 1965.

15. Seltzer, C. C., and Mayer, J.: Body build and obesity—Who are the obese? J.A.M.A., 189:677, 1964.

16. Shutter, L., and Garell, D.: Obesity in children and adolescents: A double blind study with crossover, J. Am. School Health, 36:273, June 1966.

17. Stunkard, A. J., and McLaren-Hume, M.: The results of treatment for obesity, Arch. Int. Med., 103:79, 1959.

18. Tanner, J. M.: Growth at adolescence, Oxford, England, Blackwell Scientific Publications, 1962.

19. Wenzel, B. J., Stults, H., and Mayer, J.: Hypoferraemia in obese adolescents, Lancet, 2:327, 1962.

20. Wolff, O. H.: Obesity in childhood, Quart. J. Med., 24:109, 1955.

CORRECTION

An error was made in the redaction of an article on Dermatopathic Lymphadenopathy by Robert A. Cooper, M.D., and associates, which appeared in the March issue of CALIFORNIA MEDICINE. In the third line of the first paragraph on page 171, the abbreviation PAS was mistakenly extended to "para-aminosalycilic acid." On galley proof the author corrected the extension to read "periodic acid-Schiff," but through oversight the correction was not made before the article appeared in print.

Total Perineal Prostatectomy For Carcinoma

Indications and Results

M. J. FEENEY, M.D., R. B. MULLENIX, M.D., R. J. PRENTISS, M.D., AND
G. E. HOWE, M.D., *San Diego*

■ *In 34 selected patients with prostatic carcinoma treated with total perineal prostatovesiculectomy, the morbidity was low and mortality nil. Fourteen patients were alive and well for over five years and 12 patients are alive and well for less than five years at the time of report. Six patients were dead, two with prostatic carcinoma. Two of the survivors had recurrent carcinoma.*

Introduction

THE PRINCIPLES OF TOTAL perineal prostatovesiculectomy developed by Young[28,29] and improved upon by other American urologists[4,5,7,12,26] have been well described. Many urologists, however, rarely or never utilize this operation since other palliative methods are often effective. All are hopeful that additional methods of endocrine therapy,[2,3,8,19] chemotherapy, radiotherapy[9,10,17] or entirely new methods of treatment will be developed.

Total surgical extirpation of the neoplasm remains the only safe method of possible cure of prostatic cancer. In view of this, it seems appropriate to review a group of patients so treated.

Material

The group was made up of all the private practice patients in whom the author did total prostatectomy during the past 15 years. The average age and condition (surgical risk) of the 34 patients, as well as methods of diagnosis were as follows:

Average age		61 years
Range in age		46-71 years
Surgical Risk		
Good		29
Fair		5
Poor		0
Method of diagnosis		
Needle biopsy		21
Open perineal		11
Frozen	9	
Paraffin	2	
Transurethral resection		2

Methods

Spinal anesthesia was used for all patients. Blood transfusion was given to 18. The Belt[4] approach was used in 14 patients in the early part of the series and the Young[28] approach more recently in 20. The Palmer(a) board was used to gain complete perineal exposure. Rectal examination was made readily available by placement of the finger cot portion of the O'Conor shield.(b) Vi-drape(c) is now used to cover the surgical field. The Raney(d) electro-coagulating forceps are most helpful. Right-angled scissors(e) assist in division of the bladder from the prostate. It is reassuring to check ureteral

---

From the Division of Urology, Department of Surgery, University of California School of Medicine, San Diego County University Hospital, San Diego.

Submitted 14 March 1966.

Reprint requests to: 3415 Sixth Avenue, San Diego 92103 (Dr. Prentiss).

(a) Palmer's board: Designed by Bean M. Palmer, M.D., 2961 Summit St., Oakland, California.

(b) O'Conor shield: C. R. Bard, Inc., Murray Hill, New Jersey.

(c) Vi-Drape: Aeroplast Corp., Dayton, Ohio.

(d) Raney coagulating forceps: V. Mueller and Co., Chicago, Illinois.

(e) Right-angled scissors: Sklar Co., Long Island, New York.

continuity with intravenous indigo carmine; if doubt exists, catheterization of the ureters can be done for reassurance. Vest's[24] anastomosis was done in 29 patients, while direct vesicourethral anastomosis[7] gave comparable results in five. A 30-ml No. 22 F Foley catheter was left indwelling for at least 10 days; the average was 13.8 days. The single Penrose drain was removed in average of seven days. Average stay in hospital was 16.3 days.

Results and Complications

Problems of unusual magnitude did not occur during the surgical procedure except for bleeding, which in all cases could be controlled. In such cases, blood was transfused to restore the volume. In two cases the rectum was inadvertently incised, but the complication was recognized and, after repair, the operation was completed. Healing was complete after three weeks of catheter drainage.

Postoperative complications were:

Complications	*Number*
Posterior flap necrosis	4
Wound infection	3
Urethroperineal fistula	2
Retrovesical bleeding	1
Catheter displacement	1

There was no incidence of complete wound separation, intravesical hemorrhage, thrombophlebitis or major cardiovascular-pulmonary complication. There were no deaths during or within three months after operation.

The late surgical complications were related to urinary incontinence: Five patients were temporarily incontinent and five others were partially incontinent for over one year. Urinary incontinence is a problem for which we do not have a solution. Sexual impotence occurred in all but two patients.

The long-term results were as follows:

	Number of Patients
Alive and well,	
Five years or more	14
Less than Five years	12
Total	26
Died with prostatic carcinoma	2
Died without cancer	4
Alive with recurrent carcinoma	2

Troublesome necrosis of the perineal skin and subcutaneous tissue anterior to the anus occurred in four patients. The incision and approach was then changed from the Belt[4] to the original method described by Young[28] in order to obtain more thickness and better vascularity of the posterior flap.

Wound infection and separation was not a serious problem. In one of two patients with temporary urethroperineal urinary fistula, healing occurred after the drain was removed; in the other, after electrocoagulation of the fistulous tract. In one case perineal reopening was necessary to replace a catheter that was extruded when the balloon deflated. The procedure now followed is to suture the Foley catheter to the penis.

Discussion

The patients chosen for total prostatovesiculectomy were carefully selected for potential curability. Prostatic carcinoma was suspected in all except two patients by the detection of a nodule or discrete firmness. Twenty-nine of the patients were considered good to excellent risks. Two patients had previous coronary thrombosis. Two had controlled diabetes mellitus, complicated in one case by duodenal ulcer and by pulmonary emphysema in the other. Their "physiological age" was estimated to be 70 years or less.

The selectivity is emphasized by the fact that we did partial prostatectomy (all methods) in 1,904 patients during the same period. Transurethral resection for prostatic carcinoma was done for 259 patients.

All the 34 patients had a strong desire for "cure" and prolongation of life. In all but one patient the neoplasm was thought to be contained within the prostatic capsule, as estimated by rectal examination, cystoscopy, urography, serum acid phosphatase determinations and additional studies when indicated. Particular care was taken to explain the risks involved; especially as applied to sexual disability and possible urinary incontinence.

Perineal needle biopsy, as an office procedure, was helpful and reliable. In several instances needle specimens were negative but frozen or paraffin sections were positive for malignant disease. No problem with "false positive" from needle specimens was encountered. The diagnosis was made in two patients from tissue resected transurethrally for presumed benign hypertrophy. Neither had evidence of carcinoma in the specimens excised at total prostatectomy.[11,18]

It is impossible to correlate the pathologist's report and the subsequent course of the patient.

Four patients had carcinoma in the seminal vesicles. At last report, one of them was alive 12 years after operation and another for 10 years without recurrence. Eleven patients were reported to have perineural invasion and two of those were alive more than five years[13] after operation at the time of this report. It is similarly not possible to make a prognosis from the report of "undifferentiated or invasive" carcinoma in this series.

This study is too small to permit statistical conclusions. Twenty-six of the 34 patients are pleased with the probable eradication of the malignant disease. Two patients died with prostatic carcinoma and in one of them death was due to coronary thrombosis. A five-year follow-up is not sufficiently long to draw conclusions regarding "cures" in prostatic carcinoma.

We prefer the perineal approach for the availability of biopsy material, ease of hemostasis and vesicourethral anastomosis, and the excellent tolerance to the operation.

In Turner and Belt's[23] report on 229 patients in 1956, a 10-year survival after total perineal prostatectomy for prostatic carcinoma was 47 per cent. Jewett, Persky, Norfleet and others* have published results supporting the usefulness of this operation.

---

*Reference Nos. 1, 6, 14, 15, 16, 20, 21, 22, 23, 25, 27.

REFERENCES

1. Atherton, L. D., and Atherton, L.: Aggressive surgery for carcinoma of the prostate, J. Urol., 91:294-297, 1964.

2. Barnes, R. W.: Results of palliative treatment of early carcinoma of the prostate, J. Urol., 70:489-490, 1953.

3. Barnes, R. W., and Emery, D. S.: Management of early prostatic carcinoma, Calif. Med., 91:57-61, 1959.

4. Belt, E., Ebert, C. E., and Surber, A. C.: A new anatomic approach in perineal prostatectomy, J. Urol., 41:482-497, 1939.

5. Belt, E.: Radical perineal prostatectomy in early carcinoma of the prostate, J. Urol., 48:287-297, 1942.

6. Colby, F.: Carcinoma of the prostate; Results of total prostatectomy, J. Urol., 69:797-806, 1953.

7. Colston, J. A. C.: Campbell's "Urology," Vol. 3, W. B. Saunders Co., Philadelphia, 1963.

8. Emmett, J. L., Greene, L. F., and Papantonian, A.: Endocrine therapy in carcinoma of prostate gland; 10 year survival studies, J. Urol., 83:471-484, 1960.

9. Flocks, R. H., Culp, O. A., and Elkins, H. B.: Present status of radioactive gold therapy in the management of prostatic cancer, J. Urol., 81:178-184, 1959.

10. George, F. W., Carlton, C. E., Jr., Dykhuizen, R. F., and Dillon, J. R.: Cobalt-60 telecurietherapy in definitive treatment of carcinoma of prostate, J. Urol., 93: 102-109, 1965.

11. Goodwin, W. E.: Radical prostatectomy after previous prostatic surgery, J.A.M.A., 148:799-803, 1952.

12. Hinman, F., Jr.: The early diagnosis and radical treatment of prostatic carcinoma, Calif. Med., 68:338, 1948.

13. Hudson, P. B., Finkle, A. L., and Totten, R.: Prostatic cancer, Cancer, 5:1225-1228, 1952.

14. Jewett, H. J.: Radical perineal prostatectomy for cancer of the prostate: An analysis of 190 cases, J. Urol., 61:277-280, 1949.

15. Jewett, H. J.: Radical perineal prostatectomy for carcinoma, an analysis of cases at Johns Hopkins Hospital, 1904-1954, J.A.M.A., 156:1039-1041, 1954.

16. Jewett, H. J.: Treatment of early cancer of the prostate, J.A.M.A., 183:373-375, 1963.

17. Kerr, H. D., Flocks, R. H., Elkins, H. B., Culp, O. A., and Evans, T. C.: Follow-up study of 100 cases of carcinoma of the prostate treated with radioactive gold, Radiology, 64:637, 1955.

18. Montgomery, T. R., Whitlock, G. F., Nohlgren, J. E., and Lewis, A. M.: What becomes of patients with latent or occult carcinoma of prostate? J. Urol., 86:655-658, 1961.

19. Nesbit, R. M., and Baum, W. C.: Endocrine control of prostatic carcinoma; clinical and statistical survey of 1,818 cases, J.A.M.A., 143:1317-1320, 1950.

20. Norfleet, C. H., Jr., and Brooks, R. E., Jr.: Radical perineal prostatectomy, Southern Med. J., 55:860-861, 1962.

21. Russel, T., Austen, G., Jr., and Persky, L.: Experiences with operable carcinoma of the prostate, Ohio Med. J., 59:485-487, 1963.

22. Scott, W. W.: An evaluation of endocrine therapy plus radical perineal prostatectomy in the treatment of advanced carcinoma of the prostate, J. Urol., 91:92-102, 1964.

23. Turner, R. D., and Belt, E.: Radical prostatectomy for cancer: Results, J. Urol., 77:62-77, 1957.

24. Vest, S. A.: Radical perineal prostatectomy, modification of closure, Surg., Gyn., and Obst., 70:935-937, 1940.

25. Vickery, A. L., Jr., and Kerr, W. S., Jr.: Carcinoma of the prostate treated by radical prostatectomy, Cancer, 16:1598-1608, 1963.

26. Weyrauch, Henry M.: Surgery of the Prostate, W. B. Saunders Co., Philadelphia, 1959.

27. Whitmore, W. F., Jr.: The rationale and results of ablative surgery for prostatic cancer, Cancer, 16:1119-1132, 1963.

28. Young, H. H.: The early diagnosis and radical cure of carcinoma of the prostate. Being a study of 40 cases and presentation of a radical operation which was carried out in four cases, Johns Hopkins Hosp. Bull., 16: 315-321, 1905.

29. Young, H. H.: The cure of cancer of the prostate by radical perineal prostatectomy. History, literature and statistics of Young's operation, J. Urol., 53:188-253, 1945.

Cherish the Capacity to Change

JOSEPH T. WEARN, M.D., *Brooklin, Maine*

The following address was given at the Gold-Headed Cane Ceremony, University of California School of Medicine, on 3 June 1966. At this event on the eve of Commencement Day, the senior student who has been chosen by his classmates and professors in the Department of Medicine as the one who best exemplifies the qualities of a true physician is given a gold-headed cane. This carries on the English tradition of a cane which was passed from physician to physician from 1689 to 1825, and was carried successively by Drs. John Radcliffe, Richard Mead, Anthony Askew, David Pitcairn and Matthew Baillie. The original cane now rests in the Hall of the Royal College of Physicians in London.

FIRST LET ME EXPRESS to you how much I appreciate being invited to talk at your Gold-Headed Cane Ceremony. When I read the list of distinguished physicians who had delivered the previous addresses, I realized that I was honored indeed to follow such eminent members of the medical profession. It was my privilege to have known many of the speakers, one in particular, Dr. William J. Kerr, who initiated this Gold-Headed Cane Ceremony. I treasure my friendship with him which began in our days together in the Harvard Medical School. This ceremony is a splendid memorial to his success in reemphasizing the art of medicine in the care of the patient.

The Gold-Headed Cane itself gives us some intimate views of the art of medicine as practiced by the physicians who carried it. It was the art of medicine that they practiced, for the science of medicine was practically non-existent at that time. Therapy consisted of counter-irritants, bleeding, sweating, purging, cupping and blistering. Patients were bled as many as five times daily.

And for such ailments as pain in the shoulder the following two prescriptions were used: "*Rx*—Barley, liquorish, raisens; *Rx*—Emulsion of linseed oil, sugar candy, barley and crabs' eyes."

Plants, herbs and roots for therapeutic use were frequently grown by physicians in their own gardens. It is true that William Harvey in 1628 had demonstrated the circulation of the blood, and Linnaeus, Malpighi, Mayow, Lower, Lavoisier and Vieussens had published some of their pioneering works, and The Cane carriers must have been well aware of them, but a century was to pass before the significance of these experiments could be appreciated. Sydenham was just bringing out his treatise on the course and treatment of disease based on his own observations. He died as the Gold-Headed Cane began its autobiography in 1689 while in the hands of John Radcliffe.

Fortunately for them, all the carriers of The Cane were well off financially and were able to avail themselves of university educations. Richard Mead, for instance, traveled widely over the continent, studied at Utrecht and Leyden and got his degree of Doctor of Medicine at Padua. Anthony Askew likewise traveled extensively, spent money freely on rare manuscripts, studied at Leyden and got his M.D. degree at Cambridge. It doesn't require much imagination to draw the conclusion that they not only sampled but put to thorough test the best foods and wines of the countries in which they sojourned.

Having read how these scholarly physicians who carried the Gold-Headed Cane pursued their med-

---

Hord Professor of Medicine, Emeritus; Dean, Emeritus, School of Medicine, Western Reserve University Consultant, Harvard Medical School and Western Reserve University.

Submitted 3 August 1966.

Reprint requests to: Department of Medicine, University of California Medical Center, San Francisco 94122 (Dr. Lloyd H. Smith, Jr.).

ical education, my curiosity was aroused about medical education in general in that day and how it managed to undergo such a complete change in order to arrive at its present state. What role were the medical schools and the hospitals of that time playing in medical education?

On the continent there were several well established universities with medical schools. In Italy, the first school was established at Salerno in the ninth century, to be followed by those at Bologna and Padua three centuries later. Then in 1231, by decree, Salerno was designated as the only school in Italy where medicine could be taught. In France at Montpellier, medicine in some form was taught as early as 1134. The hospital there, one of the earliest, was established in 1198 and anatomy was taught in 1285.

The University of Paris offered medicine in the Twelfth Century, and before a medical building was constructed in 1505, the Cathedral of Notre Dame was used for medical teaching.

In England, Oxford and Cambridge were founded probably in the Thirteenth Century, and the Scotch schools at St. Andrews, Glasgow and Aberdeen were established in the Fifteenth Century.

In Oxford and Cambridge medical "science" was taught, but only as a part of a general philosophical education. Eleven years were required to obtain an M.D. degree and the earliest one conferred at Oxford was given in 1312. What part of the 11 years had to be spent in residence is not clear, but medical students were able to travel and attend lectures on the continent. There was no hospital in either university. In fact, it is only comparatively recently that these schools have had hospitals for medical teaching.

In London, St. Bartholomew's and St. Thomas' Hospitals were founded in the Middle Ages but clinical lectures and clinical teaching were not permitted in them until the first part of the Eighteenth Century. Thus in the time of John Radcliffe and Richard Mead none of the English or Scotch medical schools was connected with hospitals.

Several forms of license could be sought in England. The license from the College of Physicians required a University degree, usually from Oxford or Cambridge. That of the Company of Barber Surgeons or the Society of Apothecaries required apprenticeship training.

In the early Seventeenth Century, attending surgeons at St. Bartholomew's or St. Thomas' Hospitals could bring their apprentices to observe. This gave the surgeons prestige and increased the number of applicants for apprenticeship. The fees which the preceptors charged were none too modest, sometimes reaching 500 guineas a term. St. Thomas' Hospital ruled in 1717 that apprentices should not be permitted to bring their friends into the hospital to view the dressing of patients.

There were in addition "walking pupils" who paid a fee to the hospital for being allowed the privilege of observing hospital practice for a month or two.

A university medical education consisted of Latin and Greek, and extensive readings in these languages, including the medical classics of Hippocrates, Rhazes, Galen and others. Medical teaching consisted chiefly of theoretical discourses or reading by the instructor from the classics. There was one course of 50 lectures on the "Aphorisms of Hippocrates" and endless discourses on "the fevers." The University M.D. degree so obtained was mostly for prestige, and only the well-to-do, with few exceptions, could afford it.

In some schools on the continent, a discourse was required for the degree of Doctor of Medicine. A formal public ceremony was held to accept the candidate into the dignified "medical corporation." The whole faculty marched in to the ringing of bells. The candidate's professor then reviewed and criticized the student's discourse, and the candidate took an oath that he would always fulfill his duties to the faculty and the whole medical profession. A doctor's hat was placed on his head and a ring on his finger to signify his knightly rank. A golden belt was fastened on him and a book of Hippocrates was opened before him. He was then invited to sit beside the President, who embraced him and conferred his blessing. The ceremony closed with the thanks of the new doctor, and was followed by a banquet offered to all members of the faculty. The expenses of this ceremony were paid by the candidate. This, in addition to the fees paid and the presents which were given to members of the faculty and to various persons, added to the costs of a medical education.

The Cane carriers had an education of the University type but, although thus armed, did little or nothing to advance medicine in their day. If they did not make such an effort, it could scarcely be expected that those with more limited training and certainly with much less income would take the forward steps in this field.

By far the most common type of medical education in the Seventeenth and Eighteenth Centuries was that of the apprenticeship, which was shorter in duration and less expensive. Inasmuch as this was the only kind of medical education available in the American colonies, and was given by physicians coming from England, we might turn our attention to early medical education in our country.

In the Seventeenth Century when the London Company or the West India Company sent settlers to America, it made contracts with surgeons or apothecaries to accompany the settlers and take care of them for a given period. The New England Puritans were more inclined to turn to religious healers. Thus Deacon Samuel Fuller came on the Mayflower and attended the Pilgrims in Plymouth until he died in 1632. The Reverend Thomas Thatcher, of the Old South Church in Boston, issued the first printed medical document in the British Colonies. The subject was The Treatment of Small Pox.

Graduates from English universities read some of the medical classics in the course of their general education and were, therefore, sought after for medical advice. John Winthrop, the first governor of Connecticut, a graduate of the University of Dublin, was in frequent demand for such advice. In the British colonies those who administered to the sick were called "healers," while the Dutch referred to them as "comforters of the sick."

The trend in medical training in the colonies was determined to a great extent by the environmental demands. There were, of course, no medical schools and the apprentice system was used almost entirely to meet the demand for physicians, which increased as the colonies grew. The relatively few physicians from the old countries, either surgeons or apothecaries, had no difficulty in finding apprentices.

There were no requirements or standards for admission to an apprenticeship in the Seventeenth and Eighteenth Centuries and some of the apprenticeships were of only six months' duration. As would be expected, many abuses occurred, and the point was finally reached where, in Connecticut and Rhode Island, licenses were required. But no examination or any special requirements were necessary, and licensure was, therefore, ineffective. This state of affairs persisted almost without change well into the Eighteenth Century.

The general attitude of the public of that day is of interest. In Massachusetts the witchcraft court was dissolved in 1692 but witch hunting did not cease immediately and it raised its head at the time of a smallpox epidemic in Boston in 1721. The Reverend Cotton Mather, through reading the Philosophical Transactions of the Royal Society, had learned of the protection afforded against smallpox by inoculation of pus from the lesions of active cases. He persuaded Dr. Zabdiel Boylston to inoculate 250 persons. All but six of those who were inoculated recovered, whereas the uninoculated who caught the disease had a mortality of over 50 per cent. The public, apparently giving its attention to the deaths among the inoculated, raised a terrific hue and cry against Mather and Boylston. Their houses were stoned, the windows broken and they were insulted on the streets and threatened with death. Despite this, Boylston used the inoculation in the next epidemic in Boston, again with success.

The number of practitioners in the colonies increased with the demand, and the supply of medical help was said to be ample. So great was the competition that physicians coming from Europe, after graduating from medical schools with the best medical education available, in many instances were not successful as practitioners, as they were unable to compete with the healers, who charged low fees. As a result many of these medical school graduates went into business as merchants, to the great disadvantage of the standards of medical practice.

As the colonies grew, the demand for better care increased; more educated physicians came to serve as preceptors, and they were more exacting in their standards. Gradually the length of apprenticeship was increased, the requirements were made more exacting and the end product improved. The apprentice moved into the house of the local practitioner, his preceptor-to-be, and bound himself by legal contract to perform certain duties.

I should like to read you parts of a legal contract in which Hollister Baker bound himself as apprentice to Benj'n Gott, his preceptor, in Marlborough, Massachusetts, in 1734:

This Indenture Witnesseth, that Hollister Baker, a minor, aged about 16, son of Mr. Edm'nd Baker, late of Marlborough, in the Co. of Middlesex gent. deceased. Of his own free will and accord and with the consent of Benj'n Woods, of Marlborough, in county aforesaid, his guardian, doeth put and bind himself to be an apprentice unto Benj'n Gott, in county aforesaid, physician, to learn his art, trade, or mystery and with him the said Benj'n Gott, after the manner of an apprentice, to dwell and serve from the day of the date hereof, for and during the full and just term of five years and 4 months next ensuing and fully to compleat and ended. During all of which said term the said apprentice his said master and mistress honestly and faithfully shall serve; their secrets keep close; their lawful and reasonable commands everywhere gladly, do and perform. Damage of his said master and mistress he shall not wilfully do; his master's goods he shall not waste, embezel, purloin or lend unto others, nor suffer the same to be wasted or purloined; but to his power shall forthwith discover and make known the same to his master and mistress.

Taverns nor alehouses he shall not frequent, or cards or dice or any other unlawful games he shall not play. Fornication he shall not commit, nor matrimony contract with any person during said term. From his master's service he shall not at any time unlawfully absent himself, but in all things as a good, honest and faithful servant and apprentice shall bear and behave himself towards his said master during the full Five Years and Four Months, commencing as aforesaid.

And the said Benj'n Gott for himself doeth covenant promise grant and agree unto and with his said apprentice in manner and form following. That is to say, that he will teach the said apprentice or cause him to be taught by the best ways and means that he may or care, the trade, art or mystery of a physician, according to his own best skill and judgment (if the said apprentice be capable to learn) and will find and provide for and unto said apprentice good and sufficient meat, drink, washing and lodging during said term both in sickness and in health; his mother all said term finding said apprentice all his clothing, of all sorts, fitting for an apprentice during said term; and at the end of said term to dismiss said apprentice with good skill in arithmetick, Latin and also in the Greek grammar.

In testimony whereof, the said parties to these present indentures have interchangeably set their hands and seals the eighth day of January, in the Fourth year of the reign of our souveragn, Lord George, Ye Second, by the Grace of God, of Great Britain, France & Ireland and in the year of our Lord one thousand seven hundred and thirty-three-four.

As early as 1647 Massachusetts made the legal provision that students of medicine "may have the liberty to reade anatomy and to anatomize once in four yeares, some malefactor, in case there be such as the Courts shall allow of." But there was almost no opportunity for "anatomizing" or even to witness dissections, and not even the preceptors knew anatomy.

At the end of the first third of the Eighteenth Century new hospitals were built in London. Guy's was added to St. Bartholomew's, with the aid of Richard Mead, who at that time was in possession of the Gold-Headed Cane. The Royal Infirmary in Edinburgh and other hospitals were built and the staffs of these institutions soon gained renown. By that time some of the Americans who had graduated from Harvard, King's College and William and Mary, and who had completed apprenticeships with the more outstanding physicians in this country, elected to pursue their studies further and went to Edinburgh, Leyden, Paris and other places. Many remained in England and few returned.

It is to the few young men who did return that the first real awakening in medicine in this country is due. Having seen the best medicine in Europe, they were determined to raise the standards in this country.

John Kearsly had come to Philadelphia in 1711 and trained as apprentices William Shippen and John Bard, whose sons in turn studied medicine. William Shippen, Jr., spent five years as an apprentice and then went to Europe to continue his studies. John Redmond, another preceptor in Philadelphia, trained John Morgan, Benjamin Rush and Casper Wistar, all of whom studied subsequently in Europe. In 1765, these three became professors in the Philadelphia Medical College, later the School of Medicine of the University of Pennsylvania. Morgan and Shippen served as General George Washington's Surgeons General, and Benjamin Rush signed the Declaration of Independence.

Benjamin Rush says that in the more than five years of his apprenticeship (February 1761 to July 1766) he was absent only 11 days and only three evenings. He prepared medicines, visited the sick, performed the offices of a nurse and kept books and accounts. He found the confinement conducive to business and study and, to quote him, "[the duties] produced in me habits of industry and business which never left me."

A similar group in New York founded King's College, later the College of Physicians and Surgeons of Columbia. Harvard Medical School was started in 1783.

In this same year the University of Edinburgh Medical School, which was begun in 1726, required a three-year medical course, one year of which had to be in residence in Edinburgh.

The first permanent hospital in the colonies was the Pennsylvania Hospital, founded in 1751 by Thomas Bond with the aid of Benjamin Franklin. By 1765 a faculty had been assembled at the College of Philadelphia with William Shippen, Jr., in Anatomy, Surgery and Midwifery (admission was by card signed by Shippen for a fee of 20 pistoles —$24.00); John Morgan in Botany and Chemistry; Thomas Bond in Clinical Lectures and Benjamin Rush in the Theory and Practice of Physic. It was assumed from the beginning that clinical teaching would be carried on in the Pennsylvania Hospital.

The M.D. degree required three years but very few students took the Doctor of Medicine course, as the course for the degree of Bachelor of Medicine was much shorter.

These activities in the colonies were concomitant with the periods of Richard Mead and Anthony Askew.

Gradually the apprenticeships were liberalized. The apprentices lived outside the preceptor's home and paid a fee in lieu of rendering service. The apprenticeship system of medical education lasted well into the Nineteenth Century, although the loss of confidence in the system increased steadily. About one hundred years ago fully 80 per cent of the practitioners in America had never been in a medical school.

Nevertheless, as the abuses in the apprentice system increased and as the public lost faith in the healers, the attendance in medical schools began gradually to increase. Even in the schools, standards were low, as many were proprietary or purely commercial and new schools were appearing rapidly. There were no admission requirements except the fee; and in some there were no examinations. A group of practitioners could form themselves into a faculty, whether qualified or not, acquire a building and, however inadequately equipped, get a license and run a medical school as a sideline to their practice. The fees went into their pockets, and while some of these medical schools had connections with universities, they were the loosest sort of affiliations. Many had no connections whatever with a college or university. In the beginning the required courses covered six to eight months. This grew to two years but the terms were only four months in each year. In many schools the same lectures were repeated in the second year.

The important point to observe in this situation is that medical education and the practice of medicine were *in statu quo*. The medical faculties seemed content to have it remain there. There were very few serious attempts to advance knowledge in the field, and the type of education determined the type of practice and quality of medical care. Both left a great deal to be desired. The medical educators not only did not themselves bring about changes for the better, they resisted change in any form. And this resistance seems to have been a characteristic of medical educators until comparatively recent times. Many of the important progressive changes in medical education have been instigated by persons or forces outside of medicine.

The American Medical Association was founded in 1847 and it made efforts to improve the state of medical education, at first without much effect. It was at about this time that Dr. Hugh Huger Toland established the first medical school to survive in California. The Toland Medical College, as he called it, came into being in 1864. It affiliated with the University of California in 1873 to become the University of California Medical School.

One of the most striking instances of action from outside occurred at Harvard in 1869 when Charles W. Eliot became president at the age of 35. He began immediately to attend and preside at the medical faculty meetings, a new experience for the medical faculty. In his first annual report he said, "The whole system of medical education in this country needs thorough reformation." He began immediately to work toward this end and, of course, ran into very stubborn resistance from the faculty. Here again that resistance to change showed itself. Oliver Wendell Holmes, the poet, then professor of Anatomy, at first opposed Eliot's reforms but later supported them in a half-hearted way. Holmes wrote, "He [Mr. Eliot] comes to the meeting of every faculty, ours amongst the rest, and keeps us up to eleven and twelve o'clock at night discussing new arrangements. I cannot help being amused at some of the scenes we have in our medical faculty—this cool, grave, young man proposing in the calmest way to turn everything topsyturvy, taking the reins into his hands and driving as if he were the first man that ever sat on a box. I say amused, because I do not really care about most of the changes he proposes, and I look on it a little as I would at a rather serious comedy. 'How is it, I should like to ask,' said one of our number the other evening, 'that this faculty has gone on for eighty years, managing its own affairs and doing it well—for the medical school is the most flourishing department connected with the college (sic)—how is it that we have been going on so well *in the same orderly path for eighty years,* and now within *three* or *four* months it is proposed to change all our modes of carrying on the school—it seems very extraordinary, and I should like to know how it happens.' 'I can answer Dr. Bigelow's question very easily,' said the bland, grave, young man. 'There is a new president.' "

Before Eliot became president there had been no requirements for admission and many of the students could scarcely read or write. The fee was the only requirement. The medical course had been three winter terms of four months each—a total of one year. Lectures and courses were haphazard and repetitive. Oral examinations were held, and the student had to pass five out of nine subjects to get a diploma. He then went without further training into practice. President Eliot put in entrance requirements, increased the three terms to nine months each, made the courses progressive rather than haphazard, and required written examinations in which the student had to pass all courses. This epochal change was gradually adopted by a few other medical schools, but the many commercial and proprietary schools were not interested and most schools were without standards into the early part of the Twentieth Century. Medical men resisted change.

Then many drastic changes began to occur with the appearance of laboratories in departments of biochemistry, pathology and physiology. Medical research began to raise its head. The Johns Hopkins Medical School was opened in 1893 with the requirement of a bachelor's degree for entrance.

In 1910 medical education was shaken to its roots by the publication of Abraham Flexner's report of a study that was carried out at the request of the American Medical Association. Here again a non-medical individual's study and recommendations for reforms of medical education resulted in a veritable revolution. The commercial and proprietary schools dropped out of existence, the relationship of medical schools to their universities became firm, standards rose, school and hospital relationships improved, a scientific base for medicine was started and financial support of medical education by foundations and generous individuals made the renaissance of medical education a reality.

The sweeping changes brought about by The Flexner Report helped prepare the medical schools for the great awakening they were about to experience, namely, the beginning and rapid increase of research in medicine.

Many schools began to institute the full time system for their faculties, which made it possible for them to devote a great deal of time to research. As a result a steadily increasing mass of new medical knowledge was forthcoming. World Wars I and II brought into sharp focus many of the shortcomings and needs in medicine, and this led directly to the outpouring of federal funds for research. Before World War II funds for medical research were, relatively speaking, a trickle. Now the stream from the Federal Government has become a torrent.

As a result, whole groups of diseases have been conquered and the burgeoning mass of medical knowledge has become so great that no student can master it and no faculty member can teach it.

This has led to another stupendous but healthy change in medical education; but this time medical educators themselves are making the change, which is still very much in progress. Many schools are looking with a severely critical eye at the content and methods employed in their curricula. A number have made radical changes and more are in the making.

Another change is occurring which concerns you who are entering upon your medical careers, and you must without delay prepare to play your role in guiding it lest forces outside of medicine seize control of it—which they stand poised to do.

Medical knowledge has been and is continuing to increase with such rapidity that those engaged in medical research are focusing their attention on narrower and more circumscribed fields and studying these in greater depth. As an example, when I was in medical school, investigators were trying to identify the hormones produced by the pituitary gland. Today they are unraveling the molecular structure of the growth hormone. Of this you in this school are well aware. This trend in medical research, to study narrower fields in greater depth, has carried over into the clinical field and one finds clinicians acquiring greater knowledge in narrower or more limited fields—in other words, specializing.

A definite trend is now obvious, namely, more and more young physicians are becoming specialists and fewer are going into internal medicine, pediatrics and general practice. If this trend continues, family physicians will be scarcer and scarcer and patients will have to determine for themselves which specialist to consult. This will lead to chaos, and is wholly unfair to those whom the profession serves. While the profession thus splinters itself into narrower and narrower specialties the patient remains a whole human being, and more than ever before will need a wise physician to whom he can turn. This physician must be able to render comprehensive and continuing health care to his patients and, therefore, must have a broad and sound knowledge of internal medicine and access to all medical services in this community. He will serve as the counselor to the patient and his family. He will arrange for consultations with specialists or for care in a hospital, turning over responsibility when indicated, but retaining an overall responsibility. He can work informally or formally with a group of physicians, but would be the physician of first contact. His training must be broad and thorough enough to give him stature equal to that of any specialist. His importance at this time cannot be overestimated, for his patients will expect him to assume overall care of them, coordinate the opinions of consultants if they are needed, and in the end solve the patients' problems. His responsibility will be the continuous maintenance of health.

One important contributing factor to the serious if not alarming situation is the fact that the great increase of research in medical schools has moved with such speed and has so occupied the attention of medical educators that medical care in the broad sense has lagged as a result. Don't misunderstand me. The research has brought untold benefits, and it must not be curtailed, although greater emphasis on quality than on quantity might be considered. But medical care must not suffer; indeed it must be brought abreast of research, and this without delay.

In some medical schools there has been so much concentration on the research programs that the faculties are made up only of men whose prime interest is in research. As a result the students have only these men as models. Very few medical schools or university hospitals today have comprehensive medical care clinics or adequate facilities for exemplary teaching of comprehensive continuous medical care for their students or for furnishing models for such care to the community. The great majority of medical students go into practice, and it is imperative that adequate opportunities be given them for preparing themselves for this field. A few medical schools and university hospitals are moving rapidly to correct this situation, but unless correction can be made without delay, communities and forces outside of medicine will act.

Having grown up in the clinic of Francis Peabody, I had the opportunity of seeing the care of the patient at its best. His simplicity, humility and true spirit of service, together with an inborn sense of compassion, were examples that one doesn't forget. And I have come to believe that these traits are essential for the truly great physicians. Steeped as you are in the art and science of medicine, you can be assured that a rewarding and happy career lies before you, but only if you realize that at this point your education is beginning anew and that your desire to learn must continue. Medicine is ever changing; it has changed in the past, is changing at present and will change in the future. To meet this challenge you must cherish your capacity to change with it.

Project Head Start

An Evaluation of the Medical Components in California

ARNOLD GILBERT, M.D., ARLINE LEWIS, M.P.H., AND ROBERT W. DAY, M.D.,
Berkeley

■ *Half of 1,135 children medically examined as a part of Project Head Start in California had one or more conditions that warranted referral to a physician or dentist, and only one-fifth of these were under care. In the judgment of the examining physicians, one-third of the referable medical conditions were described as "major." Follow-up procedures were variable and not very successful.*

Increased local medical society participation in planning the health services for these children is recommended as an especially important step in securing care for the problems that are identified.

PROJECT HEAD START, a major program in the "war against poverty," was launched in February 1965. Initially, this education and child development program planned to enroll 100,000 preschool children throughout the United States. Because of the tremendous community response to the project and the vigor with which it was administered, the goals were quickly expanded. By April, approximately 2,600 communities and agencies had submitted project applications. Nationally, 561,000 children and 41,000 teachers participated in programs at 13,000 individual centers.[9] Eighty-six communities in California were granted $2,545,239 for a program that served over 21,000 children.

Although Project Head Start was primarily an education program for deprived children, much attention was given to the provision of health services. The Bureau of Maternal and Child Health of the California State Department of Public Health decided to seek information about the medical aspects of the proposed program in 1965 during its initial year of operation in two components:

1. A survey by medical students of the health services provided in 28 local Head Start projects in 12 counties representative of all geographic areas of the state.*

2. Cooperation with five counties in the collection of comparable health data on more than 1,000 children in 28 local projects. A special health evaluation form was developed for this purpose. Follow-up procedures were subsequently ascertained.

Survey of Health Services in 12 Counties

A not unexpected finding of this survey, because of the speed with which Head Start was developed, was that the centers visited differed widely in examination procedures and resources used, with only a minimum degree of standardization derived from the use of the health guides and medical examination forms distributed by the Federal Head Start Office.

- Of the 28 centers visited, 12 obtained medical services from physicians who were the personal acquaintances of project staff. Five projects obtained physicians through medical societies; four

---

Submitted 5 October 1966.

Reprint requests to: Bureau of Maternal and Child Health, State Department of Public Health, 2151 Berkeley Way, Berkeley 94704 (Dr. Gilbert).

*Collins, R. C., Dorman, J. M., Shapiro, E. R., Epidemiology Research Training Program, Division of Research, California State Department of Public Health. In part supported by National Institutes of Health Training Grant 5T1 GM 21408.

used public health physicians; one obtained a physician from the welfare department; and two sent children to private physicians' offices.

- Eight projects used the local health department for help in planning, and 12 programs used public health nurses directly in the projects.
- Ten projects reported county medical societies were directly involved in their program, and 12 reported participation by dental societies.
- Medical examinations were carried out by pediatricians exclusively in five centers, by general practitioners only in 12 centers, and by combinations in six centers. Time for examinations ranged from five to 30 minutes.
- Medical histories were generally taken by project nurses. In five projects, the history was taken by teachers.
- Nineteen centers employed dentists to provide a thorough examination. Nine centers used nurses or physicians for dental screening.
- Seventeen of the centers performed urinalysis; only 12 determined hemoglobin or hematocrit.
- Psychological and developmental testing varied from tests for every child to tests only for children referred by their teachers. Some testing was done in 24 of 28 programs.
- Vision screening was done at all centers, hearing screening at 22 and tuberculin testing at 21.
- All centers checked immunization status. Immunizations were given in 14 programs by health department personnel, in six at clinics, in five by private physicians, at one by a public health nurse and in one by a school team.
- Proposed follow-up procedures were very variable. At the time of the survey, while the projects were in process, plans for follow-up had not yet been implemented. Of the 28 centers, 11 had made some arrangements for continued observation of the children after the projects terminated. All the programs planned to transmit medical evaluations to the schools the children would attend in the fall.

Cooperative State-Local Study

The health appraisal form developed by the Bureau of Maternal and Child Health was used in

TABLE 1. — *Selected Demographic Characteristics of Children Examined Under Project Head Start (Per Cent)*

	Number of Children Examined		
	Total	*Rural*	*Urban*
	1,135	444	691
	Per Cent of Each Group		
Age			
Under 4 years	1.5	0.7	2.0
4 to 5 years	46.5	33.8	54.7
5 to 5½ years	35.2	38.7	32.9
5½ and older	15.8	25.9	9.3
Not reported	1.1	0.9	1.2
Race			
White	43.8	76.8	27.5
Anglo	17.7	27.5	11.4
Mexican	26.1	49.3	11.1
Negro	37.1	12.6	52.8
Other	11.7	2.7	17.5
Not reported	7.4	7.9	7.1
Father's Occupation			
Professional, managerial, sales	8.5	2.7	12.2
Craftsmen, foreman, operatives	27.8	15.8	35.6
Private household workers, service workers	11.4	6.3	14.6
Laborers, farm or other	27.2	49.3	13.0
Not reported, none or unable to code	25.1	25.9	24.6
Education of Mother			
Grade School	25.7	40.1	16.5
High School	53.4	39.4	62.4
College	8.9	3.2	12.6
Not reported	12.0	17.3	4.5
Welfare*			
All groups	36.5	47.1	29.7
White Anglo	35.3	42.6	24.1
White Mexican	42.6	52.1	15.6
Negro	42.0	58.9	39.5
Other and not reported	18.4	21.3	17.6

*Indicates families on welfare at time of examination and some with history of having been on welfare. Probably excludes some children with history of welfare, not currently receiving assistance.

28 Head Start Centers in five counties chosen to represent rural and urban populations and on the basis of their willingness to cooperate with this evaluation. All children receiving an examination at each center were included. The study embraced 1,135 children (Table 1).

All medical examinations were performed by or under the direct supervision of pediatricians. A State Health Department team performed examinations in two counties. Dental examinations were performed by dentists at Head Start Centers and in private offices, or by nurses and physicians as a regular part of the medical examinations. Developmental testing was done by nurses, medical students and, in a few cases, physicians. The medical history was obtained from parents by nurses, teachers or physicians, usually when the child was brought for physical examination.

The examining physicians categorized medical and dental conditions as referable or nonreferable and indicated whether or not each condition was currently under care. The physicians were also asked to provide a guide for the urgency of securing follow-up services by classifying each condition as "major" or "minor." No criterion for this classification were used except the judgment of the individual physician. Dental caries, if present, were categorized as "mild" or "severe."

Five hundred and eighty-two children (51.3 per cent of those examined) had 774 referable conditions, an average of 1.3 per child. The most common condition listed was dental caries (Table 2). In only 12 per cent of cases was this dental disease said to be under care. Exclusive of dental disease, 73 per cent of the referable conditions were not under care at the time of the examinations. One-third of these referable medical conditions was considered major. A large number of possible problems were identified by screening tests (Table 3).

TABLE 2.—*Frequently Noted Referable Conditions* in Children Examined Under Project Head Start*

	Number	*Per Cent*
Total Conditions	774	100.0
Dental caries	303	39.1
Diseases of nervous system and sense organs	96	12.4
Allergic, endocrine metabolic and nutritional conditions	64	8.3
Disease of respiratory system	59	7.6
Mental, psychoneurotic and personality disorders	50	6.5
Others	202	26.1

*Groupings conform to the International Classification of Diseases.

Follow-up of Selected Conditions

Whether those children who had been identified as needing care or further evaluation had actually received the necessary attention was ascertained four months after the medical examinations by distributing questionnaires to the participating Head Start projects. The questionnaires listed each child and his conditions. Information was requested concerning the following conditions: Major referable medical problems; severe dental caries; low hemoglobin; positive reaction to tuberculin.

Replies were received from projects that served 1,022 of the 1,135 study participants. Of these children, 283 had "major referable" medical problems—anemia, positive reaction to tuberculin test, and severe dental disease. There were 331 such conditions, more than one of them in one child in some instances.

The standard uses to assess "adequacy" of follow-up was: (1) Initial follow-up obtained, and (2) additional care not needed, or needed and obtained.

Of the 331 conditions selected for follow-up study there were 269 that were not currently under care. Only 20 per cent of the 269 conditions not under care were deemed "adequately followed." Positive, tuberculin test reaction and referable medical conditions were more likely to receive follow-up services than low hemoglobin or dental caries (Table 4). Differences among the geographic areas were great.

Methodology

There are many problems in attempting a "follow-up of medical follow-up" such as was done in this study. Although the questionnaires concerning follow-up were distributed four months after the initial examinations, the information obtained may

TABLE 3.—*Results of Screening Tests of Children Examined Under Project Head Start*

Test	*Children Tested*	*Per Cent Abnormal*
Hearing	625	7.7
Vision	775	7.7*
Urinalysis	580	5.3
Hemoglobin	470	3.2†
Tuberculin	627	2.6

*Vision under 20/40, one eye.
†Hemoglobin less than 10 gm per 100 ml of blood.

TABLE 4.—*Follow-up of Selected Conditions in Children Examined Under Project Head Start*

	Dental Caries	*Low Hemoglobin*	*Positive Tuberculin*	*"Major" Medical Problem*
Total number of referable conditions	174	16	17	124
Not under care	159	12	15	83
Informed of need for follow-up	111	7	12	49
Initial follow-up obtained	45	2	12	37

reflect the status of follow-up care which existed at the termination of the summer projects, rather than at the time the questionnaires were distributed. Furthermore, the arbitrary standard of adequate follow-up used here does not take into account children who may have obtained follow-up services without the knowledge of the Head Start personnel from whom information was obtained.

While it would have been preferable to study adequacy of follow-up by interviewing the parents of each child at a designated interval after the conclusion of the summer project, the logistics of doing so made such an undertaking impossible. The pertinent question for such field program evaluations is whether it is better to have some information than none at all. Even limited information concerning the problems of follow-up in Project Head Start may contribute to the future development of successful medical programs.

The seriousness of the illness that was discovered in these Head Start children cannot be adequately described by merely listing conditions and the frequency of occurrence. Medical conditions that are included under the same diagnostic category vary in significance. For example allergic disease may be incapacitating, or only a slight annoyance.

Although an attempt was made to circumvent the difficulties of evaluating the seriousness of the disease that was discovered by categorizing all abnormalities as either major or minor, there are many limitations to this method. Conditions that seem major to one physician may be overlooked or reported as minor by another. Variable numbers of medical problems of varying degrees of seriousness will be detected in the same population by different physicians, partly because of differences in the goals for and circumstances under which they perform examinations, as well as individual differences.[3]

TABLE 5.—*Comparative Indices of Past Medical Care Among Children of Three Categories of Low Income Families in California (Source: California Department of Public Health Birth Records)*

Group	*Mother with Late or No Prenatal Care*	*County Hospital Birth*
Head Start Study Children	11.3	44.8*
California Negroes	23.9	51.4
California Farm Laborers	33.9	51.2

*Head Start Study Children born in California.

Discussion

Over 310,000 California families have incomes at or below the Aid for Dependent Children criteria for need.[6] During July 1965, almost 90,000 children, four to six years of age, were welfare recipients in California. Although the criteria for approval of Head Start projects required that 85 per cent of participating children be from families with low incomes,* because of pressures of time and the decentralized structure of program development, it is unlikely that these standards were uniformly applied. More than 35 per cent of the parents in this study came from occupation groups not usually associated with poverty.

The extent to which the children evaluated in this study reflect the health of California's poor is not clear. By some standards, their health care was better than that of the more affluent. The mothers of the children in the study reported earlier prenatal care and less frequent history of county hospital delivery than large segments of California's documented poor[1,7] (Table 5). Although adequacy of immunization ordinarily decreases with increasing age, the immunization status of the four- to six-year-old children in this study was better than that of younger California children from families with income less than $3,000 reported elsewhere.[5,11]

It is possible that the families whose children participated in these programs were not those who most needed the services, and that they were self-selected by their aggressiveness and social mobility and by their ability to use the opportunity that Head Start offered. It is also possible that the most seriously impoverished communities lacked the leadership and skill required to develop Head Start projects.

Many of the children studied were observed to have untreated medical problems which were considered "major" by the examining physicians. The impact of this untreated illness is difficult to interpret; but, considering only the subjective judgments of the examining physicians, one-third of all referable medical conditions were described as "major." In general, "major" referred to conditions like advanced phimosis with obstruction of the urinary stream or a heart murmur requiring thorough evaluation, rather than flagrant undiagnosed heart disease or previously untreated congenital dislocation of the hip.

---

*Limits of eligibility for a family of four was set at $3,000 and for a family of eight at $5,000.

It seems difficult to separate the failure of the Head Start projects to have accomplished more than just identification of conditions not under care—80 per cent failure—from the rapidity with which the projects developed. There was little time to plan for arranging medical follow-up, and there was no assignment of continued responsibility for the children after the summer program ended. Often there was inadequate communication between the sponsoring schools and health departments. Even in programs where the health department staff were participants in the project activities, the responsibility they assumed was variable. In some projects, it was the receipt of the State follow-up survey forms that initiated the follow-up activity.

Failure of parents to assume responsibility for securing care also contributed to the deficiencies of follow-up. Replies to the follow-up questionnaires indicated that at least two-thirds of the families about whom information was requested had been informed of the presence of a problem by the project personnel. Some families had made appointments for medical and dental attention but were known not to have followed through. Others had completed the first appointment but did not obtain needed additional care. Lack of finances for care was infrequently mentioned as a deterrent to care. Some families did not think the condition serious and felt that care was not necessary or that the child would "grow out" of the ailment. Some children had been seen by a physician just before the Project Head Start examination and were pronounced "normal" or "well," so that the parents would not believe or accept the present referral for care. In no case did the returned questionnaires indicate that resources for care were not available to diagnose or treat the specific condition.

Clearly, the difficulties that these children had in getting the care they needed are not confined to the Head Start Project. Review of their experience with well-child care indicated that, although 85 per cent of them had received well-child services, only 68 per cent had been vaccinated and only 62 per cent had adequate DPT immunization.

Conclusion

Head Start provides a unique opportunity to evaluate carefully the health of preschool children from low income families at a time when they are not acutely ill. For the discovery of health problems that have previously been overlooked or accepted, the advantage of an organizational structure that permits the examination of "well" children is very great.[4] Although a great deal of uncared-for illness of varying severity was noted in this study, there was little success in getting the problems under care. The following are suggestions for improving the health components of Head Start projects:

Planning and Evaluation

- Representatives of local medical societies and health departments should participate in the planning of the medical component of Project Head Start.
- Procedures should be established for review of the medical program while the project is in operation and again after the summer is over.

Examination Procedures

- Every effort should be made to have the screening examinations of Head Start children done by physicians and dentists who will have continuing responsibility for the care of the children.
- Medical examinations should be conducted near the outset of the Head Start Project so that the initiation of referral can be begun while the children are attending the program sessions and there is easy access to families, as well as maximum parent motivation.
- Parents should be present during the medical examination. Pre-recorded histories will facilitate communication.

Follow-Up

- Participating physicians and dentists should focus the attention of the Head Start Project staff on those children for whom special efforts to secure full and adequate follow-up care must be made.

- The process of guiding these families to the health services they need should be the definite responsibility of specific project personnel, preferably public health nurses.

- Some members of the Head Start staff or local health department personnel should assume continuing responsibility for follow-up *after* the summer project has concluded.

Participation of the Poor

- Special attention should be given to employing poor people in the projects, especially for recruitment of needy children, and to facilitate follow-up and carry out other project responsibilities related to health services. The effectiveness of paid "health aides" has been demonstrated in California in farm labor communities.[8]

- Health education activities for parents would be an important addition to these projects and should be part of an over-all program of parent participation.

- Selection of children would be improved by the use of public health nurses, who are familiar with the target community and experienced in working with disadvantaged families in their homes, to contact families.

Improvement in the medical services for Project Head Start may not be easily attained. Preliminary information from a 1966 Survey of Head Start Projects indicates that many of the practices and problems described in this study persisted during the second summer of operation.[2]

Despite implementation of the recommendations presented here, with adequate financial support for health services, there will continue to be severe limitations on the efficacy of short-term Head Start Projects. There will remain the difficulty of developing communication with, and access to, otherwise busy practicing physicians so that satisfactory arrangements for medical evaluation and care can be made. This is particularly true for health problems that require long-range planning and prolonged treatment.

In contrast to full-time project employees or teachers, who are employed by Project Head Start during summer vacations, physicians have a continuous responsibility to the patients who constitute their private practice. For physicians to make an effective community contribution by their work in the Head Start program, project administrators must make the organization of medical services a high priority—certainly not an afterthought. On the part of the medical community, delegation of responsibility by county medical societies for liaison with Project Head Start to a particular individual would undoubtedly improve coordination and subsequent care.

The ways in which family centered preschool education programs can be used for providing high quality medical services to disadvantaged children need careful exploration.[10] These programs make such children, who are often characterized as "hard to reach," available to physicians for counseling, diagnosis and treatment in a situation structured to maximize trust and mutual understanding.

Private pediatric care is preferable to impersonal public medical care but is ordinarily more available in middle class neighborhoods than the urban ghettoes and rural slums that are served by Project Head Start. Development of techniques that improve the medical services provided for children served by these projects is an obvious challenge to the ingenuity of physicians and public health personnel. Consideration of new uses of paramedical personnel and innovations in the use of screening tests for identifying organic and emotional health problems seem especially to require attention, as do simpler matters such as appointment hours and arrangements for transportation to medical services.

REFERENCES

1. California State Department of Public Health: Birth Records, 1961.
2. Dyar, Robert, Division of Research, California State Department of Public Health: Personal communication.
3. Eisner, V., Goodlett, C. B., Driver, M.: Health of enrollees in neighborhood youth corps, Ped., 38:40-43, 1966.
4. Gilbert, A., Schloesser, P.: Health needs of children of migrant workers in a Kansas day care program, Public Health Reports, 78:989-993, 1963.
5. Health Supervision of Young Children in California, California State Department of Public Health, Berkeley, 1960.
6. Kassenbaum, Gene G., Hopkins, Carl E., Katz, Alfred H., and Roemer, M. I.: An expanded California program of medical care for the poor, California State Department of Social Welfare, Sacramento, 1965.
7. Perinatal Mortality and Survival, California 1949-1959, California State Department of Public Health, Berkeley, 1963.
8. Potts, D., Miller, C. W.: The community health aide, Nursing Outlook, 12:33-36, 1964.
9. Richmond, J.: Communities in action, Presented at Annual Meeting of American Academy of Pediatrics, Chicago, October 1965.
10. Siegel, Earl: Health and day care for children of migrant workers, Public Health Reports, 79:847-852, 1964.
11. The 1964 Immunization Survey, Los Angeles County Health Department, 1965.

MEDICAL PROGRESS

Recent Concepts in the Clinical Pharmacology of Antihypertensive Drugs

ALAN S. NIES, M.D., AND KENNETH L. MELMON, M.D., *San Francisco*

■ *As yet, no perfect drug has been found which can reduce supine and standing blood pressure equally, alter primarily peripheral vascular resistance, increase flow to the kidneys and be free of untoward effects. The thiazides, which act directly on the vessels and α methyl dopa, which causes an incomplete sympathetic blockade, are the two agents which come closest to the ideal. The advances which have resulted from the discovery of the false neurotransmitters may be expected to lead to the discovery of nontoxic drugs which cause subtle changes in the nerve ending and more controlled sympathetic blockade.*

THERE IS NO EVIDENCE that consistent abnormalities of the sympathetic nervous system or of the biosynthesis, metabolism or concentrations of circulating catecholamines are etiologically related to essential hypertension.[49,54] It has, however, become clear that pharmacologic interruption of the function of the sympathetic nervous system will permit control of blood pressure. This review will summarize the mechanisms by which the commonly used antihypertensive agents affect the sympathetic nervous system and alter either systemic vascular resistance or cardiac output.

Sites and Action of Drugs

Understanding the action of certain drugs requires a knowledge of the anatomy of the sympathetic nervous system (schematically represented in Figure 1). Theoretically a blockade could be produced by decreasing sympathetic outflow from the central nervous system, blocking the autonomic ganglia, altering the sympathetic postganglionic nerve endings, or blocking the catecholamine receptors. Drugs acting directly on vascular smooth muscle would also be expected to affect blood pressure.

Central Nervous System and Autonomic Ganglia (Figure 1, A and B)

There are no effective agents which work primarily by decreasing sympathetic outflow from the central nervous system. Despite the discouraging results of surgical thoracolumbar sympathectomy,[60] pharmacologic blockade by ganglionic blocking agents proved effective in lowering blood pressure. Ganglionic blockers were discovered in 1913, but not systematically used in man before 1946. These agents stabilize the post-ganglionic nerve body to the effects of acetylcholine and thus block initiation of transmission through the post-ganglionic nerve fiber.

From the Departments of Medicine and Pharmacology, Division of Clinical Pharmacology, and the Cardiovascular Research Institute, University of California San Francisco Medical Center, San Francisco.

Supported by USPHS grants HE-09964, HE-06285, and HE-31,-854-01.

Reprint requests to: Chief, Section of Clinical Pharmacology, U.C. Medical Center, San Francisco 94122 (Dr. Melmon).

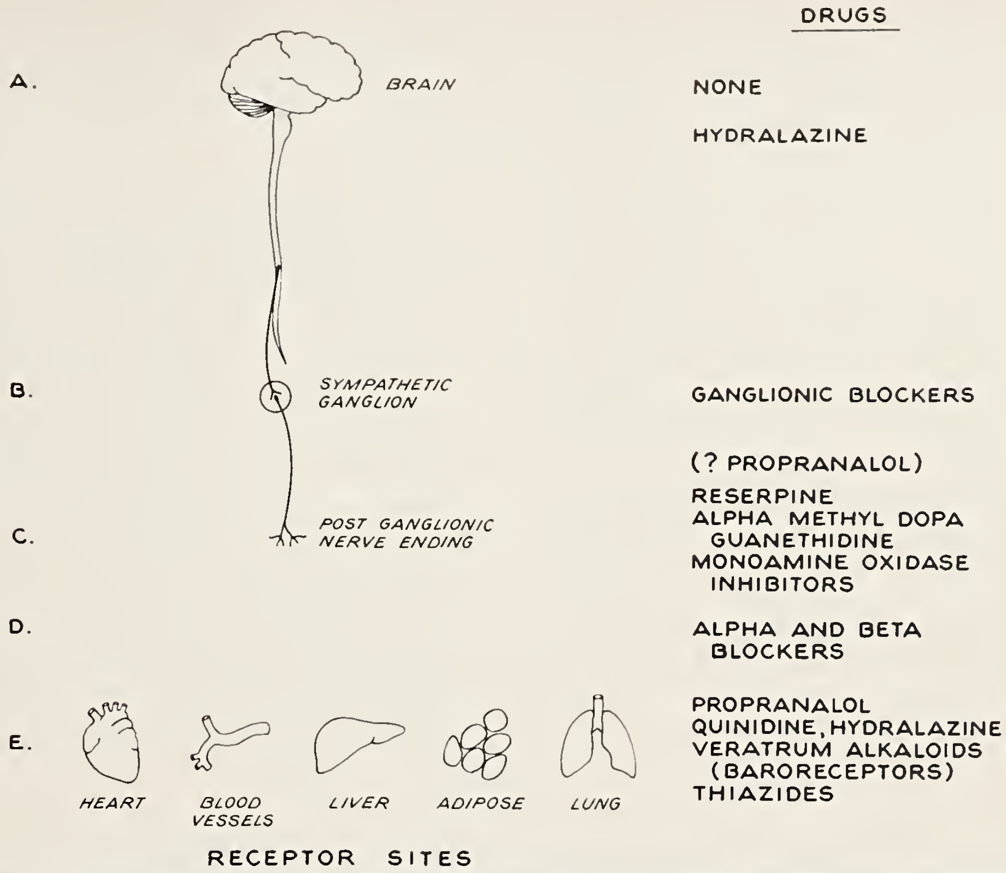

DRUGS
A.
BRAIN
NONE
HYDRALAZINE
B.
SYMPATHETIC GANGLION
GANGLIONIC BLOCKERS
(? PROPRANALOL)
RESERPINE
ALPHA METHYL DOPA
GUANETHIDINE
MONOAMINE OXIDASE INHIBITORS
C.
POST GANGLIONIC NERVE ENDING
D.
ALPHA AND BETA BLOCKERS
E.
PROPRANALOL
QUINIDINE, HYDRALAZINE
VERATRUM ALKALOIDS (BARORECEPTORS)
THIAZIDES
HEART
BLOOD VESSELS
LIVER
ADIPOSE
LUNG
RECEPTOR SITES

Figure 1.—Schematic representation of various anatomical levels at which antihypertensive drugs affecting the nervous system may work. *A*, Central nervous system. *B*, Sympathetic and parasympathetic ganglia. *C*, Postganglionic nerve body. *D*, Postganglionic nerve ending. *E*, Effector site for catecholamines.

Sympathetic Postganglionic Nerve Endings (Figure 1, C)

The modern era in antihypertensive therapy began with the realization that reserpine in clinically useful doses depletes catecholamines in the sympathetic nerve endings,[6] inhibits sympathetic reflexes[35] and lowers blood pressure. Thus, by acting primarily at the postganglionic nerve endings, drugs such as reserpine produce a selective blockade of the sympathetic nervous system without altering parasympathetic function. Reserpine may also deplete catecholamines and serotonin in the central nervous system or, when given parenterally, produce direct dilation of arteries. Using reserpine as a model agent, many workers searched for antihypertensive drugs that would act at the postganglionic nerve ending. They were to learn that catecholamine depletion may be the most obvious but not the most critical effect of an antihypertensive drug. The excitement of this search and its rewards cannot be appreciated without some knowledge of the biosynthesis, physiologic release, metabolism and inactivation of catecholamines in the nerve ending.

Catecholamines

Biosynthesis (Chart 1). The amino acid tyrosine is taken into the sympathetic neuron by active transport. In the mitochondria, tyrosine hydroxylase converts it to dihydroxy phenylalanine (DOPA), which is in turn converted to dopamine by 1-amino acid decarboxylase in the cytoplasm. Dopamine is taken up by a specialized granule, where it is hydroxylated in the β position and stored as norepinephrine.[63] The first hydroxylation is rate-limiting and may be important in end-product inhibition of norepinephrine synthesis by catecholamines.[61] The enzymes involved are not entirely specific, and substances whose structure resembles that of the natural amines and amino acids may be utilized as substrates. Likewise, the storage granules will accept other amines if they have a β-hydroxyl or a catechol (dihydroxy benzene) group.[39] The granules protect the amines from destruction, but in some pathologic states or during administration of drugs—for example, reserpine—the amines leak into the cytoplasm where they are readily oxidized by mitochondrial enzymes (Chart 2). The only product that leaves the cell in appreciable quantities after reserpine treatment is oxidized, and therefore biologically inactive.

Release. The precise way in which a nerve impulse releases norepinephrine from the nerve ending is not known. Burn and Rand[19] have postulated that acetylcholine is first released within the nerve ending and in turn discharges the contents of some granules. The evidence for this sequence is largely indirect.

Metabolism (Chart 3). Epinephrine and norepinephrine are metabolized by two enzymes, catechol-O-methyl-transferase (COMT), and monoamine oxidase (MAO) (Chart 3). Circulating exogenous or endogenous catecholamines are methylated via COMT to metanephrine and normetanephrine and then oxidized by MAO to vanilmandelic acid and methoxy-hydroxy phenyl glycol. Catecholamines within nerve endings are first acted upon by MAO and then by COMT. The final products of both pathways are the same two compounds—vanilmandelic acid and methoxy-hydroxy phenyl glycol.

False Neurotransmitters

Alpha Methyl Dopa and Alpha Methyl Norepinephrine. Hoping to alter the stores of amines in man, Oates and coworkers[45] studied the effects of α methyl dopa on the synthesis and storage of catecholamines. Although this 1-amino acid was known to inhibit decarboxylase *in vitro,* it had never produced striking effects in normotensive animals. Oates found that the 1-isomer inhibited decarboxylase *in vivo,* decreased stores of norepinephrine and lowered blood pressure of hypertensive patients. These actions were considered cause and effect until it was found that (1) norepinephrine depletion produced by α methyl dopa exceeded the duration of decarboxylase inhibition, (2) metabolism of catecholamines as reflected by vanilmandelic acid excretion were not significantly changed after α methyl dopa, and (3) agents pro-

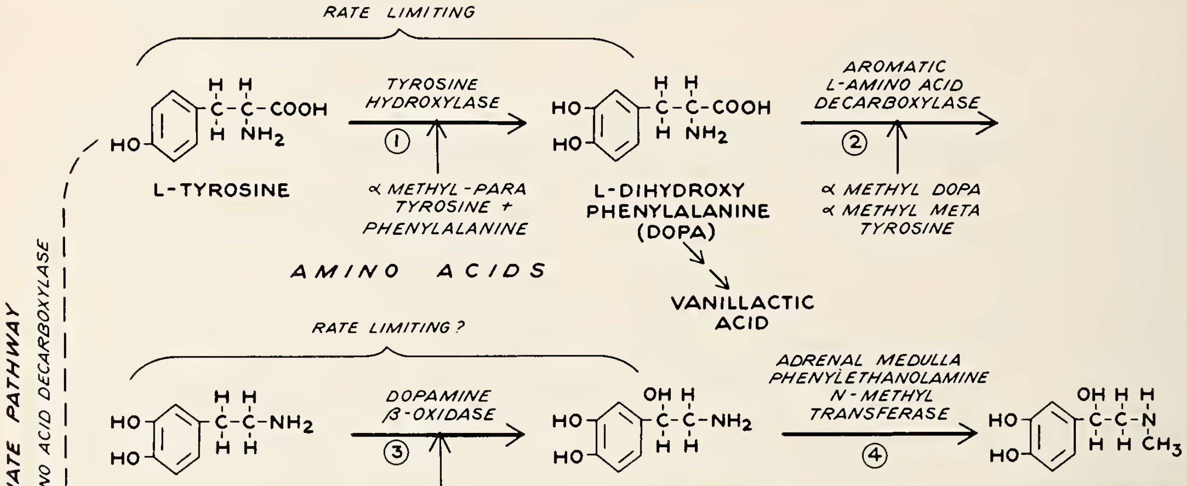

RATE LIMITING
L-TYROSINE
TYROSINE HYDROXYLASE
①
α METHYL-PARA TYROSINE + PHENYLALANINE
L-DIHYDROXY PHENYLALANINE (DOPA)
VANILLACTIC ACID
AROMATIC L-AMINO ACID DECARBOXYLASE
②
α METHYL DOPA
α METHYL META TYROSINE
AMINO ACIDS
ALTERNATE PATHWAY
AROMATIC L-AMINO ACID DECARBOXYLASE
RATE LIMITING ?
L-DIHYDROXY PHENYLETHYLAMINE (DOPAMINE)
HOMOVANILIC ACID (HVA)
DOPAMINE β-OXIDASE
③
DISULFIRAM
L-NOREPINEPHRINE
ADRENAL MEDULLA PHENYLETHANOLAMINE N-METHYL TRANSFERASE
④
L-EPINEPHRINE
CATECHOLAMINES
RABBIT ENZYME
TYRAMINE
DOPAMINE β-OXIDASE
③
OCTOPAMINE

Chart 1.—Biosynthetic pathways for catecholamine production. The bold arrows indicate major steps in synthesis. Light arrows indicate steps of minor or unestablished importance in man. Possible excretion products of catecholamines or their precursors of minor importance are indicated by diagonal arrows.

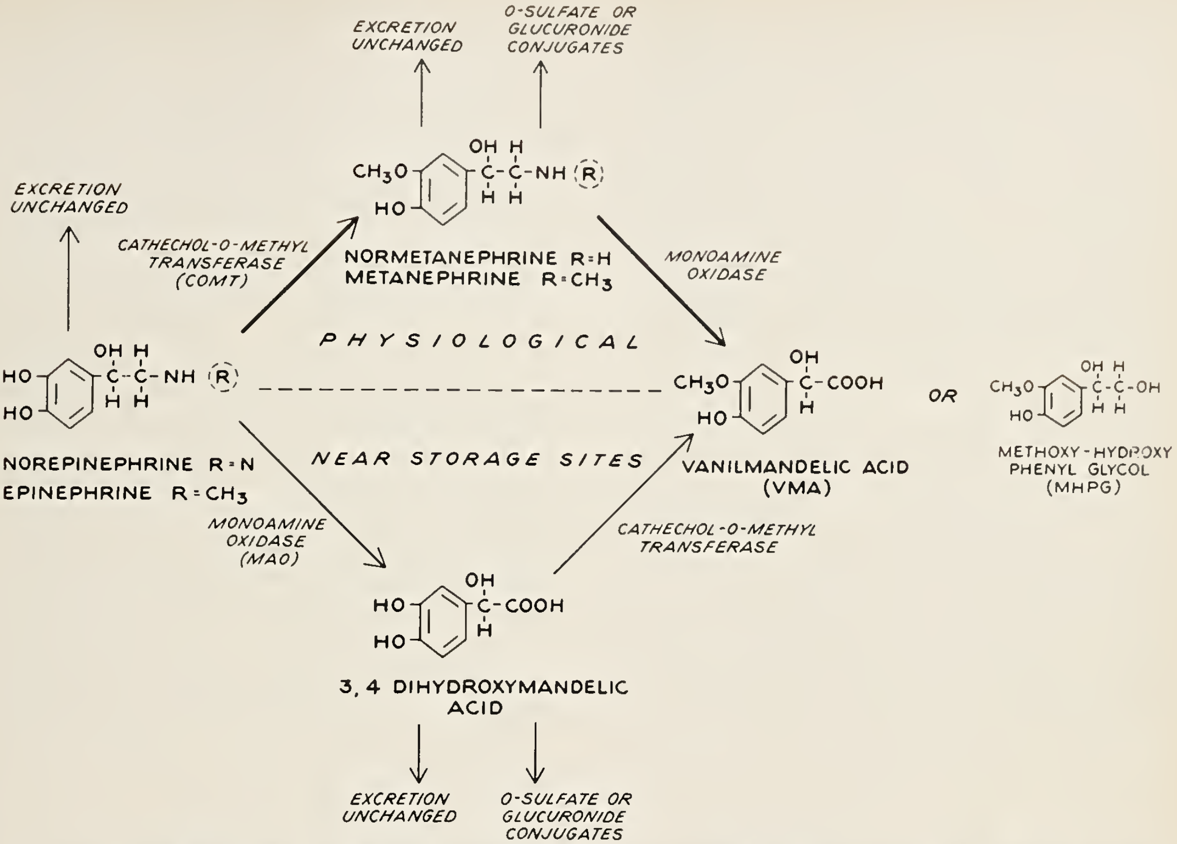

EXCRETION UNCHANGED
O-SULFATE OR GLUCURONIDE CONJUGATES
EXCRETION UNCHANGED
CATHECHOL-O-METHYL TRANSFERASE (COMT)
NORMETANEPHRINE R=H
METANEPHRINE R=CH3
MONOAMINE OXIDASE
PHYSIOLOGICAL
NEAR STORAGE SITES
NOREPINEPHRINE R=N
EPINEPHRINE R=CH3
VANILMANDELIC ACID (VMA)
OR
METHOXY-HYDROXY PHENYL GLYCOL (MHPG)
MONOAMINE OXIDASE (MAO)
CATHECHOL-O-METHYL TRANSFERASE
3, 4 DIHYDROXYMANDELIC ACID
EXCRETION UNCHANGED
O-SULFATE OR GLUCURONIDE CONJUGATES

Chart 2.—Metabolic pathways for the degradation of catecholamines. The bold arrows (top) indicate the major pathways for metabolism of circulating catecholamines, the lighter diagonal arrows the metabolic steps prominent in oxidation of amines released intracellularly. While vanilmandelic acid (VMA) and methoxy-hydroxy phenyl glycol (MHPG) are the major excretion products of metabolized catecholamines, vertical arrows indicate less important excretory products.

ducing more profound inhibition of decarboxylase did not alter synthesis or stores of catecholamines and did not lower blood pressure.[32,55] These studies set the stage for an exciting concept in pharmacology—the false neurotransmitter.

Kopin[26] first described false neurotransmitters as substances that (1) are not normally present in nerve endings, (2) are stored in the same sites as norepinephrine, (3) are susceptible to release by the same physiological and pharmacological stimuli as norepinephrine itself, and (4) would be less potent than norepinephrine. Recent data have made it necessary to extend this definition to include false neurotransmitters which (5) may or may not be chemically similar to norepinephrine, (6) may be less potent than norepinephrine or may be equipotent but released in smaller amounts, (7) may produce their effects by interrupting nervous activity or enzyme action, or (8) may not be responsible for the antihypertensive effect of these drugs.

Alpha methyl norepinephrine was found in the brains of animals treated with α methyl dopa.[4] The drug entered neurons and was itself decarboxylated and β-hydroxylated in a manner similar to the normal amino-acid substrate, dopa. The α methyl norepinephrine thus formed has been shown to displace norepinephrine, to be stored in its place in the nerve endings, and to be released in the place of norepinephrine by nerve stimulation.[41] It is a false neurotransmitter.

Investigators had assumed that false neurotransmitters would have less pharmacological potency than norepinephrine. Although Day and Rand[12] found this true of α methyl norepinephrine *in vitro,* subsequent studies showed it to be at least as potent as norepinephrine *in vivo.*[8,22,41,59] Kopin[25] provided a hypothesis to explain how sympathetic activity could be diminished in the presence of α methyl norepinephrine. He has preliminary data that indicate that: (1) The synthesis of catecholamines may be decreased by α methyl norepinephrine's inhibition of the rate-limiting enzyme tyrosine hydroxylase. (2) If α methyl norepinephrine is a more effective inhibitor than norepinephrine, then catecholamine synthesis may cease even when total stores of neurotransmitter are less than normal and the vesicles are only partly filled. (3) When the nerve is stimulated less than normal transmitter would be released. Another possibility is that the α methyl norepinephrine may be more tightly bound and less susceptible to release by nerve stimulation. Both these explanations are unproved and the actual mechanism remains unknown.

Alpha Methyl Meta Tyrosine and Metaraminol. The discovery of α methyl norepinephrine led the way for the study of other analogues of dopa, dopamine and norepinephrine. Alpha methyl meta tyrosine is taken up in nerve endings and its decarboxylated and β-hydroxylated derivative, metahydroxy norephedrine (metaraminol, Aramine®) (Chart 3), is a false neurotransmitter. The metaraminol formed and retained in the nerves displaces norepinephrine stoichiometrically and is released by nerve stimulation.[7,53] Infused metaraminol itself can be taken up by nerve endings and will displace equimolar amounts of norepinephrine from granules. When given rapidly enough (clinically useful doses) it is an effective pressor agent. The tachyphylaxis which commonly develops to the pressor effects of metaraminol is explained by the false neurotransmitter theory.[10] Metaraminol replaces norepinephrine but has only 5 per cent of its potency; thus, when the nerve endings store metaraminol predominantly, a sympathetic "blockade" is produced and may result in hypotension unresponsive to further metaraminol. An infusion of norepinephrine reestablishes catecholamine stores and restores normal sympathetic function. Under certain conditions, metaraminol and α methyl meta tyrosine may be *antihypertensive* agents.[7,24] Crout[10] studied six hypertensive patients and produced a fall in blood pressure in four of them with small oral doses of metaraminol. He also found large amounts of metaraminol in highly innervated structures, such as the heart and spleen, of patients who had been recently treated with the

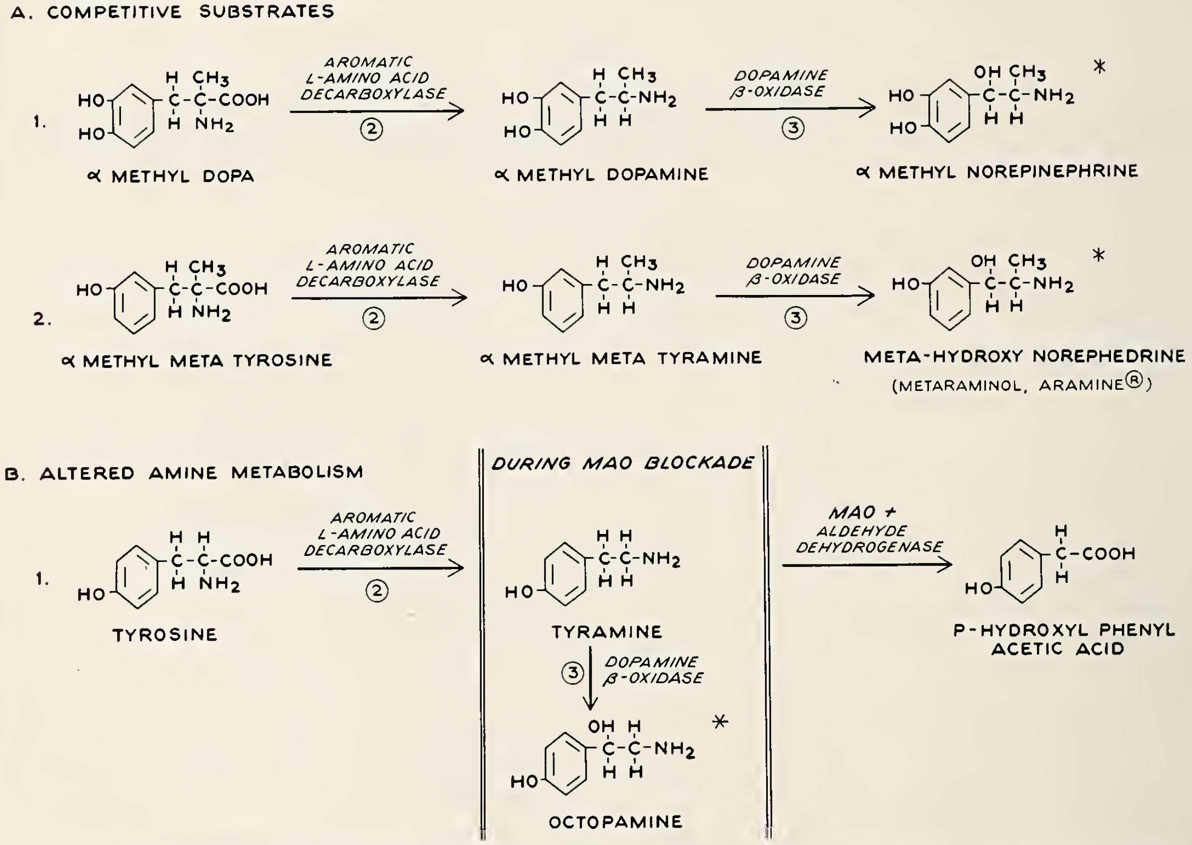

A. COMPETITIVE SUBSTRATES
1.
α METHYL DOPA
AROMATIC L-AMINO ACID DECARBOXYLASE
②
α METHYL DOPAMINE
DOPAMINE β-OXIDASE
③
α METHYL NOREPINEPHRINE
*
2.
α METHYL META TYROSINE
AROMATIC L-AMINO ACID DECARBOXYLASE
②
α METHYL META TYRAMINE
DOPAMINE β-OXIDASE
③
META-HYDROXY NOREPHEDRINE (METARAMINOL, ARAMINE®)
*
B. ALTERED AMINE METABOLISM
1.
TYROSINE
AROMATIC L-AMINO ACID DECARBOXYLASE
②
DURING MAO BLOCKADE
TYRAMINE
③ DOPAMINE β-OXIDASE
OCTOPAMINE
*
MAO + ALDEHYDE DEHYDROGENASE
P-HYDROXYL PHENYL ACETIC ACID

Chart 3.—Formation of false neurotransmitters. The false neurotransmitters (starred) are formed from amino acid substrates by the same enzymes utilized in the synthesis of norepinephrine. The numbered reactions refer to similar steps in Chart 1.

drug. Two important points are made with these studies: (1) Since metaraminol does not cross the blood-brain barrier, attention can be focused enentirely upon peripheral mechanisms of false neurotransmitters. In contrast, α methyl dopa and reserpine alter both the central and the peripheral stores of amine. (2) Since metaraminol has only one hydroxyl group on the aromatic ring, the catechol (dihydroxy benzene) structure is not necessary for a substance to masquerade as a transmitter.

Monoamine Oxidase Inhibitors. These substances, which were first used as antidepressant agents, produce postural hypotension. The hypotensive effects correlate with the degree of *in vivo* inhibition of MAO.[47] On first thought it may appear paradoxical that inhibition of MAO should result in diminished blood pressure when urinary concentrations of amines (including catecholamines) are increased.

Increased stores of catecholamines are accompanied by increased stores of other amines (false neurotransmitters).[26] These latter amines dilute the native neurotransmitter and under constant conditions of nerve stimulation, relatively less norepinephrine is released. In addition, since MAO is not responsible primarily for the biological inactivation of the *circulating* catecholamines, it is not surprising that neither the effects of sympathetic nerve stimulation nor of administered catecholamines is increased after MAO blockade. Within the nerve ending, however, MAO plays an important role in amine metabolism. It oxidizes amines to which it is exposed, including unbound catecholamines, dopamine, tyramine and many other monoamines (Chart 2).

Tyramine, the decarboxylation product of tyrosine, plays a dual role during MAO blockade. When given in high doses to normal persons, tyramine will release norepinephrine from peripheral nerves and may cause a detectable but brief rise in blood pressure. The brevity of the normal response is explained by the rapid destruction of tyramine by MAO. When the stores of catecholamines are increased, as in patients with pheochromocytoma, a given amount of tyramine will cause a greater than normal rise in blood pressure, but with normal duration. However, during MAO inhibition the destruction of tyramine is inhibited, total stores of norepinephrine increase and relatively small amounts of tyramine can provoke alarming hypertensive reactions. The "paradoxical" hypertension reported during MAO inhibition is usually associated with ingestion of a source of tyramine, such as aged cheese, wines, beer, marmite, broad beans or pickled herring.[43,52] Such hypertensive crises are a serious drawback to the clinical use of MAO inhibitors.

A metabolic product of tyramine can also produce a partial sympathetic blockade during MAO inhibition. Kopin and his associates[17,26] demonstrated that tyramine, protected by the presence of MAO inhibition, can be β-hydroxylated (Chart 3) and stored in the granulated vesicles in the nerve ending. The hydroxylated product, octopamine, is only 1 per cent as active as norepinephrine

TABLE 1.—*Comparative Properties of Guanethidine and Bretylium*

	Guanethidine	*Bretylium*
Normal postganglionic nerve conduction	+	+
Prevents release of catecholamines after nerve stimulation	+	+
Depletes catecholamine stores	+	− to ±
Uptake into nerve storage sites	+	+
1. Uptake prevented by reserpine	+	−
2. Uptake prevented by amphetamine	+	+
3. Uptake prevented by cocaine, imipramine and desipramine	+	−
Release from nerve endings:		
1. Normal nerve stimulation*	?	+
2. Released by acetylcholine	−	−
Other actions:		
1. Prevent uptake of catecholamines into nerve endings or storage sites	+	−
2. Initial sympathomimetic effect	+(β)	± (non-specific)

*See Reference 18.

and can act as a false neurotransmitter. Substantial octopamine is accumulated by the decarboxylation of normal amounts of tyrosine. The rapid oxidation of tyramine in normal subjects prevents an accumulation of octopamine.

Guanethidine and Bretylium

The sites of action of bretylium and guanethidine have not been clearly defined. Both drugs are taken up into nerve endings and produce sympathetic blockade by preventing release of norepinephrine. Although Burn[3] suggested that both drugs interfere with the acetylcholine "link" between the nerve impulse and norepinephrine release, bretylium and guanethidine seem to act in different ways (Table 1).

Guanethidine is probably taken up into the same specific storage sites as catecholamines. This statement is based on several observations: (1) Guanethidine releases catecholamines from storage sites. (2) The uptake of guanethidine is prevented by reserpine, cocaine, imipramine and desipramine.[11,28] (3) Reserpine releases stored guanethidine. (4) Guanethidine effects can be prevented or reversed by ephedrine, tyramine or metaraminol —drugs which release norepinephrine.[2]

Guanethidine has other actions that may be of primary importance to its effects and that are not shared by bretylium. Brodie and coworkers[2,5] postulated that guanethidine causes a persistent and direct depolarization of the sympathetic "presynaptic terminal" (nerve ending), which brings about both the discharge of catecholamines and the adrenergic blockade commencing before significant catecholamine depletion occurs. This is consistent with the fact that large doses of guanethidine administered parenterally release catecholamine and simultaneously block response to postganglionic nerve stimulation.[2]

Several factors will affect the use of either drug in hypertensive patients. Patients pretreated with reserpine, cocaine, imipramine and desipramine, drugs that can block the uptake of guanethidine, may be expected to experience less than usual sympathetic blockade with guanethidine. The same patients would respond normally to bretylium because its uptake into nerve endings is unaffected by reserpine and the other drugs mentioned above. If patients are given amphetamine, which blocks the entry of both bretylium and guanethidine into the nerve ending,[11] neither may produce the anticipated effects.

Inhibitors of Catecholamine Synthesis

No drug now available will lower blood pressure by decreasing synthesis of catecholamines. The futility of inhibiting enzymes which are not rate-limiting has been well illustrated by the inability of potent decarboxylase inhibitors to decrease catecholamine synthesis or blood pressure. Recently, however, tyrosine hydroxylase has been isolated from tissues and its properties defined by Nagatsue and coworkers.[42] Isolation of the enzyme has allowed successful search for a potent inhibitor—α methyl tyrosine (Chart 3). Engelman and coworkers[16] administered the drug to 20 patients and found that catecholamine synthesis, as measured by decreased excretion of vanilmandelic acid, was reduced in all patients by as much as 75 per cent. The 14 patients with pheochromocytoma improved symptomatically but at least four of the six with essential hypertension did not. At present, α methyl tyrosine is not useful for treating hypertension. Another drug that blocks catecholamine synthesis *in vivo* is disulfiram (Antabuse®), which inhibits β-hydroxylation and makes this step rate-limiting.[40] Disulfiram has not been used yet in the treatment of hypertension.

Drugs Acting Distal to the Postganglionic Nerve Fiber (Figure 1, E)

Alpha and Beta-Adrenergic Blocking Agents

An ideal adrenergic blocking agent would compete with circulating catecholamines for effector sites and have no direct effects of its own. The receptors are of two kinds: (1) Alpha-receptor stimulation produces vasoconstriction of small arteries and veins, while (2) beta-receptor stimulation produces vasodilatation of arteries in skeletal muscle, increase in the heart rate and force of contraction, bronchial dilatation and most of the metabolic effects attributed to catecholamines (Table 2). Norepinephrine is primarily an α stimulator while isoproterenol is primarily a β stimulator. In fact, different sympathomimetic agents vary only quantitatively in their action on receptors, and large doses of α stimulators produce β effects and vice versa. On the other hand, adrenergic blocking agents may produce relatively selective inhibition of either α or β receptors.

Because high concentrations of circulating catecholamines are not responsible for essential hypertension, α-blockade would not be expected to be and is not effective in the treatment of that condition. However, phentolamine (Regitine®), an

TABLE 2.—*Classification of the Major Alpha and Beta Adrenergic Responses*

Organ	*Effect*	*Alpha (norepinephrine)*	*Beta (Isoproterenol)*
Heart	a. Increase rate of S.A. node discharge	0	+
	b. Increase atrial conductivity and contractility	0	+
	c. Increase conduction velocity of A-V Node	0	+
	d. Increase ventricular automaticity, conduction velocity and contractility	0	+
Lungs	a. Dilate bronchial musculature	0	+
Blood Vessels	a. Dilate arteries	0	+
	b. Constrict small arteries and venules	+	0
Metabolic Effects	a. Increase FBS, FFA and stimulate formation of cyclic 3′, 5′ AMP	0	+(?)

S.A.=Sino-atrial; FFB=Fasting Blood Sugar; FFB=Free Fatty Acids; AMP=Adenocine Monophosphate.

α-blocking agent, does have more value than for the simple diagnosis and management of a patient with pheochromocytoma. Guanethidine, parenteral reserpine and α methyl dopa all release stored catecholamines; and because of the hypertension they may produce, they are contraindicated in patients with pheochromocytoma. Before using such drugs in patients with malignant hypertension, we give intravenous injections of from 1 to 5 mg of phentolamine. Only when phentolamine produces a minimal decrease in blood pressure should the potent antihypertensive agents be given.

In recent years, there has been considerable literature on the evaluation of β-blocking agents in various diseases. Propranalol has been administered to patients with essential hypertension.[62] The drug produces a substantial fall in blood pressure in about a third of the patients treated. The mechanism of blood pressure lowering is obscure but does not always depend on the direct myocardial depressant effects which the drug possesses in addition to its β-blocking qualities. The "therapeutic" effects could conceivably be due to other known effects of the drug such as its direct depression of peripheral or postganglionic neural transmission, to its metabolic effects or to as yet unrecognized direct arterial or central actions. Regardless of its known additional actions, the drug probably does not lower blood pressure by inhibiting β-receptors. In addition, it is dangerous to use the drug indiscriminately in a hypertensive patient because it constricts coronary arteries, may convert occult to overt congestive heart failure, and has not been tested in humans over long periods. It is an experimental drug and should be used carefully.

Other Agents

Despite intensive studies by numerous investigators, the mechanism by which thiazide diuretics produce sustained hypotension is unknown. During initial therapy some of the antihypertensive effects must be related to a decreased intravascular volume. However, blood volume returns to normal during continued effective therapy. In addition, diazoxide, an investigational non-diuretic thiazide, has potent hypotensive properties. These drugs have no similarity to those affecting the sympathetic nervous system and probably work by a variety of mechanisms, among which may be direct arteriolar dilatation.[50] One of the most intriguing and basic unanswered questions regarding these drugs is the mechanism by which they potentiate the hypotensive effects of almost all other antihypertensive agents (see below).

Hydralazine drugs are among the most thoroughly investigated. They are important because they are the only ones available that produce their major hypotensive effects by direct decreases in peripheral vascular resistance without significant antisympathetic action. Unfortunately, currently available compounds in this category are not used extensively because tolerance occurs rapidly, they are relatively impotent and they produce a lupus erythematosus-like syndrome when used in high doses.[23] They may still have a place, however, when used in low doses in mild hypertension or parenterally in the initial therapy of malignant hypertension.

Clinical Comments

There have been extensive critical and informative reviews on the considerations regarding choice, dosage, useful and potentially dangerous combinations of antihypertensive agents in various clinical situations.[20,58] This review stresses the principles of clinical pharmacology not usually discussed in such reviews. We will only touch on more recent investigations and broad clinical considerations which derive from the previous section of this report.

Antihypertensive Agents for Patients With Mild Disease

Reserpine and thiazides are the most commonly used agents in this group. Phenobarbital is used but is no more effective than a placebo.[9] Rauwolfia alkaloids are effective in daily oral doses equivalent to 0.5 mg of reserpine. They are even more effective when combined with thiazide diuretics.[56,57] The most dangerous complication of chronic oral use of reserpine is severe psychic depression which may require hospitalization or lead to suicide. Other side effects include increased gastric secretion and mucosal ulceration, diarrhea, abdominal cramps and nasal stuffiness, most of which can be accounted for by decreased sympathetic "tone" with relative increase in parasympathetic activity.

Despite claims of superiority of different products by pharmaceutical companies, all thiazide diuretics produce similar therapeutic and "side" effects. Only the dosage varies, and this consideration is of minor clinical importance. Chlorothiazide (Diuril®) should be given in divided doses because its duration of action is about six to eight hours. Some congeners (chlorthalidone and polythiazide) have a longer duration of action and can be given once daily. Potassium depletion with consequent metabolic alkalosis and precipitation of digitalis toxicity may accompany thiazide treatment, and it is probably wise to give supplemental doses of potassium chloride. Enteric-coated potassium chloride tablets are variably absorbed, may cause strictures and ulceration of the small bowel and should be avoided.[27] Hyperuricemia resulting in clinical gout, hyperglycemia with worsening of existent diabetes, pancreatitis, jaundice, photosensitivity, skin rash and bone marrow depression can also result from thiazide administration.

Antihypertensive Agents for Patients with Moderate to Severe Disease

In many patients potent drugs, which include MAO inhibitors, α methyl dopa, guanethidine, parenteral reserpine, and ganglionic blockers, are needed to control the blood pressure.

Alpha methyl dopa is one of the most useful antihypertensive agents available. During effective therapy, cardiac output is well maintained and reduction in blood pressure is largely due to a fall in peripheral resistance.[14] This drug, as opposed to pargyline and guanethidine, produces a considerably greater decrease in supine pressure for a given decrease in standing blood pressure. Thus it produces significantly less symptomatic orthostatic hypotension.[46]

Some studies have shown that renal blood flow also is maintained in spite of considerable reduction in renal artery perfusion pressure.[34,38] It would seem, therefore, that the drug should be chosen for treatment of hypertensive patients with compromised renal function. Oral absorption is variable, and effective daily doses range from 500 to 3,000 mg. Maximum effects are seen in four to eight hours after an oral dose or three to four hours after intravenous administration. The duration of effect of the oral dose is usually no longer than 24 hours. Alpha methyl dopa is excreted largely by the kidney, and therefore should be used in smaller than usual doses in patients with renal failure. Common side effects are transient drowsiness and fatigue, fever with or without abnormalities in liver function, diarrhea or constipation, and skin rash. Unusual effects are lactation[51] and after months of therapy, development of reversible positive reaction to Coomb's test with or without hemolytic anemia.[37] Unfortunately, some patients become tolerant to α methyl dopa after prolonged treatment, necessitating a change in medication.

We have found that α methyl dopa is also a safe, effective and predictable agent in the management of most acute hypertensive emergencies. Intravenous doses of 500 to 1,000 mg will promote a smooth lowering of systolic and diastolic pressures, usually beginning within two hours. When patients have not received previous therapy, we have not observed uncontrollable hypotension, although occasionally one who has been treated with reserpine may be unduly sensitive to α methyl dopa. There are at least two reports of paradoxical rises in blood pressures after administration of Aldomet® (alpha methyl dopa). Such reactions are uncommon and are presumably due to release of norepinephrine.[33,36]

During hypertensive encephalopathic episodes, reserpine has been used intravenously or intramuscularly in doses up to 5 mg. Administered by this route, reserpine not only acts by depleting catecholamines but also has direct vasodilating effects on arterioles.[48] However, the onset, severity and duration of the effects of parenteral reserpine are unpredictable and occasionally dangerously dramatic.[19,31] Smoothly controlling the profound hypotension which may occur after as little as 0.1 mg may not be easy. Sympathomimetic drugs which depend on release of stored catecholamines (for example, metaraminol) will have unpredictable effects because such stores are compromised by previous treatment. Also, direct-acting sympathomimetic drugs (for example, norepinephrine) will have an enhanced effect because reserpine prevents their entrance into granules. Phenylephrine (Neo-Synephrine®) is a reasonable drug to use because it has moderately weak but direct sympathomimetic effects. Since there are more potent, short-acting and reliable agents available for use in malignant hypertension (trimethapan, α methyl dopa), perhaps the use of parenteral reserpine should be avoided.

Pargyline (Eutonyl®) is the most widely used MAO inhibitor. Its action is slow—one to three weeks for maximum effects, and the effects lasting up to three weeks after administration has been discontinued. The predominant cardiovascular effect of chronic treatment is a decrease in peripheral vascular resistance produced by sympathetic blockade. Renal blood flow is slightly reduced, cardiac output is quite well maintained[1] and mood-elevating effects are prominent. Untoward effects include excessive stimulation of the central nervous system, resulting in insomnia, nightmares and muscular twitching. Undesirable aspects of sympathetic blockade are similar to those seen during administration of other antiadrenergic agents and include symptomatic orthostatic hypotension and increased gastrointestinal motility. As mentioned earlier, the most serious objection to the use of pargyline is precipitation of hypertensive crises when norepinephrine releasers such as tyramine-containing foods are ingested or when amphetamines, phenylephrine (Neo-Synephrine®), mephentermine (Wyamine®), metaraminol (Aramine®), phenmetrazine (Preludin®) and α methyl dopa (Aldomet®) are administered.[44] The rational and effective treatment of these attacks is prompt administration of the α-blocker, phentolamine. Narcotics, antihistamines, other MAO inhibitors, imipramine and derivatives, sedatives and hypoglycemic agents may also produce undesirable effects when used in conjunction with an MAO inhibitor. Because of the severity of these undesirable effects, we rarely use MAO inhibitors for antihypertensive therapy.

Guanethidine (Ismelin®) is used when hypertension is uncontrolled by other agents. The hypotensive effect of guanethidine is predominantly due to decreased cardiac output. Postural hypotension may be pronounced. However, in patients with cardiac failure and hypertension, the cardiac output may increase because of the decreased work demand on the heart after blood pressure is lowered. Renal blood flow and glomerular filtration rate are decreased by guanethidine.[21] Effective doses range from 10 to 400 mg daily. The maximum effect of a given dose may not be seen for four to seven days and may last for four to 10 days after the drug is discontinued. Because of the long half-life of the drug, dosages should be adjusted only after several days at the same level. One experimental approach for hastening the control of the blood pressure has been to give guanethidine intramuscularly.[29] However, the variability of individual responses combined with the prolonged action of the drug would seem to make this method of treatment potentially dangerous. As with other drugs that block the sympathetic nervous system, undesirable effects are almost all extensions of the therapeutic effects. Thus orthostatic dizziness or syncope (especially in the morning or after exercise), diarrhea, failure to ejaculate, dry mouth, nasal stuffiness, and bradycardia are common. There is one report of a periarteritis nodosa-like syndrome developing in three patients taking the drug.[13]

Ganglionic blocking drugs are used infrequently in chronic therapy. The most commonly used agents are pentolinium tartrate (Ansolysen®), chlorisondamine (Ecolid®), and mecamylamine (Inversine®). The latter is the most completely absorbed. Side effects result from non-specific autonomic blockade and include dry mouth, blurred vision, constipation or paralytic ileus, impotence and urinary retention. The effects related to lack of parasympathetic effect can be helped sometimes with small doses of parasympathomimetics such as pilocarpine or bethanechol (Urecholine®). The short-acting blocker, trimethaphan camphorsulfonate (Arfonad®), is the preferred drug for treating hypertensive emergencies when slower acting drugs are undesirable. The dose of trimethaphan is variable and it must be given intravenously and titrated by a physician.[15]

Combinations of Drugs

All the sympathetic blocking agents tend to cause fluid retention. Thiazides in combination with these drugs help counteract this tendency and allow the use of significantly smaller doses of the more potent drugs. Fewer side effects occur and, unless there is a contraindication to their use, thiazides should be part of the standard antihypertensive regimen. Usefulness of other combinations of drugs is not clear. Some, such as MAO inhibitors and α methyl dopa, are hazardous. Guanethidine and α methyl dopa have been used together with success, but the incidence of adverse side effects is high. The combination may have a place, however, when neither drug alone will suffice.[30]

GENERIC AND TRADE NAMES OF DRUGS

Trimethaphan—*Arfonad*®.
Mecamylamine—*Inversine*®.
Hexamethonium—*Methium*®.
Alpha methyl dopa—*Aldomet*®.
Metaraminol—*Aramine*®.
Guanethidine—*Ismelin*®.
Imipramine—*Tofrãnil*®.
Desipramine—*Norpramine*®, *Pertofrane*®.
Disulfiram—*Antabuse*®.
Norepinephrine—*Levophed*®.
Isoproterenol—*Isuprel*®.
Phentolamine—*Regitine*®.
Chlorothiazide—*Diuril*®.
Polythiazide—*Renese*®.
Chlorthalidone—*Hygroton*®.
Hydralazine—*Apresoline*®.
Phenylephrine—*Neo-Synephrine*®.
Pargyline—*Eutonyl*®.
Mephentermine—*Wyamine*®.
Phenmetrazine—*Preludin*®.
Pentolinium—*Ansolysen*®.
Chlorisondamine—*Ecolid*®.
Bethanechol—*Urecholine*®.

REFERENCES

1. Brest, A. N., Onesti, G., Heider, C., and Moyer, J. H.: Cardiac and renal hemodynamic response to pargyline, Ann. N.Y. Acad. Sci., 107:1016-1022, 8 July 1963.

2. Brodie, B. B., Chang, C. C., and Costa, E.: On the mechanism of action of guanethidine and bretylium, Brit. J. Pharmacol., 25:171-178, August 1965.

3. Burn, J. H., and Rand, M. J.: Acetylcholine in adrenergic transmission, Ann. Rev. Pharmacol., 5:163-182, 1965.

4. Carlsson, A., and Lindqvist, M.: In vivo decarboxylation of α-methyl-dopa and α-methylmetatyrosine, Acta Physiol. Scand., 54:87-94, January 1962.

5. Chang, C. C., Costa, E., and Brodie, B. B.: Interaction of guanethidine with adrenergic neurons, J. Pharmacol., 147:303-312, March 1965.

6. Chidsey, C. A., Braunwald, E., Morrow, A. G., and Mason, D. T.: Myocardial norepinephrine concentration in man, effects of reserpine and of congestive heart failure, New Engl. J. Med., 268:653-658, 26 September 1963.

7. Cohen, R. A., Kopin, I. J., Creveling, C. R., Musacchio, J. M., Fischer, J. E., Crout, J. R., and Gill, J. R.: False neurochemical transmitters, combined clinical staff conference at the National Institutes of Health, Ann. Int. Med., 65:347-362, August 1966.

8. Conradi, E. C., Gaffney, T. E., Fink, D. A., and Vangrow, J. S.: Reversal of sympathetic nerve blockade: A comparison of DOPA, dopamine, and norepinephrine with their α-methylated analogues, J. Pharmacol., 150:26-33, October 1965.

9. Cranston, W. W.: General Principles in Antihypertensive Treatment. Antihypertensive Therapy, An International Symposium, Edited by F. Gross, Springer-Verlag, Berlin 1966, pp. 184-192.

10. Crout, J. R.: Substitute adrenergic transmitters, a newly appreciated mechanism of antihypertensive drugs, Circulation Res. (Suppl. 1), 18:120-130, June 1966.

11. Day, M. D., and Rand, M. J.: Evidence for a competitive antagonism of guanethidine by dexamphetamine, Brit. J. Pharmacol., 20:17-28, February 1963.

12. Day, M. D., and Rand, M. J.: Some observations on the pharmacology of α-methyldopa, Brit. J. Pharmacol., 22:72-86, February 1964.

13. Dewar, H. A., and Peaston, M. J. T.: Three cases resembling periarteritis nodosa arising during treatment with guanethidine, Brit. Med. J., 2:609-611, 5 September 1964.

14. Dollery, C. T.: Methyldopa in the treatment of hypertension, Prog. Cardiovasc. Dis., 8:278-289, November 1965.

15. Drugs for hypertensive crises, Medical Letter on Drugs and Therapeutics, 6:97-98, 4 December 1964.

16. Engelman, K., and Sjoerdsma, A.: Inhibition of catecholamine biosynthesis in man, Circulation Res. (Suppl. 1), 18:104-109, June 1966.

17. Fischer, J. E., Horst, W. D., and Kopin, I.: β-hydroxylated sympathomimetic amines as false neurotransmitters, Brit. J. Pharmacol., 24:477-484, April 1965.

18. Fischer, J. E., Weise, V. K., and Kopin, I. J.: Release of tritiated bretylium by sympathetic nerve stimulation, Nature, 209:778-779, 19 February 1966.

19. Fletcher, G. F.: Hypotensive reactions after small doses of reserpine given parenterally, New Engl. J. Med., 268:309-310, 7 February 1963.

20. Freis, E. D.: Antihypertensive agents I, II, and III, New Engl. J. Med., 266:507-509, 607-609, 775-777, 8 March, 22 March, and 12 April 1962.

21. Freis, E. D.: Guanethidine, Prog. Cardiovasc. Dis., 8:183-193, September 1965.

22. Haefly, W., Hürlimann, A., and Thoenen, H.: The effect of stimulation of sympathetic nerves in the cat treated with reserpine, α-methyldopa, and α-methyl metatyrosine, Brit. J. Pharmacol., 26:172-185, January 1966.

23. Hollander, W., and Wilkins, R.: The pharmacology and clinical use of rauwolfia, hydralazine, thiazides, and aldosterone antagonists in arterial hypertension, Prog. Cardiovasc. Dis., 8:291-318, January 1966.

24. Horwitz, D., and Sjoerdsma, A.: Effects of α-methyl metatyrosine intravenously in man, Life Sciences, 3:41-48, 1964.

25. Kopin, I. J.: Speaker at the Medical Staff Conference, University of California Medical Center, San Francisco, 13 July 1966.

26. Kopin, I. J., Fischer, J. E., Musacchio, J. M., Horst, W. D., and Weise, V. K.: "False neurochemical transmitters" and the mechanism of sympathetic blockade by monoamine oxidase inhibitors, J. Pharmacol., 147:186-193, February 1965.

27. Lawrason, F. D., Alpert, E., Mohr, F. L., and Mac-

Mahon, F. G.: Ulcerative obstructive lesions of the small intestine, J.A.M.A., 191: 641-644, 22 February 1965.

28. Leishman, A. W. D., Matthews, H. L., and Smith, A. J.: Antagonism of guanethidine by imipramine, Lancet, 1:112, 12 January 1963.

29. Leishman, A. W. D., and Sandler, G.: Hastening the control of blood pressure by guanethidine, Lancet, 1:668-70, 27 March 1965.

30. Leonard, J. W., Gifford, R. W., and Humphrey, D. C.: Treatment of hypertension with methyldopa alone or combined with diuretics and/or guanethidine, Am. Heart J., 65:610-618, May 1965.

31. Leonberg, S. C., Green, J. B., and Elliot, F. A.: The response of stroke patients to very small doses of parenteral reserpine, Ann. Int. Med., 60:866-870, May 1964.

32. Levine, R. J., and Sjoerdsma, A.: Dissociation of the decarboxylase-inhibiting and norepinephrine-depleting effects of α-methyl-dopa, α-ethyl-dopa, 4-bromo-3-hydroxy-benzyloxyamine and related substances, J. Pharmacol., 146:42-47, October 1964.

33. Levine, R. J., and Strauch, B. S.: Hypertensive responses to methyldopa, New Engl. J. Med., 275:946-948, 27 October 1966.

34. Luke, R. G.: Methyldopa in treatment of hypertension due to chronic renal disease, Brit. Med. J., 1:27-30, 4 January 1964.

35. Mason, D. T., and Braunwald, E.: Effects of guanethidine, reserpine, and methyldopa on reflex venous and arterial constriction in man, J. Clin. Invest., 43:1449-1463, July 1964.

36. Melmon, K. L., and Morrelli, H.: Unpublished data.

37. Methyldopa and hemolytic anemia, an editorial, Lancet, 2:151-152, 16 July 1966.

38. Morin, Y., Turmel, L., and Fortier, J.: Methyldopa: Clinical studies in arterial hypertension, Am. J. Med. Sci., 248:633-639, December 1964.

39. Musacchio, J. M., Fischer, J. E., and Kopin, I. J.: Subcellular distribution and release by sympathetic nerve stimulation of dopamine and α-methyldopamine, J. Pharmacol., 152:51-55, April 1966.

40. Musacchio, J. M., Goldstein, M., Anagnoste, B., Poch, G., and Kopin, I. J.: Inhibition of dopamine-β-hydroxylase by disulfiram in vivo, J. Pharmacol., 152:56-61, April 1966.

41. Muscholl, E., and Maitre, L.: Release by sympathetic stimulation of α-methylnoradrenaline stored in the heart after administration of α-methyldopa, Experientia, 19:658-659, December 1963.

42. Nagatsu, T., Levitt, M., and Udenfriend, S.: Tyrosine hydroxylase. The initial step in norepinephrine biosynthesis, J. Biol. Chem., 239:2910-2917, September 1964.

43. Neussle, W. F., Norman, F. C., and Miller, H. E.: Pickled herring and tranylcypromine reactions, J.A.M.A., 192:726-727, 24 May 1965.

44. Oates, J. A., and Doctor, R. B.: Antihypertensive agents which impair adrenergic neurone transmission, Postgraduate Medicine, 37:58-64, January 1965.

45. Oates, J. A., Gillespie, S., Udenfriend, S., and Sjoerdsma, A.: Decarboxylase inhibition and blood pressure reduction by α-methyl-3,4-dihydroxy,d,1-phenylalanine, Science, 131:1890-1891, 24 June 1960.

46. Oates, J. A., Seligman, A. W., Clark, M. A., Rousseau, P., and Lee, R. E.: The relative efficacy of guanethidine, methyldopa and pargyline as antihypertensive agents, New Engl. J. Med., 273:729-734, 30 September 1965.

47. Orvis, H. H., Tamanga, I. G., Horwitz, D., and Thomas, R.: Correlation of hypotensive effects and urinary tryptamine levels during pargyline therapy, Ann. N.Y. Acad. Sci., 107:958-965, 8 July 1963.

48. Parks, V. J.: Mechanism of vasodilator action of reserpine in man, Clin. Sci., 20:289-295, June 1961.

49. Peart, W. S.: Catecholamines and hypertension, Pharmacol. Rev., 18:667-672, March 1966.

50. Peters, G.: Pharmacology of Diuretics. Antihypertensive Therapy, An International Symposium, Edited by F. Gross, Springer-Verlag, Berlin 1966, pp. 31-57.

51. Pettinger, W. A., Horwitz, D., and Sjoerdsma, A.: Lactation due to methyldopa, Brit. Med. J., 1:1460, 1 June 1963.

52. Pressor attacks during treatment with monoamine-oxidase inhibitors, an editorial in Lancet, 1:945-946, 1 May 1965.

53. Shore, P. A., Busfield, D., and Alpers, H. S.: Binding and release of metaraminol: Mechanism of norepinephrine depletion by α-methylmetatyrosine and related agents, J. Pharmacol., 146:194-199, November 1964.

54. Sjoerdsma, A.: Relationships between alterations in amine metabolism and blood pressure, Circulation Res., 9:734-745, May 1961.

55. Sjoerdsma, A., Vendsalu, A., and Engelman, K.: Studies on the metabolism and mechanism of action of methyldopa, Circulation, 28:492-502, October 1963.

56. Smith, W. M., Backman, B., Galante, J. G., Hanowell, E. G., Johnson, W. P., Koch, C. E., Jr., Korfmacher, S. D., Thurm, R. H., and Bromer, L.: Cooperative clinical trial of α-methyldopa, Ann. Int. Med., 65: 657-671, October 1966.

57. Smith, W. M., Damato, A. N., Galluzzi, N. S., Garfield, C. F., Hanowell, E. G., Shinson, W. M., Thurm, R. H., Walsh, J. J., and Bromer, L.: The evaluation of antihypertensive therapy—cooperative clinical trial method, Ann. Int. Med., 61:829-846, November 1964.

58. Sokolow, M., and Perloff, D.: The choice of drugs and the management of essential hypertension, Prog. Cardiovasc. Dis., 8:253-257, November 1965.

59. Stone, C. A., and Porter, C. C.: Methyldopa and adrenergic nerve function, Pharmacol. Rev., 18:569-576, March 1966.

60. Tcherdakoff, P., Vaysse, J., Lacombe, M., Oudéa, P., Mourad, J., Tarrette, F., and Milliez, P.: The Present Status of Symptomatic Surgical Treatment for Arterial Hypertension. Antihypertensive Therapy, An International Symposium, Edited by F. Gross, Springer-Verlag, Berlin 1966, pp. 394-418.

61. Udenfriend, S.: Tyrosine hydroxylase, Pharmacol. Rev., 18:43-52, March 1966.

62. Waal, H. J.: Hypotensive action of propranolol, Clin. Pharmacol. Ther., 7:588-598, September-October 1966.

63. Wurtman, R. J.: Catecholamines, New Engl. J. Med., 273:637-646, 693-700, 746-753, 16, 23, and 30 September 1965.

CASE REPORTS

Urticaria from Contact With Water

THEODORE A. TROMOVITCH, M.D.,
San Francisco

RECENTLY TWO PATIENTS complained of developing hives on contact with water.

Reports of Cases

CASE 1.—A 27-year-old white woman had history since age nine or earlier of the development of small white papules surrounded by bright red rings whenever the skin remained in contact with water for more than five minutes. The lesions developed regardless of the temperature of the water and occurred on bathing, showering or swimming. They were not associated with heating of the body, exercise or emotional upset. There was no daily, weekly or seasonal variation or pattern of occurrence. Otherwise the patient was in excellent health and through the years had learned to minimize the problem. She avoided swimming and learned to bathe by showering rapidly and drying herself quickly. Longer showers were followed within 10 minutes by the development of pruritic lesions that persisted for a half hour to an hour. Even washing her face caused lesions unless she rapidly dried after washing. The lesions had occurred at one time or another on almost all parts of the body except the palms and soles, but mainly on the trunk. The patient said a brother had a similar hypersensitivity response, but the brother was not available for examination.

When physical examination was carried out no evidence of skin disease was observed. A number of tests were done on separate skin areas known to have been affected previously. Application of tap water at room temperature for five minutes caused lesions of cholinergic urticaria type—a central white papule surrounded by a zone of erythema. The papules were 2 to 3 mm in diameter and the area of erythema 3 to 5 cm. Application of distilled water evoked the same reaction. Application of alcohol under an impervious dressing so as to prevent evaporation produced no response. Nor was there reaction to application of ice cubes in a thin plastic film so that the skin became quite cold but no moisture touched it. Application of a hot pack contained in a thin plastic film caused the skin to warm to the point of erythema, but no lesions appeared. Friction to the areas had no effect. No reaction on the skin of a normal person was noted when it was rubbed with a gauze pad that had first been soaked in distilled water and used to swab the skin of the patient. However, where the patient's skin remained wet after it was swabbed, urticarial lesions developed.

CASE 2.—The patient, a 24-year-old white woman, had first noted two years previously, during the sixth month of pregnancy, that hive-like lesions developed wherever her skin was in contact with water for longer than 30 minutes. Baths or showers for shorter periods did not cause the lesions, which were white papules surrounded by red rings. Such lesions were not produced by exercise, emotional upset or hot weather. The lesions occurred anywhere on the body except the palms and soles but were mainly on the trunk. Other members of the family did not have similar problems.

Application of tap water and distilled water for 30 minutes produced characteristic urticarial lesions of cholinergic type, but application for 5 or 15 minutes did not produce lesions. Application of heat, cold and alcohol (as described in Case 1) did not cause lesions.

From the Department of Medicine (Dermatology), University of California Medical Center, San Francisco.

Submitted 6 September 1966.

Reprint requests to: 909 Hyde Street, San Francisco 94109.

Discussion

Shelley and Rawnsley[1] described the first three cases of "aquagenic urticaria" and suggested that water is the key exogenous factor in causing classical cholinergic urticaria in such cases. They postulated that a toxic substance is formed by water acting on sebum or the sebaceous gland. This toxic substance, they speculated, is absorbed, causes perifollicular mast cells to degranulate, releasing histamine and resulting in cholinergic urticaria.

It is well established that "foreign" substances that do not occur within the normally developing epidermis or in normal sebaceous secretions, sometimes do appear on the skin—for example, substances associated with bacterial action. The potential factor in the present cases and others like them may therefore be a product of bacterial breakdown. If this is the case, it most probably is a water-soluble substance, for water incited the lesions and alcohol did not.

Testing of many more cases will be required to determine whether all cholinergic urticaria is aquagenic urticaria or aquagenic urticaria is one specific type.

At present the best therapy is avoidance of unnecessary contact with water. Antihistamines by mouth give partial control.[1]

REFERENCE

1. Shelley, W. B., and Rawnsley, H. M.: Aquagenic urticaria, contact sensitivity to water, J.A.M.A., 189:895-898, 21 September 1964.

Duodenal Ulcer with Massive Hemorrhage Complicating Pregnancy

CHARLES G. KING, M.D., ROBERT STEEDMAN, M.D., AND J. EDWARD BERK, M.D.,
Los Angeles

RARELY DO COMPLICATIONS of peptic ulcer occur during pregnancy. In the present case a massive hemorrhage from duodenal ulcer that occurred in the fifth month of pregnancy was successfully treated surgically.

Report of a Case

A 30-year-old pregnant Caucasian woman was admitted to the Medical Service of the Los Angeles County General Hospital, Unit II, on 14 April 1966 at 5 a.m. because of hematemesis and melena that had occurred two hours earlier. This was her fourth pregnancy. In June 1963, during the sixth month of her second pregnancy, she was put in hospital for hematemesis, which responded readily to medical management. She also said she had had epigastric distress for the past nine years. To relieve this distress, she ingested approximately two quarts of milk daily together with numerous antacids. Heartburn and regurgitation had also been noted when lying down. Roentgenographic examination of the upper gastrointestinal tract in 1960 was said to have revealed deformity of the duodenal cap with delayed passage of barium and hypertrophic gastritis.

The patient took from eight to ten tablets of acetylsalicylic acid daily because of pain in the back. She had had bronchial asthma intermittently since the age of five and she took Tedral®* for it. She also admitted to having been addicted to heroin.

Her last normal menstrual period was early in September 1965. The expected day of confinement was mid-June 1966. There had been no vaginal bleeding during this gestation and no symptoms to suggest toxemia. She had not had prenatal care.

The blood pressure at the time of admission was 140/80 mm of mercury, the pulse rate 130 beats per minute and the respiratory rate 20 per minute. The conjunctivae were pale. The breasts were enlarged with darkly pigmented areolae. The anteroposterior diameter of the chest was increased and the lung sounds were distant but clear. No abnormality was noted on cardiac examination. Minimal epigastric tenderness was noted. The height of the fundus was 2 cm above the umbilicus. No fetal heart tones were heard. Bowel sounds were hyperactive. There were old scars in both antecubital areas. On pelvic examination the cervix was closed and soft and the uterus was enlarged.

The initial hematocrit was 32 volumes per cent. Bright red blood was vomited several times shortly after admission and eight units of blood were administered in the first three hours. Because of re-

---

From the Departments of Medicine and Surgery, University of California, California College of Medicine, and the Los Angeles County General Hospital, Unit II, Los Angeles.

Submitted 24 October 1966.

Reprint requests to: Department of Medicine, University of California, California College of Medicine, 1721 Griffin Avenue, Los Angeles 90031 (Dr. Berk).

*A mixture of theophylline, ephedrine and phenobarbital.

peated hematemesis and the large amount of blood replacement required, surgical treatment was elected. At operation, gastrotomy and duodenotomy revealed a bleeding duodenal ulcer. The ulcer bed was ligated and vagotomy and pyloroplasty were performed.

The patient did well postoperatively except for dehiscence of a small part of the wound, which was managed conservatively. The fetal heart tones remained inaudible after operation, but a fetal electrocardiogram demonstrated a heart rate of 140 per minute.

The patient was discharged 28 April 1966 in good condition and with pregnancy uninterrupted. She was readmitted in labor on 27 July 1966 with signs of preeclampsia. The blood pressure was 240/130 mm. Magnesium sulfate was administered and she was delivered of a viable male infant the following morning. The blood pressure taken 25 minutes after delivery was 140/80 mm. Both the patient and the baby were discharged 31 July 1966.

Review of Literature

The frequency of active peptic ulcer disease during pregnancy is difficult to state because of reluctance to expose pregnant women to radiation. A summary of some reports which have appeared in the literature dealing with the frequency of occurrence of complicated and uncomplicated peptic ulcer disease in pregnant women is given in Table 1. These reports have been chosen as representative because the reference case material in each was sizable.

MacNalty[14] studied 770 puerperal deaths in 1937 and found only one death from hemorrhage due to gastric ulcer. Hooker[9] studied 350,000 pregnancies over a three-year period from 1930 to 1932 and found only one death from perforation of a gastric ulcer. Sandweiss[18] studied 70,310 pregnancies seen in the 10-year period from 1928 to 1937 in various Detroit hospitals and found one death due to perforated duodenal ulcer.

In 1943 Sandweiss,[19] in a review of the literature on deaths due to perforation and hemorrhage from gastroduodenal ulcer during pregnancy and the puerperium, found only 14 cases. In seven cases they were gastric ulcers, and in seven duodenal. All of the 14 patients died postpartum, and premature delivery occurred in four. In virtually every case the diagnosis of ulcer was not considered because of its rarity, and in only one of the patients was a history of previous ulcer symptoms elicited.

Johnson[11] reviewed 12,041 pregnancies in private patients seen between 1939 and 1953 and found two with duodenal ulcer, one complicated by hemorrhage and one by perforation. O'Connell[16] reported three cases of duodenal ulcer occurring in women who had no symptoms before becoming pregnant. Durst[7] reviewed all the deliveries at the Milwaukee County Hospital and seven other hospitals in Milwaukee for the years 1944 to 1953 in order to determine the incidence of peptic ulcer complicating pregnancy. He found six cases, only one of them fatal, in 149,491 deliveries. In 1948 Bernstine[3] reported four cases of hemorrhage from peptic ulcer in pregnant women. All of these women had been treated with progesterone before the onset of the gastrointestinal hemorrhage. All responded to conservative management and in only two was the diagnosis subsequently documented by roentgenologic examination. In the same year, Carangelo[4] reported four cases of massive gastrointestinal hemorrhage in pregnancy, with three deaths. Three of the four patients had an eclamptic syndrome with hypertension, edema, convulsions, oliguria and albuminuria, and all four had a septic course with fever. The onset of labor in three of the patients occurred between 24 and 48 hours after the onset of hematemesis. There were three stillbirths and one

TABLE 1.—*Reported Frequency Occurrence of Peptic Ulcer Disease in Pregnancy*

				Location			*Complications*	
Authors	*Material Studied*	*No. of Cases Studied*	*No. With Ulcer*	*Unclassified*	*Gastric*	*Duodenal*	*Hemorrhage*	*Perforation*
Hooker (1930-1932)	Pregnancies	350,000	1	....	1	....	....	1
MacNalty (1937)	Puerperal Deaths	770	1	....	1	....	1	....
Sandweiss et al. (1939)	Pregnancies	70,310	1	....	....	1	....	1
Johnston (1953)	Pregnancies	12,041	2	....	....	2	1	1
Durst et al. (1955)	Deliveries	149,491	6	5	....	1	1	....

premature but viable infant. In none of these cases, however, was the diagnosis confirmed.

In 1957 Vasicka[20] reported a case of massive gastrointestinal hemorrhage occurring in a pregnant woman who was subsequently shown to have peptic ulcer. Gastric resection with gastroduodenostomy was performed in the 20th week of gestation. This was the first reported case of surgical intervention for this complication during pregnancy. Hypertension, albuminuria and vaginal hemorrhage developed in the 28th week, and a viable infant was delivered by cesarean section during the 33rd week. Crisp,[6] in 1960, reported four cases of hemorrhage from peptic ulcer in pregnant women. The hemorrhage occurred in one case in the 37th week of pregnancy, in one in the 39th week, in one approximately two hours postpartum, and in the fourth on the second postpartum day. Ulcers, specified as duodenal in one case but not specified as to location in the others, were documented radiographically in each case. All four of the patients responded to medical management.

Lindell,[13] in 1962, reported a case of perforated ulcer occurring in a 26-year-old secundigravida in the ninth month of gestation. On surgical exploration a 1 cm perforation on the greater curvature side of the pylorus was repaired. The next day the patient was delivered of a 5½-pound infant. The mother and baby did well and left the hospital eight days postoperatively. Winchester[21] reported a case in which operation for perforation of a duodenal ulcer was done in the 34th week of gestation. The perforation was closed with omentum. The patient did well and was delivered of a viable infant in the 37th week.

The case here presented appears to be the fourth reported instance of successful operation for massive hemorrhage from duodenal ulcer occurring during pregnancy and the first case in which vagotomy and pyloroplasty for ulcer disease were concurrently performed.

Discussion

Pregnancy seems to have a beneficial effect on peptic ulcer,[5,18] but the reasons for this are not clear. Izak[10] and Gryboski[8] noted that plasma pepsinogen rises in the last trimester of pregnancy, reaching a maximum on the first postpartum day. Murray[15] examined the gastric secretion of pregnant women, both in the basal state and after the administration of 0.04 mg of histamine acid phosphate per kilogram of body weight. He noted decreased secretory response during pregnancy, mainly during the first 30 weeks.

Two possible explanations for diminished hydrochloric acid secretion have been offered. The first is the antisecretory effect of plasma histaminase, which increases in pregnancy, sometimes as much as a thousandfold.[1] Barnes[2] showed that plasma histaminase begins to rise about seven weeks after the last normal menstrual period and that the peak is reached in the 26th to 28th weeks of gestation. Preparations of histaminase administered to dogs with Heidenhain pouches were shown to diminish histamine-stimulated secretion.[17] Furthermore, the administration of histaminase inhibitors appear to augment both basal and gastric secretory response to a meal.[12]

The second explanation offered for diminished hydrochloric acid secretion in pregnancy is the influence of some hormonal factor.[20] It is known that the sex incidence of peptic ulcer is equal in both the prepubertal and postclimacteric years. However, during the child-bearing years (and the corresponding ages in man) peptic ulcer occurs dominantly in men in a ratio of four or five men to one woman. This has encouraged the belief that hormonal factors play a role in women during the child-bearing years. The effects of anterior-pituitary substances, estrogen, progesterone and lactogenic hormone have all been studied, but none of these have definitely been shown to diminish hydrochloric acid secretion.[20] Relaxin, a hormone present early in pregnancy with a tendency to diminish toward the latter part of pregnancy,[22] may be involved in some way, but little is known of its relationship to hydrochloric acid secretion and peptic ulcer disease.

The benefits derived from pregnancy in women with peptic ulcer disease are apparently short-lived. In Clark's[5] series, there was recurrence in almost every case within two years postpartum. Of additional note is the fact that in most of the cases that have been reported, complications have occurred either very late in the pregnancy or immediately postpartum.

Summary

A case of massive hemorrhage from duodenal ulcer occurring in the sixth month of pregnancy is presented. This unusual occurrence was treated successfully by vagotomy, pyloroplasty and ligation of the bleeding site. The literature dealing with the appearance of complications of peptic ulcer during pregnancy is reviewed and factors influencing gastric secretion during pregnancy are discussed. Symptoms of peptic ulcer disease are characteristically ameliorated by pregnancy. Complications are especially uncommon and when they do occur they tend to develop during the last trimester or immediately postpartum.

REFERENCES

1. Ahlmark, A.: Significance of blood histaminase in pregnancy, Lancet, 2:406-407, 23 September 1944.

2. Barnes, L. W.: Serum histaminase during pregnancy, Obstet. Gynec., 9:730-732, June 1957.

3. Bernstine, J. B., and Friedman, M. H. F.: Peptic ulcer in pregnancy, Amer. J. Obstet. Gynec., 56:973-976, November 1948.

4. Carangelo, J., and Efstation, T. D.: Massive gastric hemorrhage in pregnancy, Amer. J. Obstet. Gynec., 56: 191-194, July 1948.

5. Clark, D. H.: Peptic ulcer in women, Brit. Med. J., 1:1254-1257, 6 June 1953.

6. Crisp, W. E.: Pregnancy complicating peptic ulcer, Postgrad. Med., 27:445-447, April 1960.

7. Durst, J. B., and Klieger, J. A.: A report of a fatal hemorrhage due to peptic ulcer in pregnancy, Amer. J. Obstet. Gynec., 70:448-451, August 1955.

8. Gryboski, W. A., and Spiro, H. M.: The effect of pregnancy on gastric secretion, New Engl. J. Med., 255: 1131-1134, 13 December 1956.

9. Hooker, R.: Maternal mortality in New York City. A study of all puerperal deaths. Commonwealth Fund, N.Y. Acad. Med. Comm. on Public Health Relations, p. 234, 1930-1932.

10. Izak, G., Rachmilewitz, M., Stein, Y., Berkovici, B., Sadovsky, A., Aronovitch, Y., and Grossowicz, N.: Vitamin B_{12} and iron deficiencies in anemia of pregnancy and puerperium, Arch. Intern. Med., 99:346-355, March 1957.

11. Johnston, J. L.: Peptic ulcer and pregnancy. Deaths from perforation and hemorrhage of peptic ulcer during pregnancy, Obstet. Gynec., 2:290-296, September 1953.

12. Liepins, K. W., Ivy, A. C., and Suzuki, R.: Effect of certain histaminase inhibitors on gastric secretion, Amer. J. Physiol., 195:209-215, October 1958.

13. Lindell, A., and Tera, H.: Perforated gastroduodenal ulcer in late pregnancy, operated upon with survival of both mother and child, J. Obstet. Gynec. Brit. Comm., 69:493-495, June 1962.

14. MacNalty, A. S.: Ministry of Health Report in an Investigation into Maternal Mortality in Great Britain and Wales. Presented by the Minister of Health to Parliament, April 1937. Printed and Published by His Majesty's Stationery Office.

15. Murray, F. A., Erskine, J. P., and Fielding, J.: Gastric secretion in pregnancy, J. Obstet. Gynec. Brit. Emp., 64:373-381, June 1957.

16. O'Connell, W. T.: Peptic ulcer complicating pregnancy, Amer. J. Obstet. Gynec., 70:416-419, August 1955.

17. Rostorfer, H. H., and Laskowski, M.: Action of histaminase preparations in the heidenhain dog, Amer. J. Dig. Dis., 12:337-339, October 1945.

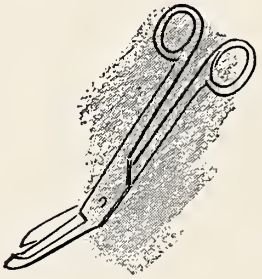
18. Sandweiss, D. J., Saltzstein, H. C., and Farbman, A. A.: The relation of sex hormones to peptic ulcer, Amer. J. Dig. Dis., 6:6-12, March 1939.

19. Sandweiss, D. J., Podolsky, H. M., Saltzstein, H. C., and Farbman, A. A.: Deaths from perforation and hemorrhage of gastroduodenal ulcer during pregnancy and puerperium, Amer. J. Obstet. Gynec., 45:131-136, January 1943.

20. Vasicka, A., Lin, T. J., and Bright, R. H.: Peptic ulcer and pregnancy. Review of hormonal relationships and a report of one case of massive gastrointestinal hemorrhage, Obstet. Gynec. Survey, 12:1-13, February 1957.

21. Winchester, D. P., and Bancroft, B. R.: Perforated peptic ulcer in pregnancy, Amer. J. Obstet. Gynec., 94: 280-281, 15 January 1966.

22. Zarrow, M. X., Holmstrom, E. G., and Salhanick, H. A.: The concentration of relaxin in the blood serum and other tissues of women during pregnancy, J. Clin. Endocr., 15:22-27, January 1955.



Disorders of Esophageal Motility

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California Medical Center, San Francisco. Taken from transcriptions, they are prepared by Drs. Martin J. Cline and Hibbard E. Williams, Assistant Professors of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine.

Dr. Smith*: The patient this morning will be presented by his private physician, Dr. Howard Shapiro.

Dr. Shapiro*[1]: Thank you, Dr. Smith. The patient is a 28-year-old Caucasian male who had had known difficulty in swallowing since childhood. This dysphagia was more pronounced with solids than with liquids, and he would frequently regurgitate undigested food eight to ten hours after a meal. In the several years before admission to hospital, his difficulties were complicated by substernal discomfort which generally occurred at night and would awaken him from sleep. This discomfort was relieved somewhat by antacids. He had no difficulty in maintaining his nutrition. A trial of antacid therapy and of viscous xylocaine failed to relieve the symptoms of dysphagia.

Results of physical examination were within normal limits. An upper gastrointestinal series revealed cardiospasm with moderate dilatation of the upper esophagus. Early vermiform movements and secondary peristaltic stripping motions were demonstrated at fluoroscopy. Esophageal motility studies showed a normal appearing gastroesophageal sphincter. There was no relaxation of the gastroesophageal sphincter to swallowing, and the swallowing waves were simultaneous throughout the esophagus. There was no evidence of peristalsis on the motility studies.

An esophagogastric myomectomy (Heller procedure) was done through a transverse abdominal incision. Since the time of operation the patient has remained quite well with the exception of some heartburn early in the recovery period. Follow-up esophageal motility studies continued to show a failure of peristalsis with simultaneous appearance of the swallowing waves throughout the length of the esophagus.

(The patient enters.)

Dr. Shapiro: Could you describe the major symptoms that you had before coming to my office?

Patient: I could not swallow food very well and I regurgitated two to eight hours after eating. It was rather painful at times.

Dr. Shapiro: How much weight have you gained since the operation?

Patient: I weigh about five pounds more than I did before the operation.

Dr. Shapiro: Have you had any symptoms since?

Patient: Not really.

Dr. Shapiro: Have you noticed any heartburn since the operation?

Patient: No.

Dr. Smith: Dr. Dietschy, would you like to bring out any points?

---

*Lloyd H. Smith, Jr., M.D., Professor & Chairman, Department of Medicine.

*[1] Howard A. Shapiro, M.D., Assistant Clinical Professor of Medicine.

Dr. Dietschy*[2]: I wonder if you would comment in more detail on the pain which you had at the very beginning of your troubles. Were you having chest discomfort at any time of the day or night?

Patient: No, the pain did not start until about two and a half years ago, and I had trouble swallowing for about five years before that.

Dr. Dietschy: You were a teenager when this began?

Patient: Yes.

Dr. Dietschy: Since then, you are sure that you have had no burning sensation in your chest?

Patient: It feels more like gas.

Dr. Dietschy: Is it a sense of fullness rather than a burning sensation?

Patient: Yes.

Dr. Dietschy: Does it tend to rise up in your chest?

Patient: Yes.

Dr. Dietschy: And is this relieved by taking antacids?

Patient: No, it is not.

Dr. Dietschy: Thank you. (Patient leaves.)

Dr. Smith: The x-ray films will be discussed by Dr. Zboralske.

Dr. Zboralske*[3]: A preoperative study demonstrated pronounced dilatation of the esophagus. The lower esophagus reveals a typical bird-beaking appearance. No peristalsis was identified during the time of fluoroscopy. Postoperative studies demonstrated much less dilatation of the esophagus but some persistent mild narrowing distally, although it does empty quite promptly. Again, no esophageal peristalsis was observed at fluoroscopy.

Dr. Smith: We are delighted to have as our major discussor this morning, Dr. John M. Dietschy from the University of Texas Southwestern Medical Center in Dallas. He is a member of the Department of Medicine there and assistant professor of medicine at the University of Texas Southwestern Medical School. Today he will discuss disorders of esophageal motility. Dr. Dietschy.

Disorders of Esophageal Motility

Dr. Dietschy: The patient presented by Dr. Shapiro manifests many of the findings commonly seen in the esophageal disorder known as achalasia. I would like to use the problem presented by this patient as the basis for a brief review of the normal physiology of the esophagus, and then to discuss two disorders of esophageal motility: Tertiary contractions and achalásia.

The esophagus is a muscular tube, made up of an outer longitudinal and an inner circular layer of muscle, which connects the lower end of the pharynx with the stomach. It is closed off both at its upper and at its lower ends by barrier mechanisms which prevent the inadvertent entrance of secretions and food from the mouth and stomach into the body of the esophagus. Anatomically the superior barrier mechanism consists primarily of a specialized bundle of the inferior pharyngeal sphincter, the cricopharyngeous muscle, which inserts on the lateral margins of the cricoid cartilage and passes strap-like around the posterior aspect of the esophagus, where it can act as an efficient sphincter.

The inferior barrier mechanism, on the other hand, is much more complicated and controversial. There is no specialized muscle band demonstrable at the lower end of the esophagus in the human, although such an anatomic structure can be found in certain lower animals. Nevertheless, there is a discrete area found at the lower end of the esophagus, the so-called high pressure zone, which behaves physiologically as a sphincter. Other mechanisms which are thought to contribute to the inferior barrier include the diaphragmatic sling formed around the esophagus as it passes through the diaphragmatic hiatus, the acute angle present at the esophagogastric junction, rosette formation by gastric mucosa pushing up into the base of the esophagus, and finally, the intra-abdominal portion of the esophagus which is subject to intra-abdominal pressure. While all of these may contribute together to the inferior barrier mechanism, of particular importance to the discussion today is the high pressure zone or inferior esophageal sphincter.

The Sequence of Events in Swallowing

The sequence of events which occur in the esophagus during the normal act of swallowing may be followed by placing pressure-sensing devices at various levels of the pharynx, esophagus and stomach. In the resting esophagus two areas of elevated intraluminal pressure can be demonstrated; these are located at the levels of the upper and lower esophageal sphincters. Within the body of the esophagus one finds a slightly negative pressure with variations in the baseline caused by the respiratory cycles. On the abdominal side of the diaphragmatic hiatus, the respiratory variations are reversed, and the pressure in this area is usually close to atmospheric pressure.

---

*[2] John M. Dietschy, M.D., Assistant Professor of Medicine, University of Texas Southwestern Medical School.

*[3] F. Frank Zboralske, M.D., Assistant Professor of Radiology.

Swallowing begins as the tongue is pushed up against the hard palate and swept backwards as the nasal pharynx is closed and the pharyngeal sphincters contract. This results in the development of an abrupt rise in pressure in the hypopharynx. Just preceding these events in the hypopharynx, however, the superior esophageal sphincter relaxes, allowing the liquid or solid bolus to be pushed into the upper portion of the esophagus by the pharyngeal pressure wave. Here there is a momentary delay following which a primary peristaltic wave is initiated in the upper part of the esophagus. This peristaltic wave is then propagated down the body of the esophagus at a rate of 2 to 3 cm per second. Just before the arrival of the primary peristaltic wave at the lower esophageal sphincter the resting pressure in this area relaxes so that the bolus may pass through the esophagogastric junction into the stomach.

This sequence may be observed easily at fluoroscopy. As the patient begins to swallow and high pressure develops in the hypopharynx, a liquid bolus of barium suspension will be forced downward and almost instantaneously fill the entire body of the esophagus. In contrast, if the bolus is solid food, then the pharyngeal pressure wave merely pushes it into the top of the esophagus where it remains momentarily until primary peristalsis is initiated. In either case one then observes a contraction wave which begins at the upper end of the esophagus and moves downward pushing the bolus ahead of it. Just before the arrival of this wave at the lower end of the esophagus the lower sphincteric region opens and the bolus is conveyed into the cardiac portion of the stomach.

Primary peristalsis, then, is that integrated series of events seen in the esophagus which is initiated by the act of swallowing and whose function is to convey liquids and solids from the mouth into the stomach.

Secondary Esophageal Peristalsis

A second type of normal motility is known as secondary peristalsis. In this situation, peristalsis is not initiated by the act of swallowing, but by distension of some part of the body of the esophagus. If the esophagus is distended at a particular level, then at this point a peristaltic wave is initiated; the wave sweeps down the esophagus and the lower esophageal sphincter relaxes as it does with a primary peristaltic wave. There is no retrograde conduction of peristalsis upward from the point of distension and no relaxation of the superior sphincter occurs.

One can observe this sequence occurring in secondary peristalsis under the fluoroscope. For example, in a patient with esophageal regurgitation, one may observe reflux of barium meal from the stomach back into the body of the esophagus. The barium seldom moves upward very far, however, before the esophagus responds with a secondary peristaltic wave which sweeps downward, clearing the esophageal lumen of the regurgitated barium.

Secondary peristalsis, then, is that integrated series of events seen in the esophagus which is initiated by distension of the body of the esophagus and whose function is to clear the esophageal lumen of material refluxed from the stomach or left behind in the body of the esophagus by the primary peristaltic wave.

Abnormal Motility

I would like now to discuss two forms of abnormal esophageal motility which give rise to distinct clinical syndromes. The first of these is the syndrome of tertiary contractions in which the motility disorder involves primarily the lower half of the body of the esophagus but spares the lower esophageal sphincter; the second is achalasia (cardiospasm) in which abnormal function of the lower half of the esophagus as well as the inferior esophageal sphincter can be demonstrated.

In tertiary contractions normal pharyngeal and superior sphincteric mechanisms are present, but instead of a primary peristaltic wave sweeping down the esophagus in an orderly, propagated manner, there is disordered, irregular contraction of the entire lower one-half or two-thirds of the body of the esophagus with the development of very high intraluminal pressures. Furthermore, in response to a single swallow one may observe repetitive contractions of this area of the esophagus. Yet, despite this abnormality of peristaltic activity in the body of the esophagus, the lower esophageal sphincter does relax; this is an extremely important point in differentiating this syndrome of tertiary contractions from the syndrome of achalasia.

Tertiary contractions appear quite dramatic when viewed at fluoroscopy. They may be mild, appearing only as indentations or slight rippling motions in the body of the esophagus. Not infrequently they are more severe with irregular contractions of the entire length of the esophagus, or in some patients one may occasionally observe pronounced segmental contractions with pseudodiverticulum formation. This lesion has made a tremendous impression on the imagination of roentgenologists, who have applied many colorful and confusing terms to this lesion. Schatzki was the first to refer to this motility as "Kräuselung"—that is, rippling or curling of the esophagus. Subsequently, this motility disorder as seen roentgenographically has been referred to as the rosary-bead deformity, the string-of-pearls deformity, the corkscrew esophagus, spastic pseudodiverticulosis or segmental diverticulosis, and, finally, as the Barony-Teschendorf syndrome—a syndrome named for two German roentgenologists who first described this disorder. There is neither experimental nor clinical evidence to separate any of these particular forms of the motility disorder. I believe, therefore, that these should all simply be referred to as "tertiary contraction" of the esophagus.

Characteristics of Tertiary Contractions

This disorder of esophageal motility is commonly seen during examination of the upper gastrointestinal tract, particularly in patients over the age of 50. Attacks of tertiary contractions are commonly transient, and between episodes patients are usually found to have entirely normal primary and secondary peristaltic activity. Fortunately, the vast majority of patients found to have tertiary contractions have no symptoms related to this motility disorder. Very rarely, however, dysphagia and substernal pain associated with tertiary contractions of the esophagus develops. It is this clinical syndrome which is known as "symptomatic tertiary contractions" or "diffuse spasm" of the esophagus. In general there is no correlation between the apparent severity of the tertiary contractions as seen fluoroscopically and the severity of the pain-dysphagia syndrome associated with these attacks.

The pain of symptomatic tertiary contractions is fairly characteristic. It is typically substernal in location and in severe cases may radiate into the neck or shoulders. It varies in quality from a rather dull, uncomfortable sensation to a severe, constricting pain beneath the breast bone. It can mimic the pain of myocardial infarction in every way and, furthermore, may be relieved by nitroglycerin and amyl nitrate. Attacks of symptomatic tertiary contractions are usually initiated by those events which normally activate primary or secondary peristaltic activity in the body of the esophagus. Thus, the act of swallowing may precipitate an attack of pain and dysphagia. Similarly, the recumbent position, which enhances gastro-esophageal reflux, may initiate episodes of substernal pain in the middle of the night. Such attacks are quite irregular in frequency, duration and intensity. Since the patient may manifest entirely normal esophageal motility between attacks, the diagnosis depends upon the ability to reproduce the syndrome under fluoroscopic control when the pain and dysphagia can be shown to occur coincident with tertiary contractions of the body of the esophagus.

Since the inferior sphincter relaxes normally in this syndrome, there is usually little or no dilatation of the body of the esophagus. In addition, the esophagus in this condition does not manifest hypersensitivity to mecholyl—that is, the mecholyl test result is negative.

Achalasia

In the syndrome of achalasia, there is also disordered motility of the lower one-half to two-thirds of the body of the esophagus; but, equally important, there is also dysfunction of the lower esophageal sphincter in that this area of musculature fails to relax in response to motor activity in the body of the esophagus. Consequently, there is progressive dilatation and elongation of the esophagus in this disease. Early in the syndrome one commonly sees irregular tertiary contractions of the body of the esophagus, but as dilatation progresses and the musculature becomes atonic, these irregular contraction waves tend to disappear. The motility disorder in achalasia, then, is twofold: (1) There is loss of normal primary and secondary peristaltic activity in the lower portion of the body of the esophagus; and (2) the inferior esophageal sphincter fails to relax properly during the act of swallowing, and this functional obstruction, in turn, leads to progressive dilatation.

Patients with achalasia may manifest both substernal pain and dysphagia, but these symptoms usually begin at an earlier age than those associated with tertiary contractions. The initial symptoms of dysphagia may be irregular in intensity and occur at only irregular intervals. Eventually, however, difficulty in swallowing becomes progressively worse leading to weight loss. There may also be a sensation of substernal fullness and episodes of choking, particularly at night, when esophageal contents spill into the bronchopulmonary tree.

The diagnosis of this syndrome rests on the fluoroscopic demonstration of lack of primary and secondary peristaltic activity in the lower half of the esophagus and failure of the lower esophageal sphincter to relax properly. In addition, the body of the esophagus in achalasia responds to small doses of mecholyl with an exaggerated tetanic contraction (positive reaction to a mecholyl test).

Thus, these two disorders of esophageal motility may be summarized as follows:

Symptomatic tertiary contractions are primarily seen in elderly patients, whereas in achalasia symptoms classically begin in early middle age.

In achalasia there is motor dysfunction both of the body of the esophagus and of the inferior esophageal sphincter. In tertiary contractions, in contrast, the motility disorder appears to be limited to the body of the esophagus.

A positive reaction to a mecholyl test is seen in patients with achalasia but usually not in those with symptomatic tertiary contractions.

Moderate to pronounced dilatation of the esophagus is common in achalasia but is usually not seen in symptomatic tertiary contractions.

California
Medicine

Editor
DWIGHT L. WILBUR, M.D.

Assistant to the Editor
ROBERT F. EDWARDS
For information on preparation of manuscript, see advertising page 2

Policy Committee—Editorial Board
JOHN G. MORRISON, M.D., San Leandro
MALCOLM C. TODD, M.D., Long Beach
WILLIAM F. QUINN, M.D., Los Angeles
JOSEPH F. BOYLE, M.D., Los Angeles
HAROLD KAY, M.D., Oakland
HELEN B. WEYRAUCH, M.D.
San Francisco
DWIGHT L. WILBUR, M.D., San Francisco

EDITORIAL

A Successful Annual Meeting

THE EDUCATIONAL AND the business functions of the 96th Annual Session of the California Medical Association were carried forward in a thoroughly professional manner at the meeting held 15 to 19 April in Los Angeles.

Outstanding guest speakers, whose presentations were centered on Advances in Diagnostic Technique, the theme of this year's scientific sessions, drew good audiences even though the total physician registration at the meeting was less than in previous years. Attendance may have been affected somewhat by the weather (which in Southern California may be described as Oregonian) and by the fact that the CMA gathering followed closely upon other meetings of similar quality that were held in California.

The House of Delegates carried on its business with extraordinary harmony and dispatch, dealing with 108 resolutions and electing officers and members of the Council. Among important actions, the House:

- Set dues at $90, unchanged from the present rate.
- Reaffirmed the previously expressed opinion of the majority of the House of Delegates favoring changes in laws governing therapeutic abortion when it defeated a resolution that would have had the CMA take no official stand on recently proposed legislation on this subject in California.
- Adopted a resolution encouraging communication between the CMA and the California Council for Health Plan Alternatives.
- Commended California Blue Shield for its responsiveness to physician problems and its accomplishments in implementing Medi-Cal.
- Endorsed efforts to repeal certification and recertification provisions of P.L. 89-97.
- Encouraged efforts to eliminate Medicare's requirement for three days in hospital before admission to an extended care facility.
- Reaffirmed the stand that chiropractic services should not be covered under Medi-Cal or other government programs.
- Adopted a resolution asking the CMA to review P.L. 89-97 regulations concerning clinical laboratories operated by qualified physicians; and affirming that clinical laboratory services provided by a physician, even though he provides no other services to the patient, are medical services quite as much as are similar services rendered by physicians providing other services, and should be regulated in the same manner as all other medical services.
- Directed the Commission on Medical Services to develop and promote the use of a standard reporting form for private and government third-party payment programs.
- Recommended that the profession take the same interest and initiative in staff activities of nursing homes and extended care facilities as it has found desirable and necessary in acute hospitals.
- Called for CMA to take all possible steps to eliminate the drug formulary under the Medi-Cal program.
- Urged continuing emphasis and educational efforts on the need for physicians to include necessary identifying information on direct bills to Medicare patients.
- Favored supporting appropriate legislation requiring pasteurization of all market milk sold for human consumption.

- Emphasized the need for the medical profession to be concerned with the problem of solid waste disposal.
- Approved the intent of resolutions urging caution in the new field of weight reduction and referred the matter to the Council for study.
- Recommended that any component medical society that is near a medical school to have a committee to maintain liaison with the school.
- Urged active support be given by CMA to legislation requiring motorcycle operators to wear properly designed protective headgear.
- Commended medical schools that implement or explore ways to attract and train general practitioners.
- Voted to continue and expand educational programs for both the profession and the public with regard to marijuana, LSD and other hallucinatory drugs.

Notable in proceedings of the House was that this year the CPS Reference Committee considered far fewer resolutions than it has had before it in past years.

Dr. Malcolm C. Todd was elected President-Elect by unanimous vote, and Dr. John G. Morrison was installed as President, succeeding Dr. James C. MacLaggan. Dr. William F. Quinn was re-elected Speaker of the House and Dr. Joseph F. Boyle was elected Vice-Speaker, taking the place of Dr. Joseph W. Telford, who did not stand for re-election.

Newly elected Councilors include: Doctors Stanley A. Moore, First District, succeeding Roger C. Isenhour who was not a candidate for reelection; Homer C. Pheasant and Marvin J. Shapiro, Third District; and E. Kash Rose, Ninth District.

At its organization meeting the Council elected Dr. Albert G. Miller chairman and Dr. Harold Kay vice chairman. Dr. Helen B. Weyrauch was reappointed Secretary of the CMA and Dwight L. Wilbur editor.

All in all, the Annual Session was a successful one, and as it was coming to a close preparations for next year's meeting were already under way. San Francisco is the place. 23 to 27 March is the time.

The Chairman Retires

NEVER BETTER HAVE members of the California Medical Association House of Delegates represented their constituency than when they came to their feet and resoundingly applauded Dr. Carl E. Anderson in recognition of the great good he did this Association in the five years he served as chairman of the Council. He announced his retirement from that position at the time of the CMA Annual Session and he was honored in the House of Delegates with the presentation of tokens of appreciation by the Association and by his own Ninth Councilor District.

Dr. Anderson's term as chairman coincided with a time of critical negotiations with legislative bodies and governmental agencies, both state and national, and to these tasks he committed much of his considerable energies and intellect. Whether presiding over meetings of the Council or representing the CMA or the American Medical Association in matters of principle, of socio-economic detail or interrelationship with other organizations, Dr. Anderson was always impressively well prepared, level-headed, realistic and gentlemanly. It was these qualities—and the successes they brought to the organizations he represented—that members of the House of Delegates were applauding when he appeared before them at the Annual Session.

We join in that applause.

California Medical Association

NOTICES AND REPORTS

MALCOLM C. TODD, M.D.
The New President-Elect

The following is the speech made in the House of Delegates by Dr. Joseph F. Boyle nominating the new President-Elect of the California Medical Association.

IN THE NOMINATION OF a candidate for President-Elect of the California Medical Association, we take cognizance of those qualities which identify true medical leadership. Among those qualities, we recognize that our nominee must be of proved sound judgment, must have experience and recognized stature in his local medical society and in our state and national medical associations as well, must have the respect of his own colleagues for his professional competence and character. He must be articulate, and in these days he must also possess considerable political sophistication and awareness. Finally, of greatest importance to all of us, he must be firmly dedicated to the practice of medicine.

In Los Angeles county we are most fortunate that we have many outstanding physicians to choose among. We are also fortunate that we have one man most outstanding of all. He is a practicing surgeon in Long Beach, a graduate of Northwestern University Medical School in 1937, who after surgical residency at Cook County Hospital in Chicago and tour of duty in the United States Army, became a member of the Los Angeles County Medical Association and the California Medical Association in 1947. The list of our nom-

MALCOLM C. TODD, M.D.

JOHN G. MORRISON, M.D. . . . . President
MALCOLM C. TODD, M.D. . . . . President-Elect
WILLIAM F. QUINN, M.D. . . . . Speaker
JOSEPH F. BOYLE, M.D. . . . . Vice-Speaker
ALBERT G. MILLER, M.D. . . . . Chairman of the Council
HAROLD KAY, M.D. . . . . Vice-Chairman of the Council
HELEN B. WEYRAUCH, M.D. . . . . Secretary
DWIGHT L. WILBUR, M.D. . . . . Editor
HOWARD HASSARD . . . . Executive Director

General Office, 693 Sutter St., San Francisco 94102 • 415 776-9400
JACK CURLEY . . . . Southern California Office
1515 N. Vermont Ave., Los Angeles 90027 • 213 663-8071
RICHARD W. LEMOS . . . . Sacramento Office
1127 11th St., Sacramento 95814 • 916 444-7496

inee's many accomplishments is long. I will detail but a few of them. He became president of his medical society in Long Beach in 1953 and has been a member of the CMA House of Delegates since 1949. He has been a member of the Los Angeles County Medical Association Council for seven years, a member of the CMA Council for three terms and a delegate to the American Medical Association for eight years. He is a diplomate of the American Board of Surgery and a fellow of the American College of Surgeons, a past chief of staff of the Long Beach Memorial Hospital and he is at present or has been a member of the boards of 12 different civic and public volunteer agencies, including the Long Beach Chamber of Commerce. He has been the chairman of the CMA Commission on Communications from 1964 to the present and has been chairman of CALPAC and a member of the national board of AMPAC since the inception of these organizations in 1961. He is well known to all of you for his service to the profession of medicine in California and in this House of Delegates. But he is best known by all of us in Los Angeles County.

It is, therefore, my privilege as president of the Los Angeles County Medical Association to present in nomination for President-Elect of the California Medical Association, the unanimous choice of Los Angeles County, Doctor Malcolm C. Todd.

JOSEPH F. BOYLE, M.D.

Council Meeting Minutes

Tentative Draft: Minutes of the 529th Meeting of the Council, San Francisco, Hilton Inn, 25 February 1967.

The meeting was called to order by Chairman Anderson at the Hilton Inn, on Saturday 25 February 1967, at 9:45 a.m.

A quorum was present and acting (full roll call, including names of invited guests appears at the end of these minutes).

1. *Minutes for Approval*

The minutes of the 528th meeting of the Council, held 15 January 1967, were approved as distributed.

2. *Commission on Medical Services*

Chairman Howard reported on a joint meeting of the Commission, the Committee on Insurance and Prepayment and the California Committee of the Health Insurance Council. The Commission decided to request a similar meeting at least every six months to try to find a practical way to provide group medical insurance for individual, self-employed persons. Chairman Howard also reported that the AMA Committee on Insurance and Prepayment is taking steps to implement the CMA resolution that called for setting up minimum standards for prepaid health programs and insurance.

3. *Relative Value Studies*

Doctor Howard reported that the revised edition of the CMA *Relative Value Studies* will be utilizing a five-digit system and it was hoped that the 1967 revision of the *RVS* would be ready for submission to the Council for approval in the fall.

4. *Military Dependents' Medical Care Program*

Doctor Howard informed the Council that the Committee recently had met with General Peatfield of the Department of Defense to discuss the program. The contract is on a "usual and customary" fee basis at present, but has a ceiling. The goal is "usual and customary" with no ceilings.

5. *Endorsement of Vernon L. Nickel*

A resolution endorsing Vernon L. Nickel, M.D., a Los Angeles orthopedic surgeon, for the 1966 "Physician of the Year" award was introduced to the Council. The award is sponsored by the President's Committee on Employment of the Handicapped. The Council added its endorsement to that of the Los Angeles County Medical Association.

6. *Report of the President*

President MacLaggan reported to the Council on a meeting called by the Department of Health, Education and Welfare on P.L. 89-239 (Heart Disease, Cancer and Stroke legislation). He reported that the idea of "treatment centers" still received emphasis during the discussions on implementation of the law.

7. *Health Manpower Council*

President MacLaggan reported on the 4 and 5 February meeting of the Health Manpower Council held in San Diego. More than 80 different health organizations were represented, and a final decision to go ahead with the Health Manpower Council was made. Doctor MacLaggan updated the Council on the composition of the Health Manpower Council, which has changed some since its introduction. It is now to be a 25-man Council composed of three representatives each from government, CMA and CHA, one from the California Nurses' Association, one dental and one nursing home representative. These persons would be designated from their own organizations. The remaining 13 members of the Council will be elected from the general group, chosen by the steering committee. Doctor MacLaggan said that frequent meetings would be held and activity would be getting under way very shortly.

8. *Physician Reimbursement in Teaching And County Hospitals*

Doctor MacLaggan reported on a meeting with representatives of the California Hospital Association, CMA, and deans of medical schools in California. The meeting was held to discuss the proposed regulations establishing criteria for determination of reimbursement for services of hospital interns, residents and supervising physicians in teaching and county hospitals. The unanimous feeling of the group was that the guidelines recently promulgated under Title 18 were desirable and it was the wish that they also be established as criteria for reimbursement under Title 19. The proposed regulations stated, in part, that attending physicians in a teaching setting may submit bills on a reasonable charge basis, with the reasonableness of the charge to be determined by the carrier in the same manner and under the same rules as for all other physicians.

Doctor MacLaggan also informed the Council that it was agreed that there should be a closer liaison with the deans of medical schools and that some representatives of teaching hospitals and the medical schools should be appointed to the county review committee in any county in which there was such a teaching institution. Discussion followed on the interpretation of the regulations.

ACTION: Voted to approve the concept of "Reasonable" charges under both Titles 18 and 19 for attending physicians in county and teaching hospitals, and to approve the request that these institutions cooperate with and make available personnel to help county society review committees in reasonable charge determination.

9. *Conference on Infectious Diseases Of Pets and Patients*

It was brought to the attention of the Council that the University of California Medical Center in San Francisco is sponsoring a conference on "Infectious Diseases of Pets and Patients," in cooperation with the California Veterinary Medical Association. A request was made by the UCSF Medical Center for CMA support in recognition of the unique opportunity for these two professions to meet jointly.

ACTION: Voted to support and encourage the two organizations sponsoring the conference and send a letter to this effect.

10. *California Medical Education And Research Foundation*

Doctor MacLaggan announced to the Council that the CMERF Board had met and approved the appointment of Paul Ward as the director of the California Committee on Regional Medical Programs. The Council was also informed that Audio-Digest had donated $25,000 to CMERF again this year. This money was earmarked for education and will be used for some type of undergraduate scholarship for premedical students.

11. *Annual Session House of Delegates Meeting*

Speaker Quinn announced that those Bylaw changes and resolutions not considered at last year's House of Delegates meeting due to a lack of a quorum, need to be reintroduced this year. This does not apply, however, to Constitutional amendments. Again this year Councilors will be asked to be responsible for a reference committee of their choice during Annual Session and will be responsible for presenting to the Council, at the first meeting after Annual Session, recommendations for Council implementation of resolutions adopted by the House.

Dr. Quinn also reminded the Council that Delegates' dues must be paid by 1 April for them to be seated at the House of Delegates.

12. *1967 Conference of Component Society Officers*

Doctor Anderson called to the attention of the Council an informational report reviewing the 1967 Conference of Component Society Officers. The report gave in-depth consideration to planning, format, physical arrangements, and recommendations for next year, as well as an evaluation of each part of the meeting. Also included was a series of guides for discussion leaders. Doctor Anderson expressed the appreciation of the Council for all the efforts of those committee members who made the meeting possible.

13. *1967-68 Council Meeting Schedule*

Doctor Anderson presented to the Council for its approval the proposed schedule of Council meetings for the coming year. Discussion followed on an appropriate time to hold the Conference of Component Society Officers.

ACTION: Voted to approve the tentative schedule as submitted with the exception of the date for the Conference of Component Society Officers, which will receive further study by staff before a recommendation is presented to the Council.

14. *Committee on Legislation*

Chairman Kilroy reported on various bills pending before the State Legislature. A number of these were considered by the Council.

Doctor Kilroy introduced to the Council a new staff member of the Public Health League, Mr. Hugh McWilliams. Mr. McWilliams is a field secretary working out of the San Francisco office on membership, public relations and general contracts.

15. *Poverty Program Clinics*

Doctor Albert G. Clark, president of the San Francisco Medical Society, gave a progress report on establishing a Poverty Program Clinic in the San Francisco area. The purpose of this type of clinic is to treat persons in "target" areas, such as Hunters Point, Tenderloin, etc., whether or not they were covered by some type of insurance or welfare. He said that the society believes that as the residents of the area where the neighborhood health center is located move into the mainstream of medical care, the facility should be developed into a neighborhood recreation center or any other needed neighborhood facility.

16. *Reports of Medical School Deans*

Dean Egeberg of the University of Southern California School of Medicine briefly commented on his experiences with the anti-poverty programs and neighborhood health centers. He also expressed his appreciation for the action taken by the Council in regard to payment of physicians in county and teaching hospitals.

Dean Glaser of the Stanford University School of Medicine introduced to the Council Doctor Thomas Gonda, professor of psychiatry and recently appointed Associate Dean. Doctor Glaser reported that the curriculum study is ongoing and more thought is being given to flexibility. He said it is the feeling of many that an opportunity for a student to elect a broader range of courses provides a better basis for later years.

Dean Cullen of the University of California San Francisco Medical School also expressed his appreciation for the Council action on reimbursement of physicians in county and teaching hospitals. He relayed his appreciation to the San Francisco Medical Society for its financial support of student activities, the latest of which was donating funds to enable two medical students to attend a student conference in the East.

Dean Mellinkoff from the University of California Medical School at Los Angeles also thanked the Council for its action. He reported that the caliber of students entering medical schools seems to be rising constantly and it becomes difficult to select those who appear to be most capable.

17. *Bureau of Health Insurance, Social Security Administration*

Mrs. Mercia Kahn, Director of the Western Region of the Bureau of Health Insurance, reported to the Council that the 600 mark was reached by California in certifying extended care facilities under Medicare. She stated that the next highest number of facilities certified was 189 in New York.

18. *Woman's Auxiliary to the CMA*

Mrs. Samuel K. Bacon, president of the Auxiliary, reviewed some of the work that has been accomplished by the Auxiliary within the past year, and expressed her appreciation for being invited to attend the Council meetings during her term of office.

19. *California Nurses' Association*

Mrs. Mary K. Stanley, president of the Association, reported on the Health Manpower Council meeting in San Diego and expressed CNA's pleasure at having a permanent seat on the Council. She stated that the CNA had very high hopes for such a Council handling the manpower problems of California on a voluntary basis. Mrs. Stanley also expressed her appreciation for the invitations extended to her to attend the CMA Council meetings. She said that this would be her last meeting with the Council, as president, and thanked the Council for its courtesy to her during her term of office.

20. *California Hospital Association*

Mr. Gordon Cumming, president-elect of the CHA, reported to the Council that CHA had been exploring various possibilities of setting up a data system on a service basis into which hospitals could feed information and could serve as a repository of data for hospitals and allied organizations.

Mr. Cumming also commented on the ongoing study being conducted by CHA regarding the impact of the Medicare reimbursement formula for the operation of California's hospitals. He indicated that the information from California appears to show that all hospitals have real problems with the Medicare formula. Problems are modified by the extent that hospitals are able to call upon other sources such as tax funds or other supplementation.

Mr. Cumming and Doctor Morrison spoke to the Council regarding a joint venture of the CHA-CMA in uniform data collection. Doctor Morrison said he felt that in the interest of hospitals as well as the broad interest of planning, we should join with CHA in this effort. It was described as a separate, nonprofit corporation. Mr. Cumming said that the concept they envisioned was that the expense would be sustained by hospital payments for the service. He said that to start such a project would probably cost about $35,000 and that the CHA had this in the current year's budget, committed for this purpose. He stated that the corporation would be jointly managed and would render service to medicine, hospitals and other concerned groups such as the Committee on Regional Medical Programs, the Department of Public Health and others. He said this would be a voluntary effort.

ACTION: Voted to approve CMA's participation in a Joint Data Collection Project with CHA, with the understanding that no sizable expenditure of money will be required by CMA.

21. *State Department of Public Health*

Doctor Breslow, director of the Department, announced that Governor Reagan, in accordance with the Comprehensive Health Planning and Public Health Service Amendments of 1966 has designated the SDPH as the agency to undertake the comprehensive health planning. The program calls for the appointment of a council which would have policy making and guiding influence on the program. Doctor Breslow said that he hoped that Mr. Spencer Williams would be willing to participate personally on the council, and that it would be able to coordinate and enhance the extensive health planning already underway throughout the state.

22. *State Health and Welfare Agency*

Mr. Carel Mulder, director of the Office of Health Care Services, Health and Welfare Agency, outlined three immediate problems facing the Medi-Cal program. The first problem, he stated, lies in assuring that reasonable charges of physicians are paid with reasonable promptness. He added that this problem is being worked out in cooperation with California Blue Shield (CPS). The second problem area was described as assuring that the program as enacted last year is and can remain fiscally sound. The third area of concern is in assuring that the program develops a high quality, properly utilized system of services which remains in the mainstream of medicine. Mr. Mulder said that this was inherent in the law and the Administration had a commitment to see that this was done.

23. *State Department of Mental Hygiene*

Doctor Robert Hewitt, associate director of the Department, distributed to the Council for its information an annual report just released. Doctor Hewitt also said a report has just been completed on a study of staffing State mental hospitals, which is available from the Department.

24. *University of California—*
California College of Medicine

Councilor Grunigen presented the following resolution to the Council for its consideration:

"*Whereas* the California Medical Association has demonstrated its great interest in and goodwill for the California College of Medicine by generous monetary, moral and professional advisory support, all being consistent with its policy of uniting two professions under a single standard of medical care and increasing the number of young physicians graduating in California; and

"*Whereas* the survival of the California College of Medicine is seriously threatened due to physical and administrative deficiencies which were noted during the recent accreditation inspection and which indicate that the present geographic location is not the best for the development of a first-class medical school; and

"*Whereas* the Trustees have not met to take the necessary actions required to solve this crisis although the official accreditation committee demanded that remedial decisions be made no later than the middle of June 1967; now, therefore, be it

"RESOLVED: That the California Medical Association ask for an opportunity for its officers to present, at an early official meeting of the Trustees, a request that the Trustees act to urge the Regents quickly to assume the final authority and make the necessary decisions required to assure accreditation; and be it further

"RESOLVED: That a copy of this resolution be sent to all Regents and Trustees."

ACTION: Voted to approve the resolution.

25. *Commission on Communications*

Chairman Todd reviewed for the information of the Council, the activities of the Commission during the past several months, commenting on a detailed written report submitted to each Councilor. Doctor Todd pointed out that much staff time has been spent to upgrade and expand CMA News Services, both within the medical profession and to the public through mass media. Another Communications function which has greatly expanded is Field Services, which is making a concentrated effort to enhance face-to-face communication with county societies, individual physicians, allied health organizations and others throughout the state. Doctor Todd also reported that the Publication Services Section continues to respond to requests from other CMA commissions and committees for assistance in the preparation and production of various kinds of printed materials. In addition, this section has given much time and effort to special projects, such as helping to improve the format and materials for meetings of the CMA Council.

26. *Report of the President-Elect*

President-Elect Morrison described briefly the Congress on Socio-Economics which he attended in Chicago. He said that the meeting gave in-depth study to problems of health manpower and health facilities planning.

27. *Committee on Committees*

Chairman Morrison presented to the Council a list of recommendations for appointment to 1967 CMA commissions and committees. Several amendments and changes in the list were suggested.

ACTION: Voted to approve the list of recommendations, as amended, for submission to the 1967 House of Delegates.

The Committee on Committees also recommended:

a. That the Committee on Fees be enlarged by two members.

ACTION: Voted to approve enlarging the Committee on Fees.

b. That the objectives and functions of the Committee on Voluntary Health Agencies be reviewed by the Scientific Board.

ACTION: Voted to refer this request to the Scientific Board.

c. That the following committees be disbanded:

Committee on Scientific Information
Subcommittee on Tumor Tissue Registry
Subcommittee on Consultative Tumor Boards
Subcommittee on Cancer Education

ACTION: Voted to approve disbanding of above committees and preparation of Bylaw changes to accomplish this.

28. *California Blue Shield*

Doctor Richard Wilbur, chairman of the Blue Shield Board, reported that standard business activity had been on the increase and that during the first six weeks of this year, Blue Shield had gained about 10,000 persons in membership under standard programs and CPIC. He also stated that as much of the outpatient coverage as possible was being converted from visit deductible to cash dollar deductible, in response to a directive of the Blue Shield Board one year ago. Doctor Wilbur reported that more than 100,000 contracts had been sold or converted to a cash deductible outpatient medical benefit in the past year, which represented more than 70 per cent of the subscribers holding this type of coverage. He also said that Blue Shield was attempting to convert as many contracts as possible under the standard programs to the usual and customary fee concept. The statewide promotion and advertising campaign for the new Blue Shield "Major Medical" coverage was being deferred until 1 July, he reported.

Doctor Wilbur made a presentation to the Council summarizing the Medicare and Medi-Cal activity. From 1 July 1966 through 24 February 1967 a total of 915,000 Medicare claims had been processed and more than $24 million had been paid on behalf of Medicare beneficiaries. Under the Medi-Cal program from 1 March 1966 to 24 February 1967 a total of 11.4 million claims, amounting to $137,586,364 were paid.

Projected figures under the Medi-Cal program indicate that 19 per cent of payments will go to physicians, 13 per cent to other medical, 6 per cent to dentists, and 7 per cent for drugs. The remaining 55 per cent is paid by Blue Cross—30 per cent to nursing homes and 25 per cent to hospitals. Doctor Wilbur spoke about the continuing problem of eligibility determination under the Medi-Cal program, and Blue Shield efforts to aid the state in solving the problem. He said that as a temporary measure, Blue Shield was suggesting that physicians attach a photostat of the recipient's identification card to the billing form to establish eligibility.

Doctor Wilbur reported that on 16 February 1967 the California State Employees Association, plus two individuals named as taxpayers, filed a complaint in the Superior Court of Sacramento County seeking an injunction against further payments to Blue Cross and Blue Shield Plans for administration of the Medi-Cal program. The complaint was that the contract with the Blues violated Article 24 of the California Constitution, in that contract work which could be done by persons under the State Civil Service should not be contracted out to private enterprise. Defendants are Mr. Spencer Williams, Director of the State Health and Welfare Agency, and Mr. Houston Flournoy, State Controller, Blue Shield and Blue Cross.

Doctor Wilbur emphasized the increased utilization study activity by Blue Shield and introduced to the Council Mr. Harry Parke, who recently joined California Blue Shield as director of the utilization audit function.

29. *Medical Advisory Committee to the Division of Industrial Accidents*

Doctor Carl Anderson called to the attention of the Council a report of his 3 February 1967 meeting with members of the Medical Advisory Committee to the Division of Industrial Accidents. The report stated that due to the increased workload of the Division and the decrease in budget, the number of hearing teams within the Division had to be cut and no new activities could be undertaken at the present time. However, it was stressed that eventually there will be an opportunity to expand the programs and that some constructive thinking should be done along this line. It was recommended that the Bureau of Research and Planning might conduct a survey among physicians doing Workmen's Compensation work regarding the problems they encounter, the results of which could be helpful to the Medical Advisory Committee for future planning. It was also recommended that the Medical Advisory Committee might investigate some of the problems on its own in order to develop recommendations for the proper functioning of a rehabilitation unit in the Division. The suggestion also was made that the Medical Advisory Committee report to the Director and through him, to the Governor, and the Legislature, regarding steps that need to be taken to make the Medical Bureau of the Division more effective.

Doctor Anderson also commented on the availability of a new publication, *Register of Physicians and Surgeons (M.D.'s) for Workmen's Compensation Services in California,* which lists physicians in California who are interested in doing Workmen's Compensation work. It was published in cooperation with the California Conference of Employer Associations, the California Manufacturers Association, the California Self-Insurers Association, the California State Chamber of Commerce, the California Workmen's Compensation Institute and the CMA, and is available through Six Ninety Three Sutter Publications at $5.00 per copy.

30. *Committee on Cancer*

Doctor John Cline, representing Doctor Wood, chairman of the CMA Committee on Cancer, presented to the Council a series of recommendations regarding various activities.

ACTION: Voted to refer the requests to the Committee for Emergency Action, in consultation with the Executive Director and Representatives from the Scientific Board, for study and report back to the Council.

31. *Finance Committee*

Chairman Kay offered to the Council for consideration two additional items for the 1967-68 budget.

a. The addition of $2,600 to the budget submitted for CALIFORNIA MEDICINE, restoring that portion of the budget to its former amount.

ACTION: Voted to approve the addition of $2,600 to the budget for California Medicine.

b. An amount to be allocated in the budget for support of the LACMA Library, pending the report of the ad hoc Committee on Medical Libraries.

ACTION: Voted to approve the inclusion of an amount for support of the LACMA Library, equal to the supplemental appropriation authorized by the Council for the current fiscal year.

It was also requested that the report submitted by Mr. John Connor, the LACMA librarian, be distributed to the members of the Council for their information.

Chairman Kay asked for Council approval of the proposed budget, as amended, which will be submitted to the House of Delegates for its approval.

ACTION: Voted to approve the proposed 1967-68 Budget as amended.

Doctor Kay called to the attention of the Council a progress report on the addition of a health educator to the CMA staff. The report was submitted to the Council for its information only. It reviewed some of the discussions that have taken place regarding the function of a health educator at the CMA.

32. *Automotive and Traffic Safety*

Doctor Kay brought to the attention of the Council two bills governing the operation of two-wheeled motor vehicles which would establish:

a. Standards for protective clothing to be worn by operators;

b. Testing and licensing program for operators

ACTION: Voted to refer both bills to the Committee on Legislation for study and report back to the Council.

Doctor Kay also brought before the Council a resolution submitted by the California Society of Internal Medicine petitioning CMA to recommend that the Department of Motor Vehicles establish a panel of independent medical examiners for examinations of Class I and II drivers' licenses.

ACTION: Voted to refer to the Committee on Legislation for study and report back to the Council.

33. *Study of Current Medical Laboratory Practices* (Resolution No. 105-66)

Doctor Kay presented to the Council a letter from the California Society of Pathologists requesting CMA to designate a committee to work with them in reviewing medical laboratory practices and problems including licensure, standards, quality control and procurement of manpower.

ACTION: Voted to refer the request to the Commission on Community Health Services in consultation with the Committee on Legislation for study and report back to the Council.

34. *Role of Medicine in Society*

Doctor Watts, chairman of the Committee on the Role of Medicine in Society, submitted to the Council three reports and recommendations. The reports, "Responsibility for Quality in Health Care," "A New Relationship with Government and Others," and "Organizing for Health Care," were presented for receipt by the Council. Doctor Watts also asked for authorization to distribute the reports and recommendations, along with the report, "Prologue and Perspective," previously received by the Council, to the House of Delegates.

ACTION: Voted to authorize distribution of the four reports and recommendations, including "Prologue and Perspective," to the House of Delegates and to County Society Officers. The Appendix is to be omitted and a statement included which was amended to read that the reports were received by the Council, that no official action had been taken by the Council on the reports and recommendations, and that the distribution is being made for information only.

35. *Committee on Organizational Review And Planning*

Chairman Teall asked the Council to authorize distribution of the informational report prepared by the Committee to the House of Delegates. The report deals with the goals and objectives of the Committee on Organizational Review and Planning.

ACTION: Voted to authorize distribution of the report, for information, to the House of Delegates.

36. *Commission on Hospital Affairs*

In the absence of Chairman Halter, Councilor Telford reported to the Council. He said that it was the recommendation of the Commission regarding House of Delegates Resolution No. 1-66 (Standardization of Hospital Records) that CMA work concurrently with the California Hospital Association, the California Nurses' Association, Medical Record Librarians and selected computer specialists in an effort to standardize hospital records.

ACTION: Voted to refer to the Commission on Hospital Affairs for implementation.

Councilor Telford also presented the recommendation of the Commission that appointed physician members of hospital utilization committees not be compensated for professional review (House of Delegates Resolution 104-66). It was pointed out that this had been the position of the Council in the past.

ACTION: Voted to reaffirm previous position.

Doctor Telford called upon Mr. Goldman of the CMA staff to review briefly the progress of the Medical Staff Survey Committee to date. Mr. Goldman reported that as of 17 February 1967, a total of 407 hospitals had been visited for medical staff review. Of these, 26 were resurveys for hospital medical staffs which did not receive full approval on the initial list. He pointed out that the surveys will soon reach the 100 per cent mark.

Doctor Telford said that it was the feeling of the Commission and of the Medical Staff Survey Committee that a special study of proprietary hospitals should not be done. He pointed out that much of the intent of the study was being accomplished as the survey of all types of hospitals is conducted. It was suggested, however, that the Survey Committee maintain an up-to-date statistical tabulation on proprietary hospitals.

ACTION: Voted not to conduct a proprietary hospital study but to maintain an up-to-date statistical tabulation.

The last item Doctor Telford brought before the Council was the proposed revision of Section 346(a)(1) of the Hospital Licensing Act. The proposed revision would permit a maternity patient, particularly one with no baby or one who is not to take her baby home, to be placed in a room with "clean" non-maternity patients on a non-maternity nursing unit. The requested change refers to those mothers whose infants were stillborn or whose infants are to be placed for adoption, or remain in the nursery following delivery.

ACTION: Voted to approve the proposed revision.

37. *CMA Retirement Program (Keogh Plan)*

Mr. William Whelan of the CMA staff reported that almost 500 physicians participated in the program. He said that the program includes not only physicians but also their employees, so that the total number of participants is 856. He stated that during the next year the number participating will probably increase substantially because of an amendment to the law making the total contribution of the physician deductible beginning January 1968.

38. *Proposed Bylaw Amendments for Introduction at 1967 House of Delegates Meeting*

Vice Chairman Miller presented a proposed Bylaw amendment to the Council which would permit CMA to charter component society auxiliaries as well as component medical societies.

ACTION: Voted approval of the proposed amendment for consideration by the House of Delegates.

Doctor Miller asked for Council action on the resubmission of the Bylaw amendments that were before the House of Delegates last year and were not considered due to lack of a quorum.

ACTION: Voted to resubmit the Bylaw amendments to the 1967 House of Delegates.

39. *Legal Counsel*

Mr. Hassard stated that he would be sending to each Councilor within the week a summary of the Social Security Amendments of 1967 (H.R. 5710) with an explanatory letter.

He also brought before the Council a request from a Redding physician, George C. Stevenson, M.D., who asked that CMA appear amicus curiae.

ACTION: Voted not to enter into the litigation.

40. *CMA Policy Regarding Statements to Public or Governmental Bodies*

Chairman Anderson presented to the Council a draft of proposed CMA policy regarding statements to public or governmental bodies, along with a letter of transmittal. The proposed policy, as amended, reads as follows:

"I. The president of the CMA is the official public spokesman for the CMA and all of its committees, commissions, bureaus and scientific sections.

"II. All public statements on behalf of the CMA or any commission, committee, bureau or scientific section thereof must have prior approval by the Council of the CMA and shall be made by the President of the CMA or by others whom he, or the Council, shall designate.

"III. Any statement or official action in the name of CMA which must be made on an urgent or emergency basis precluding Council action, must be authorized by the President or the Committee for Emergency Action or, in their absence, by the Executive Director.

"IV. This in no way prohibits any individual physician members from making statements in their own names. In such situations, members of CMA committees, commissions and bureaus should make it clear that they are speaking as individuals and not in the name of CMA.

"V. Any violation of the above procedures shall be reported to the Council for appropriate action."

ACTION: Voted to approve the above as CMA policy and to transmit it to all CMA Officers, Councilors, Committee and Commission Members, Scientific Section Officers, Staff and Executive Secretaries.

41. *Nursing Home Utilization Criteria*

Doctor H. Dean Hoskins of the Committee on Fees presented and commented upon proposed criteria to assist in identification of inappropriate utilization of convalescent and nursing home services under insurance programs.

ACTION: Voted to approve the general concepts embodied in the proposed criteria and to authorize the Committee on Fees to develop them and to work with appropriate organizations on their implementation.

42. *Joint Congressional Visitation*

Following up on its 15 January 1967 action to approve a CMA-Blue Shield joint liaison effort with California legislators in Washington, D.C., the Council considered the amount of money to be allocated for this purpose.

ACTION: Voted to approve the allocation of $5,000 for the joint CMA-Blue Shield liaison effort in Washington, D.C.

43. *Membership*

Thirty applicants were voted election to Associate Membership. These were: Homer A. Casey, John R. Goldsmith, Hugh J. Lenahan, Marianne Schour, J. Austin Trolan, Alameda-Contra Costa County; Henry Loon, Lassen-Plumas-Modoc-Sierra County; Carl M. Agliozzo, Oscar Auerbach, Carmen C. Calescibetta, Lida G. Keyes, John M. Mead., Jr., Carl B. Nagel, Ralph Andrew Olsen, Henry P. Rover, Charles E. Schoettlin, Donald C. Shelby, John S. Stephens, George M. Uhl, Ronald Eugene Saul, Los Angeles County; William J. McCann, Marin County; William Gerald Burrows, Mendocino-Lake County; Colin T. Greenlaw, Sacramento County; Barbara J. Haddad, Giuseppe Incsi, Elizabeth B. Reed, San Francisco County; Jean Haber Green, Donald D. Jackson, Charles G. Merckel, Jr., Santa Clara County; Velma E. Coffin-Kwart, James H. Lyons, Ventura County.

Twenty-three members were voted election to Retired Membership. These were: A. Miles Griffin, Robert J. Harmon, Alameda-Contra Costa County; Isabel D. Brown, Robert M. Francis, Joseph William Hankins, Raymond P. Kellogg, Francis B. Linne, Dora B. Olkon, Clarence Pierson, Elroy F. Sheldon, Samuel J. Weinberg, Los Angeles County; Gilbert Chassey, San Diego County; Gunther W. Nagel, Clarence Nelson, Hale F. Shirley, Henry A. Stephenson, San Francisco County; Delbert H. McNamara, Doris E. McNamara, Santa Barbara County; Charles M. Colianni, Richard N. Jacobs, Louis Kovitz, Santa Clara County; Alexander W. Cruden, Franklin H. Garrett, Ventura County.

Reduction of dues was voted for 52 members for reasons of prolonged illness or postgraduate education.

44. *Roll Call*

Present were President MacLaggan, President-Elect Morrison, Speaker Quinn, Vice Speaker Telford, Secretary Weyrauch, and Councilors Anderson, Dwight Wilbur, Isenhour, Eastman, Melone, Todd, Gooel, Bullock, O'Connor, Ham, Rogers, Crum, Boyle, Burnett, Richard Wilbur, Miller, Watts, Fenlon, Kay, Kaiser, Yant and Grunigen and Immediate Past President Teall. Absent for cause were Councilors Taw, Maguire and Longmire.

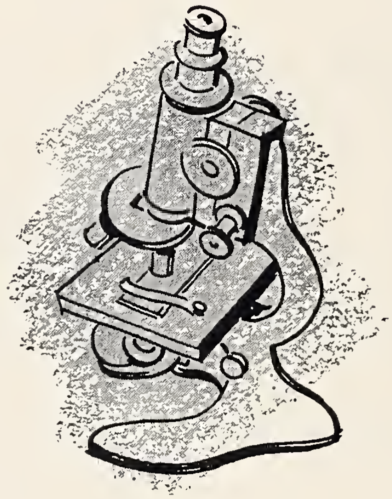
Present by invitation were CMA staff members Becker, Belser, E. Collins, J. Collins, Curley, Eberlein, Goldman, Griffith, Hetland, Jones, Klutch, Lemos, Miller, Price, Redfern, Thomas and Whelan; Messrs. Hassard and Huber, legal counsel; component society executives Scheuber of Alameda-Contra Costa, Rideout of Butte-Glenn, Garrick of Forty First, Lingerfelt of Fresno, Geisert of Kern, Brock of Imperial, Dalbec of Los Angeles, Sower of Marin, Colvin of Monterey, Bannister of Orange, Walters of Riverside, Dochterman of Sacramento, Donmyer of San Bernardino, Neick of San Francisco, Thompson of San Joaquin, Wood of San Mateo, Marvin of Santa Barbara, Donovan of Santa Clara, Brown of Sonoma, Whitehall of Stanislaus, and Bruce of Tulare; Messrs. Paton, Babb, Clark, Heller, Koch, Light, Parke, and Potloff of Blue Shield-California

Physicians' Service; Messrs. Read, Brown, Putnam and McWilliams of the Public Health League; Doctor Cullen of UC-San Francisco; Doctor Glaser and Doctor Gonda of Stanford; Doctor Mellinkoff and Doctor Dillon of UCLA; Doctor Egeberg of USC; Doctor Radl of State Department of Employment; Doctor Hewitt of State Department of Mental Hygiene; Doctor Skelly of the State Department of Social Welfare; Doctor Breslow of the State Department of Public Health; Mr. Mulder of the State Health and Welfare Agency; Mrs. Kahn of the Bureau of Health Insurance, Social Security Administration; Mrs. Bacon of the Woman's Auxiliary to the CMA; Mrs. Stanley of the California Nurses' Association; Doctor Kieran and Mr. Hampshire of the California Interagency Council on Smoking and Health; Mr. Snyder and Doctor Tolls of the California Society of Pathologists; Mr. Ward of the California Committee on Regional Medical Programs; Mr. Cattaneo of the American Hospital Association; Mr. Cumming of the California Hospital Association; Doctor Dean of the California Veterinary Medical Association; Messrs. Frisch and Kennedy of *Medical World News;* Doctor Hudson and Messrs. Gould and Uzemack of the American Medical Association; Doctors Besson, Clark, Eckmann, Garrett, Green, Herzog, Howard, Kilroy, Mazan, Murray, Steinberg Turner, and Wayburn.

45. *Adjournment*

The meeting was adjourned at 5:30 p.m.

Carl E. Anderson, M.D., *Chairman*
Helen B. Weyrauch, M.D., *Secretary*



In Memoriam

Barton, John C., Riverside. Died 15 March 1967, in Riverside, aged 59. Graduate of University of Minnesota Medical School, Minneapolis, 1934. Licensed in California in 1944. Doctor Barton was a member of the Riverside County Medical Association.

◆

Berlin, Harold D., Los Angeles. Died 11 January 1967 in Los Angeles, aged 73 of occlusive coronary arteriosclerosis. Graduate of Northwestern University Medical School, Chicago, 1920. Licensed in California in 1921. Doctor Berlin was a retired member of the Alameda-Contra Costa Medical Association and the California Medical Association, and an associate member of the American Medical Association.

◆

Brindamour, Jean-Louis Edmond, Palo Alto. Died 24 March 1967, in Palo Alto, aged 68. Graduate of Jefferson Medical College of Philadelphia, 1924. Licensed in California in 1930. Doctor Brindamour was a retired member of the San Francisco Medical Society and the California Medical Association, and an associate member of the American Medical Association.

◆

Delamere, Granville S., Marysville. Died 21 March 1967, in Marysville, aged 76. Graduate of University of California School of Medicine, Berkeley and San Francisco, 1921. Licensed in California in 1921. Doctor Delamere was a retired member of the Yuba-Sutter-Colusa County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

◆

Greenwood, Earl Norman, San Francisco. Died 28 March 1967, in San Francisco, aged 89. Graduate of Cooper Medical College, San Francisco, 1907. Licensed in California in 1907. Doctor Greenwood was a retired member of the San Francisco Medical Society and the California Medical Association, and an associate member of the American Medical Association.

◆

Griggs, Donald Ezra, Glendale. Died 9 March 1967, in Glendale, aged 66, of heart disease. Graduate of College of Medical Evangelists, Los Angeles, 1924. Licensed in California in 1924. Doctor Griggs was a member of the Los Angeles County Medical Association.

◆

Groff, Sutten Henry, Granada Hills. Died 20 March 1967, aged 79, of coronary infarction. Graduate of Drake University College of Medicine, Des Moines, Iowa, 1918. Licensed in California in 1919. Doctor Groff was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

◆

Harris, Robert Henry, Valley Center. Died 15 March 1967, in Escondido, aged 59, of heart disease. Graduate of the Rush Medical College, Chicago, 1935. Licensed in California in 1949. Doctor Harris was a member of the Los Angeles County Medical Association.

Hunter, William Edward, Los Angeles. Died 27 February 1967, in Los Angeles, aged 75, of hemorrhage due to perforated duodenal ulcer. Graduate of Harvard Medical School, Boston, 1916. Licensed in California in 1929. Doctor Hunter was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

◆

Imerman, Harold Maurice, Beverly Hills. Died 25 March 1967, in Los Angeles, aged 59, of Hodgkins disease. Graduate of St. Louis University School of Medicine, 1933. Licensed in California in 1946. Doctor Imerman was a member of the Los Angeles County Medical Association.

◆

McNeil, Edwin Ewert, Los Angeles. Died 16 March 1967, in Los Angeles, aged 61. Graduate of University of Colorado School of Medicine, Denver, 1931. Licensed in California in 1944. Doctor McNiel was a member of the Los Angeles County Medical Association.

◆

Schierman, Daniel William, Whittier. Died 12 March 1967, in Glendale, aged 71, of coronary infarction. Graduate of College of Medical Evangelists, Los Angeles, 1925. Licensed in California in 1926. Doctor Schierman was a member of the Los Angeles County Medical Association.

◆

Siefert, Alfred Camille, Oakland. Died 10 March 1967, in Oakland, aged 79, of bowel obstruction due to carcinoma. Graduate of Stanford University School of Medicine, Palo Alto and San Francisco, 1914. Licensed in California in 1914. Doctor Siefert was a retired member of the Alameda-Contra Costa Medical Association and the California Medical Association, and an associate member of the American Medical Association.

◆

Smith, Freid Lynn, Colfax. Died 18 February 1967, in San Francisco, aged 60, of esophageal hemorrhage. Graduate of Northwestern University Medical School, Chicago, 1934. Licensed in California in 1935. Doctor Smith was a member of the Placer-Nevada County Medical Society.

◆

Spasser, Irvin Charles, Los Angeles. Died 11 March 1967, in Los Angeles, aged 53, of malignant sarcoma of the right cerebral hemisphere and mid-brain. Graduate of the University of Southern California School of Medicine, Los Angeles, 1959. Licensed in California in 1960. Doctor Spasser was a member of the Los Angeles County Medical Association.

◆

Weber, Robert, Los Angeles. Died 15 March 1967, in Los Angeles, aged 56, of lymphatic leukemia. Graduate of Hamburgische Universität Medizinsche Fakultät, Hamburg, Germany, 1936. Licensed in California in 1953. Doctor Weber was a member of the Los Angeles County Medical Association.

17th annual regional postgraduate institute

LAKE TAHOE HOTEL
Incline Village, Nevada
June 14-16, 1967

SACRAMENTO VALLEY COUNTIES

Presented cooperatively by Sacramento Valley Counties Medical Societies, Loma Linda University School of Medicine, and the Committee on Continuing Medical Education, California Medical Association. A 15 hour course.

HOST: Sacramento County Medical Society
***Regional Co-Chairmen:* A. John Quinn, M.D., Robert G. Myers, M.D.**

Institute Fee: $15.00. For additional information contact Continuing Medical Education office, California Medical Association, 693 Sutter Street, San Francisco 94102. All California Medical Association members and their families are cordially invited to attend.

GUEST SPEAKER: Michael James Brennan, M.D., Professor of Medicine, Wayne State University College of Medicine; sub specialty: Clinical Hematology and Oncology; Host Tumor Relationship; Experimental and Clinical Chemotherapy; Scientific & Professional Director, Michigan Cancer Foundation.

PROGRAM THEMES:

CHEST . . . BREAST

WEDNESDAY, JUNE 14, 1967

12:30—Registration

Afternoon Session

1:15—**Welcome**—James W. Martin, M.D., President Sacramento County Medical Society

Presiding: Robert G. Myers, M.D.

1:30—**Chest Pain**—Varner J. Johns, Jr., M.D.

Panel Discussions

2:15—**Cardiac Pain**—
Moderator: William G. Bush, M.D.
Panel: Raymond Herber, M.D., Varner J. Johns, Jr., M.D., and L. Arno Lejnieks, M.D.

3:00—**Intermission**

3:15—**Musculo-Skeletal-Chest Wall Pain**—
Moderator: Bryant Sheehy, M.D.
Panel: Varner J. Johns, Jr., M.D., Walter E. Macpherson, M.D., and Max D. Shaffrath, M.D.

4:00—**Gastro-Intestinal Causes of Chest Pain**—
Moderator: John B. Reardon, M.D.
Panel: Raymond Herber, M.D., Walter E. Macpherson, M.D., and Dwight L. Wilbur, III, M.D.

THURSDAY, JUNE 15, 1967

Morning Session

7:30—**Breakfast Roundtable Discussions**—

8:30—**Motion Picture**—"Medicine in Africa"

Presiding: A. John Quinn, M.D.

9:00—**Respiratory Distress**—Walter E. Macpherson, M.D.

9:45—**Radiological Aspects of Respiratory Distress**—Geoffrey A. Gardiner, M.D.

10:20—**Intermission**

Panel Discussions

10:40—**Pulmonary Physiology and Respiratory Distress**—
Moderator: Harold M. Lowe, M.D.
Panel: Roy V. Jutzy, M.D., Walter E. Macpherson, M.D., and K. Peter Poirier, M.D.

11:15—**Respiratory Distress in the Child**—
Moderator: Glen G. Caylor, M.D.
Panel: Robert F. Chinnock, M.D., Mark Dentinger, M.D., and Geoffrey A. Gardiner, M.D.

12:00—**Luncheon**—For Physicians and Wives

Presiding: A. John Quinn, M.D.

Speaker: Alonzo Baker, Ph.D., Professor and Chairman, Department of Political Science, Loma Linda University

Afternoon Session

Presiding: Pierce A. Rooney, Jr., M.D., President-Elect, Sacramento County Medical Society

2:00—**Lumpy Breasts**—Michael J. Brennan, M.D.

Panel Discussions

2:45—**When Should Breast Lesions Be Considered Benign**—

Moderator: Frank J. Glassy, M.D.

Panel: Michael J. Brennan, M.D., Gordon P. Griggs, M.D., and Dixon L. Hughes, M.D.

3:30—**Intermission**

3:45—**Hormones and Breast Disease**—

Moderator: Richard K. Shaw, M.D.

Panel: Michael J. Brennan, M.D., Geoffrey A. Gardiner, M.D., Gordon P. Griggs, M.D., and James Reece, M.D.

4:30—**Breast Problems Related to Pregnancy**—

Moderator: J. Willison Allen, M.D.

Panel: Michael J. Brennan, M.D., Gordon P. Griggs, M.D., Alex Janushkowsky, M.D., and Franklin K. Yee, M.D.

FRIDAY, JUNE 16, 1967

Morning Session

8:30—**Motion Picture**—"The Severely Traumatized Patient"

Presiding: Robert G. Myers, M.D.

9:00—**Chest Injuries**—Ellsworth E. Wareham, M.D.

Panel Discussions

9:45—**Management of Acute Chest Injuries**—

Moderator: Donald M. Hopkins, M.D.

Panel: Roy V. Jutzy, M.D., Norman S. Lovell, M.D., and Elsworth E. Wareham, M.D.

10:30—**Intermission**

10:45—**Postoperative Pulmonary Complications**—

Moderator: Malcolm M. McHenry, M.D.

Panel: Patrick C. Dietler, Jr., M.D., Roy V. Jutzy, M.D., and Wilfred M. Huse, M.D.

11:30—**Assisted Respiration**—

Moderator: Setty E. Porco, M.D.

Panel: Roy V. Jutzy, M.D., Ellsworth E. Wareham, M.D., and Charles M. Webster, M.D.

Continuing Medical Education

Postgraduate Medical Education

JOHN B. DILLON, M.D.

Assistnat Dean and Professor of Surgery/Anesthesiology, UCLA School of Medicine; Chairman, Committee on Scientific Assemblies, and member, Committee on Continuing Medical Education, Scientific Board of the California Medical Association.

THERE IS SUBSTANTIAL recognition by physicians generally as to the necessity for continuing education in medicine. This recognition has been expressed by the Board of Trustees of the American Medical Association in the recent formation of a committee for the specific purpose of studying the problem in depth. In California the need has been stated by Watts[1] in a series of articles entitled "Medicine in Society" and by Manning[2] in "Evaluating Postgraduate Courses." This necessity is further expressed by the increased emphasis on continuing education by medical schools, medical societies, and by the various specialty groups.

Millis[3] in his foreword to "The Graduate Education of Physicians" states the problem very clearly:

> For any learned profession there are but two alternatives for establishing standards of practice and education. Responsibility can be assumed by a society as a whole, operating through government, or can be assumed by the organized profession through a voluntarily accepted self-discipline. There are no other alternatives, for, if the profession does not take responsibility, society will surely demand that the vacuum be filled and the government assume the responsibility. It is the conviction of the Commission that the profession of medicine should assume the responsibility for its standards of education and should have a mechanism adequate to the full discharge of these responsibilities.

A more direct approach is the following clipping from *Medical World News,* 20 January 1967:

> "It makes no sense to audit hospitals and not doctors," says Dr. George James, Dean of Mount Sinai School of Medicine in New York City. Hospitals must meet high standards to qualify for reimbursement of federally supported services, notes Dr. James, former New York City Health Commissioner. Yet most health care takes place away from the hospital bed. For this reason, he advocates the "auditing" of doctors also, to make sure that they are giving high-quality medical care to patients whose medical expenses are covered by Title 19 (Medicaid).

Yet with the recognition of the necessity for continuing education there is as yet no practical mechanism by which many physicians in very busy solo practice can voluntarily avail themselves of such education except in a hit or miss fashion.

A method to solve this problem has been suggested by Berman that is certainly worthy of serious consideration.[4] He proposes a period of hospital training at regular intervals with arrangements for *locum tenens* and adequate financial coverage.

This is not a problem that the medical profession can long delay solving. There is considerable evidence that the public is increasingly interested and concerned. In fact, the medical profession may be too late in this solution in view of recent statements by President Johnson asking for more practical results from research, as it was in providing a solution to the problem of medical care costs. There is no time to be aloofly philosophical. That much is certain.

The question in respect to continuing education is not *if* but *how.* That this is unanswered is demonstrated by the number of agencies involved in attempting to provide for the need. There are postgraduate programs or programs with the same intent put on at all levels of educational competence, and yet it is apparent to many educators, despite attendance statistics, that relatively few physicians are reached in an effective way. Manning[2] estimated that only 30 per cent of physicians in California participate in continuing education. And, as is said by churchmen with similar but different interests, Most of those who most need to be in church are on the golf links.

It appears clear to me that several things have happened to medicine as a whole that to a large degree have resulted from the burgeoning of tech-

---

Reprint requests to: Department of Surgery/Anesthesiology, UCLA School of Medicine, Los Angeles 90024.

nical and scientific advances of the past decade. The first is that because of improvement in communications of all types the public has become aware that there is a discrepancy between what medicine is reputed to promise, as expounded by some of its vocal "spokesmen," and what in fact it delivers, as is observed in cold reality. In the words of the Millis Report[3]: "The informed public also knows, however, that a wide gap exists between the best that medicine can offer and the lesser services actually available to many patients. Medical practice has changed greatly. Yet the judgment is widely expressed, in and out of medicine, that the changes have not been profound enough to keep pace with the growth of medical knowledge and the rise in society's expectations and demands. The current problems of medicine are in large measure problems created by its own success." I believe that this awareness is expressed by the ever increasing notice given to medical matters in the public press (*Life, Time, News Week, Wall Street Journal, Ladies Home Journal, McCall's, Readers' Digest, New Yorker,* etc.). It appears to me that the public is beginning to call for a sociological accounting by medicine. It is doing this in one way that is not widely appreciated, but is really frightening, and that is by the ever increasing allegations of negligence against physicians. "Society cannot and will not indefinitely tolerate subordination of the public weal to professional conflict or aggrandizement—whatever the profession, medicine, law, or any other. A profession is primarily for public service, not personal profitability."[5]

It appears that not only does the scientific and clinical knowledge of physicians need to be kept up to date, but that they acquire social consciousness and a real sense of responsibility to society as a whole. This latter is an area that many physicians have ignored. The public is beginning to look at medicine as a body politic with vested interest. The problems that medicine faces are discussed by Magraw[6] and Galdston[7] in a series of pointed observations on the relationship between the public and medicine. One of the most complete analyses of the present turmoil between medicine and the government is in a series of four articles by Richard Harris in the *New Yorker*[8] in which he reviews the social movement in medicine, not only internationally, but in the United States, in detail:

Dealing with the problem of science in its broadest sense, René Dubois[9] has made a most comprehensive indictment. This indictment medicine as part of science must answer and answer realistically:

> Now it can be said that it is possible to achieve almost anything we want—so great is the effectiveness of technology based upon the experimental method. Thus, the main issue for scientists and for society as a whole is now to decide *what* to do among all the things that could be done and should be done. Unless scientists are willing to give hard thought—indeed, their hearts—to this latter aspect of their social responsibilities they may find themselves someday in the position of the Sorcerer's Apprentice, unable to control the forces they have unleashed. And they may have to confess, like Captain Ahab in Moby Dick, that their methods are sane, their goal mad.

As medicine has become more scientific it certainly appears to have become less humane and more concerned with the exception than with the usual. While the American public appears to respond in a generous way to the problems of the exceptional, it responds in a violent way to personal ones; and as science tends to make the exceptional common and more personal, the public is likely to revolt. Comments are beginning to appear in the lay press more frequently on "what to do for grandma." Society is beginning to recognize, even if medicine doesn't, that while the increase in survival is desirable in principle it poses other problems which medicine has not helped to solve—rather to create.

There is no question in my mind that the brightness of the image of medicine as a whole has been tarnished or, let us say, that things being as they are in this world, the public is taking a very cold and calculating look at medicine. I believe that thus far the image of the individual physician has not been more than mildly scratched, but I am convinced that the individual physician is on trial and that the outcome of this trial is related to the continuing postgraduate education of the physician and to a very real reappraisal by physicians of their position in society and their responsibilities to it.

One of the questions that must be answered in respect to continuing medical education is: Should there be control on the numbers and sources of such effort? Superficially, it might be thought that the more the number of educational exposures or potential exposures the better. This may be true to some extent, but I believe there is a practical limit beyond which there tends to be confusion and competition for audiences. It may be that the practical limit already has been passed.

Today, besides the educational efforts of medical schools, some form of continuing education is afforded by: (1) Hospital staffs; (2) specialty associations; (3) local medical societies; (4) state or regional medical societies; (5) national medical societies (these societies may be special or general); and (6) international medical societies (special and general). Most of these sources have some form of journal or periodical in addition to movies, meetings, seminars, discussion groups and, lately, television programs.

The physician is bombarded through the mail by all types of prospectuses outlining the efforts of the various interested groups, all of whom have, at least superficially, the interest of the physician and his patients at heart. There are from time to time, unfortunately, programs that appear to have social or pecuniary gain as their purpose. Because of its very volume, much of this material is discarded unread.

There is a confusion of tongues in these multitudinous offerings, and I believe that if continued education of the physician is to take place in an orderly and useful way, some control must be put on the type and numbers of programs offered.

It would appear to me that the problem of continuing medical education indicates two very distinct concepts. The body of medicine which is close to public need and welfare as represented by the American Medical Association and its component societies, seems to be in a logical position to indicate what is required in a broad sense.

The medical school* with its educational talent and facilities should respond to the need by providing the subject matter and presentation to satisfy that need.

There is certainly no need for competition between "town and gown," for there is more to do than either element alone or both combined can accomplish in anywhere near a satisfactory manner. And certainly together they can do better than any other agencies.

It is questionable today whether the hospital staffs and medical societies can occupy the same immediate position in respect to education that they did even a decade ago. There are very few if any cities today that do not have a medical school within a few miles or hours. The nearest school may not be in the same state, but at this level of educational effort this should constitute no barrier—as indeed it does not.

This by no means suggests that hospital staffs and medical societies should not participate in educational efforts, but it does suggest that the participation not be to the extent and depth that they are educational institutions in their own right as some are attempting to be today to the perhaps unintentional exclusion of organizations that are expert in education. There is no suggestion that this format would involve very highly specialized groups except to the extent that they might be helped in content and presentation.

That this idea is not unique is evidenced by the now rapid growth of departments of "Continuing Medical Education" or "Medical Extension" in medical schools and their being utilized by at least a few state medical societies and other groups for both numbers and content of programs. In this area California is most fortunate.

BIBLIOGRAPHY

1. Watts, Malcolm S. M.: Medicine in Society: Part I—Change and challenge, Calif. Med., 101:417-21, 1964; Part II—Some dimensions of medicine in modern society, Calif. Med., 102:28-32, 1965; Part III—A role for medicine in modern society, Calif. Med., 102:133-138, 1965.

2. Manning, Phil R.: Evaluating postgraduate courses, Calif. Med., 104:326, 1966.

3. Millis, J. S.: The Graduate Education of Physicians. Published by the Council on Medical Education, Amer. Med. Assn., August 1966.

4. Berman, L. G.: A design for continuing medical education, J. Med. Education, 41:965, 1966.

5. Louisell and Williams: Trial of Malpractice Cases, p. 106, Mathew Bender & Co., 1965.

6. Magraw, Richard M.: Ferment in Medicine, W. B. Saunders Co., 1966.

7. Galdston, Iago: Medicine in Transition, Univ. of Chic. Press, 1965.

8. Harris, Richard: New Yorker, 2 July et seq. 1966.

9. DuBois, René: Horizon, Vol. III, No. 6, July 1961.

10. Layman, William A.: University affiliation for medical schools, J.A.M.A., p. 135, 18 July 1966.

---

*Medical school is used as a source rather than university because a significant number of medical schools are not university-connected and most are physically removed from university campuses, although technically part of the university.[10]

PUBLIC HEALTH REPORT

Lester Breslow, M.D., M.P.H.
Director, State Department of Public Health

For the first time in decades, the rate of infectious syphilis in California declined last year—another illustration of what the forces of organized medicine and public health can accomplish toward the improvement of the health and wellbeing of the people of the state.

Teamwork of the same order produced several landmarks in the 1960s, including an overwhelming victory over paralytic poliomyelitis; some turning back of the inroads of tuberculosis in the general population; and an alert program of early detection of cancer, particularly cancer of the cervix. And now a concerted effort is needed against the disease which strikes hard and relentlessly among our younger citizens—infectious syphilis.

What private practitioners of medicine, public health workers and an aroused citizenry can do to combat a threat to health is perhaps best illustrated by the cooperation that was brought to bear on a violent outbreak of syphilis which occurred in a Southern California county in January of 1966.

This episode revolved about a family physician and his patient, an attractive and popular 15-year-old high school girl who worried little about disease. But in January 1966 the young girl became the first of 18 persons with new cases of infectious syphilis reported in her county within a period of two weeks.

Not many cases of syphilis had been reported in the county in all 1965, and it was apparent an outbreak of infectious syphilis was in progress. The outbreak *per se* was not unique, since venereal disease has been "breaking out" for a long, long time. What was exciting were the measures taken to control the outbreak in the county.

As soon as the girl's physician made the diagnosis and began treatment, he reported the case to the local health department. He quickly accepted the department's offer of epidemiologic services and arranged an interview between the teenager and a trained interviewer from the health department. Her sex contacts were identified and located. Examination disclosed further infections. In all, more than a hundred persons were involved in the outbreak. The first patient, another 15-year-old, a five-year-old, and a seven-month-old baby with congenital syphilis were brought to treatment as a result of the investigation.

When the number of cases indicated a possible public health threat, health department officials met with the local medical society. Together they planned an attack calculated to halt further spread of the disease. Members of the society were informed that an unusual number of cases of syphilis were being found in the county, and epidemiologic services were offered to physicians in any cases they might find.

A past president of the medical society prepared taped radio spot announcements concerning the problem. The announcements informed the public that there was an unusual number of cases of infectious syphilis and that everyone should have knowledge of the disease. Pamphlets were provided to listeners desiring information about venereal disease. Members of the medical society spoke before civic groups to present additional information to the community.

The local Junior Chamber of Commerce accepted the venereal disease problem as a community project, agreeing to assist in the dissemination of still more information to residents of the county. Forming a Venereal Disease Public Awareness Committee, the Jaycees became an active part of the control program.

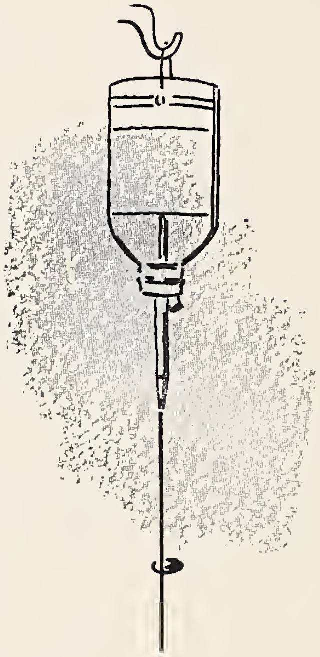
As a result of the efforts by the medical society, the local health department, the Jaycees and press cooperation, the school board took a closer look at venereal disease education in their schools; many county residents who had not been concerned with the venereal disease problem sought more enlightenment.

Today this outbreak has been controlled but the community now realizes that VD remains a problem still requiring final solution. And once again, confronted with a community health problem, private medicine and public health in one of California's 58 counties joined forces to control it.

Venereal disease may not be a sizable part of an individual physician's practice, but it must be pointed out that last year 231 deaths in California were attributable to syphilis, a disease which can be eradicated.

If we look at the diseases that have been controlled — diphtheria, typhoid, poliomyelitis, to name a few—it becomes increasingly obvious that it is the team approach that achieves control. Public health today is determined to adopt this approach to venereal disease on a statewide basis.

As the foregoing story demonstrates, the medical profession and public health can attract the general citizenry to join the endeavor to reduce deaths, mental illness and the birth defects which are directly linked to venereal disease.



NEWS & NOTES

NATIONAL • STATE • COUNTY

LOS ANGELES

University of California Regents have approved **relocation of the California College of Medicine** from Los Angeles to the Irvine campus in Orange County.

The decision followed recommendation made by a special Regents' committee which conducted a five-month study of alternate sites for the college. Trustees of the college had previously approved the move.

The Regents' committee noted that **the volume of patients was decreasing** at the present site and that a study of the Irvine area's present and future needs "indicates that a new facility at that location could well serve the rapidly growing population."

The report also noted that the present land holdings of the California College of Medicine are "entirely inadequate for a medical school" but that land already owned by the Regents at the Irvine site is available in ample quantity for a complete medical center.

* * *

The Western Divisional Meeting of the **American Psychiatric Association** will be held at the Statler Hilton Hotel, Los Angeles, October 18-22. Information may be obtained from Dr. Ralph M. Obler, 11860 Wilshire Boulevard, Los Angeles 90025.

SAN DIEGO

The San Diego County Heart Association will present its seventeenth annual Professional Symposium on Cardiovascular Diseases September 29 and 30, at the Hanalei Hotel in San Diego. Further information about this program may be obtained from Dr. James E. Lasry, 3330 Third Avenue, San Diego 92103.

SAN FRANCISCO

Dr. Peter C. Duke, a resident at University of California Medical Center, San Francisco, is the recipient of a Mead Johnson $1,000 **scholarship award** in support of graduate training in anesthesiology.

Three such awards are made annually by the American Society of Anesthesiology to resident physicians who have demonstrated meritorious work in the clinical or research fields.

* * *

The Northern California Chapter of **American College of Surgeons** will hold its annual meeting in San Franicsco, Saturday, May 20. Scientific programs on general surgery and various surgical specialties will be held morning and afternoon. Further information may be obtained from Dr. F. William Blaisdell, San Francisco General Hospital, 22nd Street and Potrero Avenue, San Francisco 94110.

SANTA BARBARA

At the seventeenth annual meeting of the **California Society of Plastic Surgeons,** held at Del Monte Lodge, Pebble Beach, April 13-15, the following officers were elected: President, Dr. Kathryn L. Stephenson, Santa Barbara; president-elect, Dr. Henry Patton, Oakland; vice president, Dr. Clifford Barber, Glendale; and secretary-treasurer, Dr. Donald Barker, Van Nuys.

The Physician's BOOKSHELF

DR. THOMAS SYDENHAM (1624-1689), His Life and Original Writings—By Kenneth Dewhurst. University of California Press, Berkeley and Los Angeles, 1966. 191 pages, $6.00.

Thomas Sydenham (1624-1689) fully merited the appellation "the English Hippocrates," possessing in good measure, as Dr. John Brown wrote in his *Horae subsecivae,* the four qualities Hippocrates says are indispensable in every good physician—learning, sagacity, humanity, and probity. Sydenham's near contemporaries held him in the deepest reverence, and Herman Boerhaave (1668-1738) went so far as to remove his hat whenever he mentioned his name. In modern times, Sir William Osler placed Sydenham in his *Bibliotheca prima* of the seventeenth century among such giants as Bacon, Harvey, Galileo, and Newton, whereas the historian Fielding Garrison categorized Sydenham as one of the greatest figures in internal medicine. Despite such high praise, few physicians of today would be able to discuss the life or nature of the contribution of this major figure in medicine, save that his name is attached eponymously to Sydenham's chorea. Presumptively, this has been due to the fact that, except for a few brief essays, the only biographies are those of Joseph Payne in English, published in 1900, and of Louis Picard in French, presented as a thesis in 1889. The present work goes far towards remedying this gross deficiency.

The author divides this small book into two parts. The first part is a brief life—of little more than sixty pages—of this Puritan captain-of-horse turned physician, his family background, his relationship to colleagues, pupils, followers, and friends, with careful attention to the contemporary scene in the transition from the Civil War to the Restoration, and an analysis of Sydenham's contribution. The second part of some hundred pages presents a number of Sydenham's writings in their original dress, namely in English. As is well known, Sydenham's writings were for the most part translated into highly idiomatic Latin by his friends so that English editions of his works are in reality re-translations which conceal the vigor and robustness of his thought and style. The author traces the sources and history of these writings in an introductory essay of great value for their understanding.

Dr. Dewhurst has compressed a great deal of information, much from original sources, into this brief biography of Sydenham. His account is most satisfying and valuable. Nonetheless, it is not without some serious weaknesses. The author concludes (page 59) that the lasting value of Sydenham's writings lay in his ridding the *pharmacopeia* of many dangerous and obnoxious remedies, his pioneering of quinine, and his simplification of treatment and reliance on fresh air, exercise, and a moderate diet. Yet most would agree that Sydenham's greatest contribution lay in his concept of the categorization of disease and establishment of the clinical entity. "All diseases then ought to be reduced to certain and determinate kinds . . .," he said His revival of the objective and naturalistic spirit of Hippocrates was the outcome of his program of research which rested upon "collecting a genuine and natural description, or history, of all diseases." Further, there is no discussion of Sydenham's "Epidemic constitution" which became for him the central feature of the etiology of disease—a theory which forced him to discard ideas of contagion and the most regressive aspect of his progressive thinking. It is also surprising that the author relies on John Ward and later writers for authority on the Latin translators of Sydenham to the omission of the written statement by Dr. John Mapletoft's own son in a copy of the *Opera Universa,* given to Dr. John Latham by the grandson, and which later came into possession of the Brighton and Sussex Medico-Chirurgical Society Library. In this copy inscribed in Sydenham's own hand, the Reverend John Mapletoft (the doctor's son and John Ward's source) enumerates those books translated by his father and those by Gilbert Havers.

Despite the lack of a deeper critical analysis of Sydenham's contributions, this is a most worthy book. It is a joy to read and provides insight and perspective on the continuing problem of the widening gap between the science and art of medicine and the true meaning of clinical medicine. Every physician will want to own this small volume and to reflect upon the significance and meaning of Sydenham as the founder of modern clinical medicine.

John B. deC. M. Saunders, M.D.

* * *

ATLAS OF VASCULAR SURGERY—Second Edition—By Falls B. Hershey, M.D., F.A.C.S., Associate Professor of Clinical Surgery, Washington University School of Medicine, St. Louis; and Carl H. Calman, M.D., F.A.C.S., formerly Assistant in Clinical Surgery, Washington University School of Medicine, St. Louis. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis, Missouri 63103, 1967. 318 pages, $19.50.

This is a second edition of an atlas which appeared in 1963. The material has been expanded to include recent developments in the general field of peripheral vascular surgery. There is still no mention of acute aortic dissections which have important peripheral manifestations, and certainly it would appear well within the intended scope of the book to present their surgical management. The illustrations are excellent and concise. Again the book affords good background material for residents and practicing surgeons, but it does not mean to be a comprehensive presentation of the field.

Norman E. Shumway, M.D.

dependability not dependence

ATARAX®
(hydroxyzine HCl) tablets, syrup

Product Information

Contraindications: Use in pregnancy: When administered to rats at doses substantially above the human therapeutic range, hydroxyzine induced fetal abnormalities. Until human clinical data are available adequate to establish safety in early pregnancy, hydroxyzine is contraindicated in early pregnancy. The drug is contraindicated for patients who have shown previous hypersensitivity to it.

Precautions: Hydroxyzine HCl may potentiate narcotics, barbiturates, meperidine and other CNS depressants. In conjunctive use, dosage of these drugs should be reduced by as much as 50 per cent. Atropine and other belladonna alkaloids are not affected by hydroxyzine. Because drowsiness may occur, patients given hydroxyzine should be cautioned against driving a car or operating dangerous machinery.

Adverse reactions: No serious side effects resulting from the oral administration of hydroxyzine have been reported and confirmed to date. Therapeutic doses seldom produce impairment of mental alertness. The transitory drowsiness that may occur usually disappears spontaneously in a few days of continued therapy or can be corrected by dosage reduction. Dryness of the mouth may be encountered at higher doses.
The absence of toxic effects on the liver and bone marrow has been demonstrated by extensive clinical use at recommended doses for more than four years of uninterrupted therapy and by experimental studies in which excessively high doses were given. Involuntary motor activity, including rare instances of tremor and convulsions, has been reported, usually with considerably higher than recommended doses.

Dosage: Ranges from 25 mg. t.i.d. to 100 mg. q.i.d.

References: 1. Fishbein, M.: J. Am. Geriatrics Soc. 10:911 (Nov.) 1962. 2. Dale, P. W.: New England J. Med. 265:185 (July 27) 1961. 3. Settel, E.: Am. Pract. 8:1584 (Oct.) 1957.

J.B. ROERIG DIVISION
CHAS. PFIZER & CO., INC.
NEW YORK, N.Y. 10017

Air Pollution's Effects on Runners

The motor-fuel oxidant type of air pollution in the Los Angeles area may hamper athletic performance, suggests a report in the March 20 *Journal of the American Medical Association.*

High school cross-country runners at San Marino, California, a Los Angeles suburb, performed poorest at track meets when oxidant air pollution was greatest during the six-year period, 1959-1964.

The Los Angeles area's oxidizing type of air pollution is caused by photochemical reactions with motor fuel. It isn't known, the *Journal* report said, whether this type of smog had a direct physiological effect on the runners, or whether it simply decreased their motivation by causing chest discomfort, a frequent complaint in smog areas.

The report, by researchers from the U.S. Public Health Service and the University of Southern California School of Medicine, also stressed that this study applied only to Los Angeles' oxidizing type of air pollution.

The study started with records of runners' performance kept by San Marino track coach Jack Bradford. Long-distance runners ordinarily would be expected to perform better as the season progresses. According to Coach Bradford's records, however, many runners failed to improve their racing times, or did not improve as much as expected during certain track meets.

When the dates of these meets were compared with records of the Los Angeles County Air Pollution Control District, it was found that poorest running performance coincided with periods of greatest oxidizing air pollution near the high school track.

Other factors, such as temperature, relative humidity, wind velocity and wind direction showed no correlation to runners' performance. Correlations were less striking or absent with other measures of air pollution, such as concentrations of air-suspended particles, carbon monoxide, or oxides of nitrogen.

Because the runners were at sea level, where oxygen is plentiful, it is unlikely that performance was decreased by oxygen lack, the report said.

"Oxidizing pollution is definitely irritating to the eyes, and athletes often complain of chest discomfort after exercising in the Los Angeles area on days with high pollution," the report said.

Slower running thus may be due to discomfort, rather than to the direct physiologic effects of air pollution, the report said.

The authors are Walborg S. Wayne, M.S., and Robert E. Carroll, M.D., M.P.H., of the National Center for Air Pollution Control, U.S. Public Health Service, Cincinnati, Ohio, and Paul F. Wehrle, M.D., of the University of Southern California School of Medicine and the Los Angeles County General Hospital, Los Angeles.

Could it be something more than growing old that "gets her down"?

For people who eat more than one meal a day

A PLEGINE (Phendimetrazine bitartrate) tablet taken an hour before each meal affords peak activity at mealtime, when it is needed most. Reminds patients to eat according to your prescribed diet plan.

t.i.d./Plegine®

BRAND OF
Phendimetrazine bitartrate

provides peak appetite suppression when it's needed most

Indications: Recommended in the management of excessive appetite leading to obesity.

Cautions and Contraindications: No adverse effects on blood pressure, heart rate, and respiration have been reported. However, as is true for all medications of this type, PLEGINE (Phendimetrazine bitartrate) is not recommended for patients with coronary disease, severe hypertension, or thyrotoxicosis, and should be used with caution in highly nervous or agitated individuals.

Side Effects: There have been occasional reports of insomnia and nervousness. Rare instances of mouth dryness, nausea, blurring of vision, dizziness, constipation, and stomach pain have been noted.

AYERST LABORATORIES New York, N.Y. 10017 · Montreal, Canada

6706

OFFICERS

OF THE CALIFORNIA MEDICAL ASSOCIATION

(Continued from Page 5)

Committee on Health Facilities Planning

Laurence A. Mosier, Garden Grove	1967
John W. Gates, Visalia	1967
Joseph W. Telford, San Diego	1967
Jean F. Crum, Downey	1968
Glenn A. Pope, Sacramento (Chmn.)	1968
Richard Taw, Los Angeles	1968
James W. Dalton, Santa Barbara	1969
Paul C. Doehring, Glendale	1969
Pierre Salmon, San Mateo	1969

CONSULTANTS: Robert F. Schell, San Rafael; Jack Lomas, Beverly Hills.

COUNCIL COMMITTEES

Finance Committee

Harold Kay, Oakland (Chmn.); Roger C. Isenhour, San Diego; Frank C. Melone, Ontario; Joseph W. Telford, San Diego; Malcolm C. Todd, Long Beach.

Committee on Legislation

Dan O. Kilroy, Sacramento (Chmn.); Stuart C. Knox, Los Angeles; Harold E. Wilkins, Downey; John Rumsey, San Diego; Samuel R. Sherman, San Francisco.

Advisory Board to Woman's Auxiliary

James C. MacLaggan, San Diego (Chmn.); John G. Morrison, San Leandro; William F. Quinn, Los Angeles; Carl E. Anderson, Santa Rosa; Eugene Hoffman, Los Angeles.

Benevolence Fund Operating Committee

Clyde L. Boice, Palo Alto (Chmn.); Alexander Fraser, San Francisco; Elizabeth Mason Hohl, Los Angeles; Don C. Musser, San Francisco; George Wolf, Fresno; Dudley M. Cobb, Jr., Los Angeles.

Easy on the Budget...
Easy on the G.I. Tract

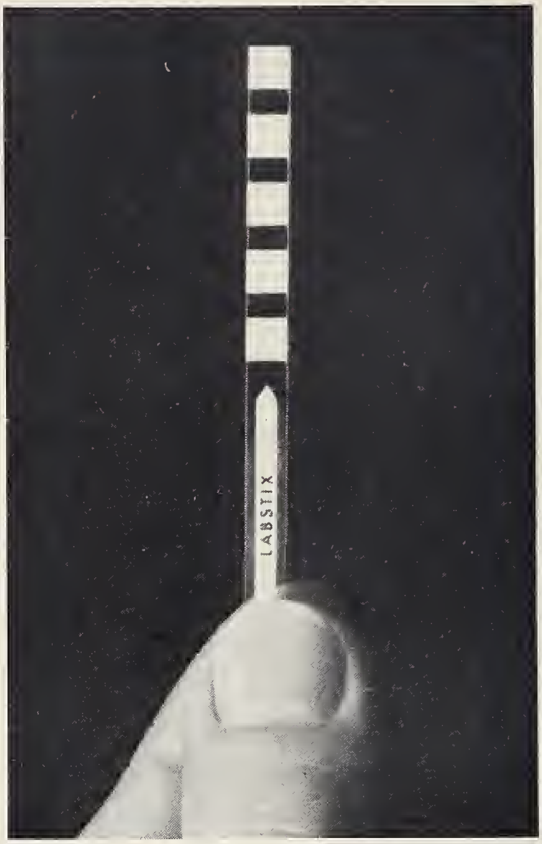
Tablets & Elixir

For Iron Deficiency Anemia

FAMOUS
Fergon®
brand of FERROUS GLUCONATE

C.M.A.P. CODE # 5261

BREON
BREON LABORATORIES INC.
Subsidiary of Sterling Drug Inc.
90 Park Avenue, New York, N.Y. 10016



Take five...

LABSTIX® provides 5 important urinary findings*—on a single reagent strip! That's *more* information than you can get from any other single reagent strip. You know the results in just 30 seconds—while the patient is still in your office—and readings are reliable and reproducible. LABSTIX is easy to handle, too. Never goes limp, even when wet, because it's made with clear, firm plastic. And results with LABSTIX are easy to read—color contrast between the test areas and the transparent plastic is clearly defined. An unexpected "positive" from testing with LABSTIX may help in detecting hidden pathology before marked symptoms are manifest.

*Blood; ketones; glucose; protein, and pH.

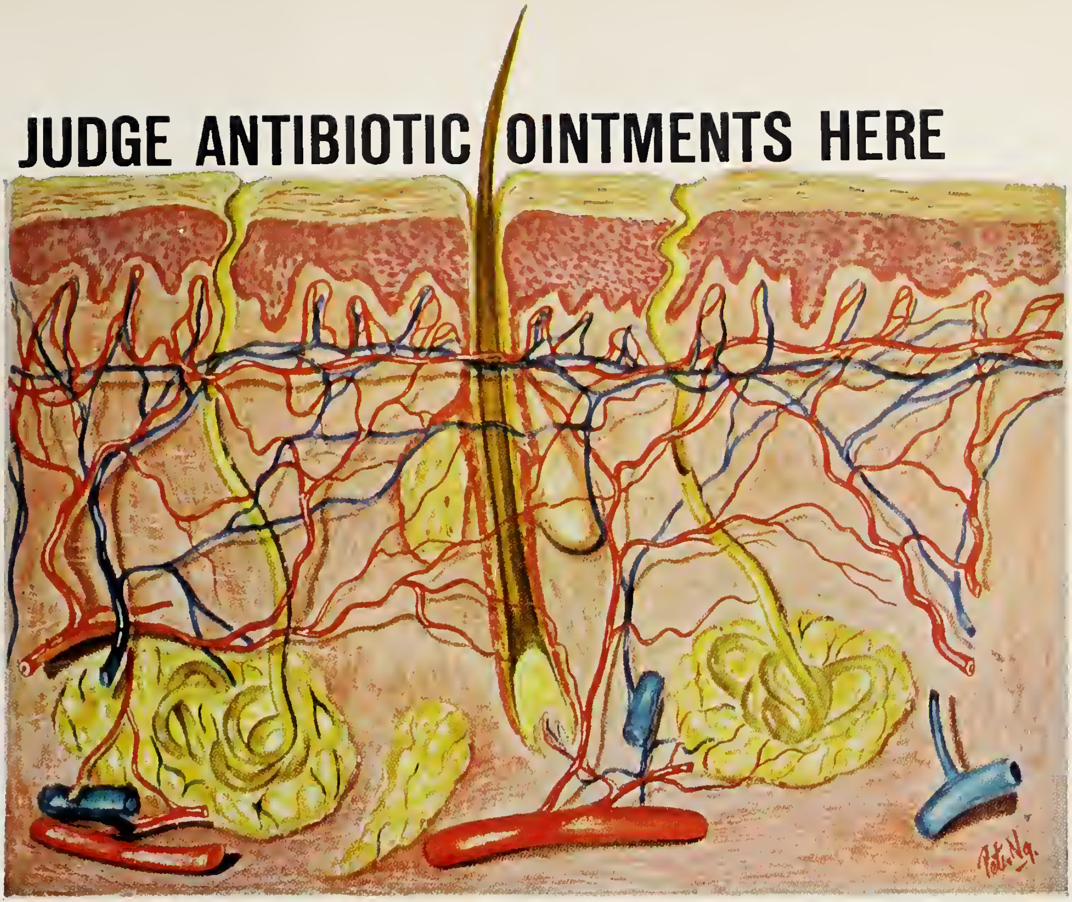
AMES COMPANY
Division Miles Laboratories, Inc.
Elkhart, Indiana 46514

Ames

Note: *AMERICAN HOSPITAL FORMULARY SERVICE CATEGORY NUMBER 36:88*

40167

JUDGE ANTIBIOTIC OINTMENTS HERE



Results on skin are final proof of any topical antibiotic's effectiveness

No in vitro test can duplicate a clinical situation on living skin. 'Neosporin' (polymyxin B—bacitracin—neomycin) Ointment has consistently proven its effectiveness in thousands of cases of bacterial skin infection. The spectra of the three antibiotics overlap in such a way as to provide bactericidal action against most pathogenic bacteria likely to be found topically. Diffusion of the antibiotics from the special petrolatum base is rapid since they are insoluble in the petrolatum, but readily soluble in tissue fluids. The Ointment is bland and nonirritating.

Caution: As with other antibiotic preparations, prolonged use may result in overgrowth of nonsusceptible organisms and/or fungi. Appropriate measures should be taken if this occurs. Articles in the current medical literature indicate an increase in the prevalence of persons allergic to neomycin. The possibility of such a reaction should be borne in mind.
Contraindications: This product is contraindicated in those individuals who have shown hypersensitivity to any of its components.
Supplied: Tubes of 1 oz., ½ oz. with applicator tip, and ⅛ oz. with ophthalmic tip.
Complete literature available on request from Professional Services Dept. PML.

'NEOSPORIN'®
brand

POLYMYXIN B-BACITRACIN-NEOMYCIN
OINTMENT

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N.Y.

Officers OF THE CALIFORNIA MEDICAL ASSOCIATION

General Officers

President: John G. Morrison, 333 Estudillo, San Leandro 94577. 415 483-1332

President-Elect: Malcolm C. Todd, 2865 Atlantic Avenue, Long Beach 90806. 213 426-1355

Past President: James C. MacLaggan, 525 Hawthorn Street, San Diego 92101. 714 233-6519

Speaker of the House of Delegates: William F. Quinn, 1401 South Hope Street, Los Angeles 90015. 213 749-0181

Vice-Speaker of the House of Delegates: Joseph F. Boyle, 1136 West Sixth Street, Los Angeles 90017. 213 482-9891

Chairman of the Council: Albert G. Miller, 77 San Mateo Drive, San Mateo 94401. 415 343-9074

Vice-Chairman of the Council: Harold Kay, 411 Thirtieth Street, Oakland 94609. 415 832-4822

Secretary: Helen B. Weyrauch, 450 Sutter Street, San Francisco 94108. 415 392-6909

Editor: Dwight L. Wilbur, 655 Sutter Street, San Francisco 94102. 415 673-4080

Executive Director: Howard Hassard, 693 Sutter Street, San Francisco 94102. 415 776-9400

General Counsel: Peart, Hassard, Smith and Bonnington, 44 Montgomery Street, San Francisco 94104. 415 982-8585

* * *

District Councilors

First District—Stanley A. Moore (1970), 2466 First Avenue, San Diego 92101. Imperial and San Diego Counties. 714 234-7341

Second District—Frank C. Melone (1968), 527 N. Palm Avenue, Ontario 91761. Inyo, Mono, Riverside and San Bernardino Counties. 714 986-1104.

Third District—Henry V. Eastman (1970), 661 W. First Street, Tustin 92680. Orange County. 714 544-4560

Fourth District—Los Angeles County—

- Office No. 1—Vacant
- Office No. 2—Elmer F. Gooel (1969), 465 N. Roxbury Drive, Beverly Hills 90210. 213 271-5745
- Office No. 3—Homer C. Pheasant (1970), 2300 S. Hope Street, Los Angeles 90007. 213 747-0301
- Office No. 4—Lewis T. Bullock (1968), 3875 Wilshire Boulevard, Los Angeles 90005. 213 382-2209
- Office No. 5—Joseph P. O'Connor (1969), 595 E. Colorado Boulevard, Pasadena 91101. 213 792-2154
- Office No. 6—Marvin J. Shapiro (1970), 16237 Ventura Boulevard, Encino 91316. 213 STate 8-0280
- Office No. 7—Wilbur G. Rogers (1968), 1122 N. Brand Boulevard, Glendale 91202. 213 241-3125
- Office No. 8—Jean F. Crum (1969), 8355 E. Third Street, Downey 90241. 213 869-1091
- Office No. 9—Vacant

Fifth District—Joseph F. Maguire (1970), 2755 Loma Vista Road, Ventura 93003. San Luis Obispo, Santa Barbara and Ventura Counties. 805 648-2567

Sixth District—Ralph W. Burnett (1969), 515 Truxton Avenue, Bakersfield 93301. Amador, Calaveras, Fresno, Kern, Kings, Madera, Mariposa, Merced, San Joaquin, Stanislaus, Tulare and Tuolumne Counties. 805 327-4411

Seventh District—Monterey, San Benito, San Mateo, Santa Clara and Santa Cruz Counties.

- Office No.1—Richard S. Wilbur (1970), 300 Homer Avenue, Palo Alto 94301. 415 321-4121
- Office No. 2—Albert G. Miller (1968), 77 San Mateo Drive, San Mateo 94401. 415 343-9074

Eighth District—San Francisco County—

- Office No. 1—Malcolm S. M. Watts (1969), 909 Hyde Street, San Francisco 94109. 415 776-3838
- Office No. 2—Roberta F. Fenlon (1970), 490 Post Street, San Francisco 94102. 415 986-1391

Ninth District—Alameda and Contra Costa Counties—

- Office No. 1—Harold Kay (1969), 411 Thirtieth Street, Oakland 94609. 415 832-4822
- Office No. 2—William F. Kaiser (1970), 3000 Colby Street, Berkeley 94705. 415 843-6515

Tenth District—E. Kash Rose (1970), P. O. Box 2038, Napa 94558. Del Norte, Humboldt, Lake, Marin, Mendocino, Napa, Solano and Sonoma Counties. 707 224-7811

Eleventh District—James H. Yant (1968), 2600 Capitol Avenue, Sacramento 95816. Alpine, Butte, Colusa, El Dorado, Glenn, Lassen, Modoc, Nevada, Placer, Plumas Sacramento, Shasta, Sierra, Siskiyou, Sutter, Tehama, Trinity, Yolo and Yuba Counties. 916 446-5278

Twelfth District—Forest J. Grunigen (1967), 3919 Beverly Boulevard, Los Angeles 90004

Scientific Board Refresentative—William P. Longmire, Jr. (1970), UCLA Department of Surgery, Los Angeles 90024. 213 272-8911

* * *

COMMISSION ON *Medical Services*

George Babbin, Sacramento
Ralph Burnett, Bakersfield
Elmer F. Gooel, Beverly Hills (Secty.)
Arthur F. Howard, Fresno (Chmn.)
Ben D. A. Miano, San Bernardino
William H. Thompson, San Mateo

Committee on Fees

Roger C. Isenhour, San Diego	1968
Alfred J. Murrieta, Jr., Los Angeles	1968
Maurice S. Salomon, San Francisco	1968
William H. Thompson, San Mateo (Chmn.)	1968
Homer C. Pheasant, Los Angeles	1969
Glenn A. Pope, Sacramento	1969
Leo Bell, San Mateo	1969
Joseph F. Boyle, Los Angeles	1969
Charles Anderson, Oakland	1969
Gerson R. Biskind, San Francisco	1970
Wallace A. Gerrie, Jr., Newport Beach	1970
H. Dean Hoskins, Oakland	1970
Robert L. Watson, Jr., Los Angeles	1970

CONSULTANT: Nelson S. Keeler, Oakland.

Committee on Federal Medical Care Programs

Arthur F. Howard, Fresno (Chmn.)	1968
John S. McKee, Jr., San Diego	1968
Charles B. Canby, Van Nuys	1970
Merlin A. Hendrickson, Rialto	1970
Thomas A. O'Sullivan, Tustin	1970

Committee on Veterans Affairs

Charles B. Hudson, Oakland
Ben D. A. Miano, San Bernardino (Chmn.)
Robert L. Watson, Jr., Los Angeles

CONSULTANT: Lloyd E. Gould, Napa.

Committee on Mediation

Richard L. Taw, Los Angeles	1968
George Babbin, Sacramento (Chmn.)	1969
Albert G. Clark, San Francisco	1969
Eli A. Layon, Brawley	1970
James Picklesimer, Whittier	1970
Alfred V. Bateman, Escondido	1970

Committee on Insurance and Prepayment

Ralph W. Burnett, Bakersfield (Chmn.)	1968
Stanley J. Murasky, Reseda	1968
Ivan Neubauer, San Jose	1969
Thayer A. Smith, Norwalk	1969
Oscar W. Hills, San Mateo	1970
Dave F. Dozier, Sacramento	1970

* * *

COMMISSION ON *Public Agencies*

Lewis T. Bullock, Los Angeles
Joseph P. Cosentino, Sacramento (Secty.)
William F. Kaiser, Berkeley
Stuart C. Knox, Los Angeles
Howard W. Lindsey, Jr., San Mateo
Albert G. Miller, San Mateo (Chmn.)
Joseph P. O'Connor, Pasadena
Stanley A. Skillicorn, San Jose
Howell E. Wiggins, San Diego

CONSULTANT: Carl E. Anderson, Santa Rosa.

SUBCOMMITTEE ON LONG-TERM CARE FACILITIES: William Kaiser, Berkeley (Chmn.); Bernard J. Michela, Long Beach; Albert G. Miller, San Mateo; Pierre Salmon, San Mateo; Stanley A. Skillicorn, San Jose; John T. McLaughlin, Glendale.

Committee on Public Health

Albert G. Miller, San Mateo	1968
Stanley A. Skillicorn, San Jose (Chmn.)	1968
James Malcolm, Oakland	1968
Alfred F. Kandlbinder, Monterey	1969
Samuel S. Woolington, Long Beach	1969
Robert L. Watson, Jr., Los Angeles	1969
Robert L. Garrett, Vallejo	1970
Karl L. Schaupp, Jr., San Francisco	1970
Allen F. Sterling, San Bernardino	1970

Committee on Mental Health

Alfred Auerback, San Francisco	1968
David H. Payne, Los Angeles	1968
Jack B. Lomas, Beverly Hills	1968
Joseph J. Furlong, San Francisco	1969
Stuart C. Knox, Los Angeles (Chmn.)	1969
Ira H. Degenhardt, San Rafael	1970
Emmanuel Silver, Palo Alto	1970
John A. Stroud, III, Sacramento	1970
Rudolph B. Toller, Stockton	1970

Committee on Welfare Medical Care Programs

Orrin S. Cook, Jr., Sacramento	1968
Robert F. Schell, Chico	1968
William F. Quinn, Los Angeles	1969
James Dalton, Santa Barbara	1969
Arthur F. Howard, Fresno	1969
Howard W. Lindsey, Jr., San Mateo (Chmn.)	1969
Clyde L. Boice, Palo Alto	1970
Vincent P. Carroll, Laguna Beach	1970
Thomas N. Elmendorf, Willows	1970
Philip L. Pillsbury, San Francisco	1970

CONSULTANT: Edgar Rosen, Sacramento.

SUBCOMMITTEE ON ADOPTIONS: Walter Beckh, San Francisco; Fred M. Kay, Fullerton; Robert J. McNeil, Los Angeles (Chmn); Donald Minkler, Berkeley; Dale W. Ritter, Chico; Ben J. Siebenthal, Sacramento.

Committee on Occupational Health and Rehabilitation

Edward J. Zaik, Los Angeles	1968
Packard Thurber, Jr., Los Angeles	1968
Thomas Gucker, III, Los Angeles	1968
Herbert A. Lints, Oakland	1969
Patrick J. Maloney, Merced	1969
Bernard J. Michela, Long Beach	1969
Herbert A. Holden, San Leandro	1970
John H. Leimbach, Jr., San Francisco	1970
Howell E. Wiggins, San Diego (Chmn.)	1970

* * *

COMMISSION ON *Community Health Services*

Wayne P. Chesbro, Berkeley
Charles G. Hutter, Jr., Hollywood
Gerald G. Jampolsky, Tiburon (Secty.)
Harold Kay, Oakland (Chmn.)
John R. Lee, Mill Valley
Thomas Lyons, La Mesa
Pierre Salmon, San Mateo
Leopold J. Snyder, Fresno
James H. Yant, Sacramento

Committee on Rural Health

Leopold J. Snyder, Fresno (Chmn.)	1968
Erwin P. Brauner, Visalia	1968
Carrol W. Goss, La Mont	1968
Franklin L. Murphy, Oroville	1969
Thomas Rossitto, Auburn	1969
M. Marlin Clark, Delano	1969
Mitry P. Ajalat, Calexico	1970
Richard W. Johnson, Arbuckle	1970

Committee on School and College Health

Glenn E. Austin, Los Altos	1968
Ruth A. Frary, Watsonville	1968
Frederick A. Frye, San Diego	1968
Tom W. Robinson, Newport Beach	1968
Henry Bruyn, Berkeley	1969
Elmer F. Gooel, Beverly Hills	1969
B. Otis Cobb, Martinez	1970
Byron H. Demorest, Sacramento	1970
Gerald G. Jampolsky, Tiburon (Chmn.)	1970

Committee on Health Care of the Aging

Charles E. Schoff, Jr., Sacramento	1968
Raymond D. Manchester, Visalia	1968
Leo A. Wagner, Novato	1969
Lloyd E. Gould, Napa	1969
Walter Batchelder, San Mateo	1969
Pierre Salmon, San Mateo (Chmn.)	1970
Floyd L. Weregeland, Laguna Hills	1970

Committee on Disaster Medical Care

Wayne P. Chesbro, Berkeley (Chmn.)	1968
Michael J. Hitchko, Eureka	1968
John Heald, San Francisco	1969
J. T. McNally, Stockton	1969
Donald E. Barker, Van Nuys	1970
Charles C. Henderson, San Mateo	1970

Committee on Automotive and Traffic Safety

Arnold R. Friesen, Pasadena	1968
Laurance A. Mosier, Garden Grove	1968
Thomas W. Lyons, La Mesa (Chmn.)	1969
Joseph F. Maguire, Ventura	1969
Robert Tour, San Francisco	1969
George C. Snively, Sacramento	1970
B. G. Stewart, Bakersfield	1970

CONSULTANTS: Julian A. Waller, Berkeley; Julian M. Sether, Los Angeles.

Committee on Medical Aspects of Sports and Physical Fitness

George S. Buehler, Whittier	1968
Philip H. McFarland, Fullerton	1968
Tom W. Robinson, Newport Beach	1968
Charles G. Hutter, Jr., Hollywood (Chmn.)	1969
Arnold A. Nutting, San Rafael	1969
John F. Blinn, Stockton	1970
Robert K. Kerlan, Los Angeles	1970

Committee on Environmental Health

Walter P. Ellerbeck, Los Angeles	1968
Reginald H. Smart, Los Angeles	1968
Grace M. Talbott, San Francisco	1968
Douglas Campbell, San Francisco	1969
John R. Lee, Mill Valley (Chmn.)	1969
Robert L. Hippen, San Diego	1970
George A. Martin, Redding	1970

Committee on Blood Banks

Elmer Jennings, Long Beach	1968
Claudius Y. Gates, San Francisco	1968
Harlan F. Fulmer, Fresno	1968
Osman H. Hull, Monterey	1969
James H. Yant, Sacramento (Chmn.)	1970

CONSULTANT: Melvin L. Sommer, Beverly Hills.

* * *

COMMISSION ON *Communications*

Frederick Ackerman, Pleasant Hill
L. Morgan Boyers, Sacramento (Secty.)
Roberta F. Fenlon, San Francisco
Elmer F. Gooel, Beverly Hills
Robert Hood, Jr., Van Nuys
Walter W. Hopps, Los Angeles
James C. MacLaggan, San Diego
George Schwartz, Beverly Hills
Richard S. Wilbur, Palo Alto
Leonard M. Asher, Beverly Hills

EX-OFFICIO: John G. Morrison, San Leandro; Malcolm C. Todd, Long Beach; Albert G. Miller, San Mateo.

* * *

Scientific Board

Edmund L. Keeney, La Jolla	1968
Seymour J. Brockman, Los Angeles	1968
S. Austin Jones, Los Angeles	1968
Hobart M. Kelly, Riverside	1968
Saul J. Robinson, San Francisco	1968
Eugene Ziskind, Los Angeles	1968
William P. Longmire, Jr., Los Angeles (Chmn.)	1968
R. Theodore Bergman, Los Angeles	1968
John B. deC. M. Saunders, San Francisco	1968
Herbert A. Holden, San Leandro	1968
David A. Wood, San Francisco	1968
Arthur A. Clinco, Los Angeles	1968
Bert L. Halter, San Francisco	1969
Leland B. Blanchard, San Jose	1969
Norman E. Levan, Bakersfield	1969
L. S. Goerke, Los Angeles	1969
Ellis Jones, Pasadena	1969
Carl M. McCandless, San Francisco	1969
J. Blair Pace, Oceanside	1969
Donald W. Petit, Los Angeles	1969
Glenn A. Pope, Sacramento	1969
Robert J. Prentiss, San Diego	1969
Edward B. Shaw, San Francisco	1969
Ralph E. Worden, Los Angeles	1969
Henry D. Brainerd, San Francisco	1970
Charles J. Tupper, Davis	1970
Jerome W. Bettman, San Francisco	1970
Vernon L. Nickel, Los Angeles	1970
John B. Dillon, Los Angeles	1970
Justin J. Stein, Los Angeles	1970
Charles E. Grayson, Sacramento	1970
Victor Richards, San Francisco	1970
David Rubin, Los Angeles	1970
Keith P. Russell, Los Angeles	1970
Edward J. Zaik, Los Angeles	1970
William W. Belford, San Diego	1970

COUNCIL REPRESENTATIVES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.

EX-OFFICIO: Dwight L. Wilbur, San Francisco.

Executive Committee

William P. Longmire, Jr., Los Angeles (Chmn.)	1968
John B. Dillon, Los Angeles	1969
Donald W. Petit, Los Angeles	1969
Edward B. Shaw, San Francisco	1969
Justin J. Stein, Los Angeles	1970
Charles J. Tupper, Davis	1970
Keith P. Russell, Los Angeles	1970

COUNCIL REPRESENTATIVES: Lewis T. Bullock, Los Angeles; Malcolm S. M. Watts, San Francisco.

Committee on Nominations

Robert J. Prentiss, San Diego	1969
Edward B. Shaw, San Francisco (Chmn.)	1969
R. Theodore Bergman, Los Angeles	1970

Committee on Scientific Assemblies

John B. deC. M. Saunders, San Francisco	1968
Arthur A. Clinco, Los Angeles	1968
Ellis Jones, Pasadena	1969
Donald W. Petit, Los Angeles	1969
Carl M. McCandless, San Francisco	1969
John B. Dillon, Los Angeles (Chmn.)	1970
Victor Richards, San Francisco	1970
Henry D. Brainerd, San Francisco	1970
Vernon L. Nickel, Los Angeles	1970

CONSULTANT: Claron Oakley, Los Angeles.

Committee on Continuing Medical Education

Saul J. Robinson, San Francisco	1968
Donald W. Petit, Los Angeles (Chmn.)	1969
Bert L. Halter, San Francisco	1969
J. Blair Pace, Oceanside	1969
John B. Dillon, Los Angeles	1970
Charles E. Grayson, Sacramento	1970
Charles J. Tupper, Davis	1970

Area Representatives

Thomas J. Fuson, Fresno
Kenneth M. Joye, Auburn
Alfred F. Kandlbinder, Monterey
Robert H. Quillinan, Sacramento
Robert S. Quinn, Santa Rosa
Edward Shanbrom, Santa Ana
Raymond F. Tatro, San Bernardino
J. Roy Wittwer, Eureka
George J. Wittenstein, Santa Barbara

Committee on CALIFORNIA MEDICINE

William W. Belford, San Diego	1968
Herbert A. Holden, San Leandro	1968
Edward B. Shaw, San Francisco	1969
Charles J. Tupper, Davis	1969
David Rubin, Los Angeles (Chmn.)	1970

Committee on Maternal and Child Care

Charles M. Blumenfeld, Sacramento	1968
Herbert A. Holden, San Leandro	1968
Hobart M. Kelly, Riverside	1968
Leland B. Blanchard, San Jose	1969
L. S. Goerke, Los Angeles	1969
Keith P. Russell, Los Angeles (Chmn.)	1970
Richard Leonards, San Francisco	1970
Russell W. Mapes, Beverly Hills	1970
Eugene Sandberg, Palo Alto	1970

Committee on Cancer

John G. Walsh, Sacramento	1968
Robert W. Jamplis, Palo Alto	1968
David A. Wood, San Francisco	1968
Lewis W. Guiss, Los Angeles	1969
Robert J. Prentiss, San Diego	1969
Victor Richards, San Francisco	1969
Charles E. Gravson, Sacramento	1970
Jean F. Crum, Downey (Chmn.)	1970
James Helsper, Pasadena	1970

Committee on Adverse Drug Reactions

John F. Murray, Fresno (Chmn.)	1968
Russell J. Tat, San Francisco	1969
William W. Thayer, Stockton	1969
Edward R. Bloomquist, Glendale	1969

CONSULTANTS: John W. Brown, Berkeley; Ralph W. Weilerstein, San Francisco; Kenneth Melman, San Francisco; Ronald Okun, Los Angeles.

SUBCOMMITTEE ON DANGEROUS DRUGS: Edward R. Bloomquist, Glendale; Norman A. Gale, San Diego; Edward T. Jewett, Santa Rosa; William F. Quinn, Los Angeles (Chmn.) Helen B. Weyrauch, San Francisco.

CONSULTANT: Ralph W. Weilerstein, Berkeley.

Committee on Cardiovascular Diseases

Harold I. Griffeath, San Francisco (Chmn.); Lewis T. Bullock, Los Angeles; George Griffith, Los Angeles; David J. New, Modesto.

CONSULTANT: John Connolly, Los Angeles.

* * *

BUREAU OF *Research and Planning*

J. Brandon Bassett, Oakland
G. Robert Ellison, Pomona
H. Russell Fisher, Glendale
John Sheehy, Riverside
Carl E. Anderson, Santa Rosa (Chmn.)

EX-OFFICIO: Malcolm S. M. Watts, San Francisco; Jean F. Crum, Downey.

* * *

COMMISSION ON *Professional Welfare*

Bert L. Halter, San Francisco
George K. Herzog, Jr., San Francisco (Chmn.)
Francis E. West, San Diego
James H. Yant, Sacramento (Secty.)

Committee on Physicians Group Insurance

Carl Goetsch, Berkeley	1968
George K. Herzog, Jr., San Francisco (Chmn.)	1968
John Hogan, San Diego	1969
Homer C. Pheasant, Los Angeles	1969
Robert X. DeMangus, Glendale	1970
John B. Long, Sacramento	1970

Liaison Committee to Medical Schools

Stanley Cowell, Los Angeles	1968
Frank J. Novak, Menlo Park	1968
Bert L. Halter, San Francisco (Chmn.)	1969
Alonzo Neufeld, Los Angeles	1969
Herbert Jenkins, Sacramento	1969
Samuel R. Irvine, Beverly Hills	1970
Francis E. West, San Diego	1970

EX-OFFICIO: John G. Morrison, San Leandro; Malcolm C. Todd, Long Beach; Albert G. Miller, San Mateo.

Medical Review and Advisory Committee

John B. Dillon, Los Angeles	1968
Carl Goetsch, Berkeley	1968
Donald N. Donisthorpe, Los Angeles	1969
Eugene M. Webb, San Francisco	1970
James H. Yant, Sacramento (Chmn.)	1970

CONSULTANT: Don Harper Mills, Long Beach.

Liaison Committee to State Bar of California

Carl Goetsch, Berkeley	1968
Francis E. West, San Diego (Chmn.)	1968
Henry A. Brown, San Mateo	1969
Robert J. McNeil, Los Angeles	1969
David Rubsamen, Berkeley	1970
Marvin J. Shapiro, Encino	1970

* * *

COMMISSION ON *Allied Health Professions and Services*

Roger W. Barnes, Los Angeles (Secty.)
Forest J. Grunigen, Los Angeles
Frank C. Melone, Ontario (Chmn.)
Thomas W. Ledwich, Napa
C. Gerald Scarborough, San Jose
Richard H. Schug, Long Beach

Committee on Paramedical Personnel

Gregory Bard, San Francisco	1968
Elmer Jennings, Long Beach	1968
Gerson R. Biskind, San Francisco	1969
Leonard M. Asher, Beverly Hills	1969
Joseph Schaefer, Santa Rosa	1969
Richard H. Schug, Long Beach	1969
Frank C. Melone, Ontario (Chmn.)	1970

Committee on Other Professions

William D. Evans, Los Angeles	1968
C. Gerald Scarborough, San Jose (Chmn.)	1968
J. Gordon Epperson, Oakland	1969
Lewis T. Bullock, Los Angeles	1969
Burt L. Davis, Palo Alto	1970
George S. Buehler, Whittier	1970

Liaison Committee to California Medical Assistants Association

Peary Benn Berger, Inglewood	1968
Michael F. Brodkin, Beverly Hills	1968
Henry Bodner, Van Nuys	1968
William F. Forestelle, Seaside	1968
Leland W. Bonar, Redding	1968
Sanford E. Feldman, San Francisco	1969
Richard H. Schug, Long Beach (Chmn.)	1970
Charlotte Baer, San Francisco	1970

Committee on Medicine and Religion

Max Nelson, National City	1968
Frank J. Glassy, Sacramento	1969
Richard J. Lescoe, Torrance	1969
Roger W. Barnes, Los Angeles (Chmn.)	1970
Albert E. Long, Daly City	1970

Committee on Voluntary Health Agencies

Thomas W. Ledwich, Napa (Chmn.)	1968
J. Brandon Bassett, Oakland	1969
Harney M. Cordua, Jr., San Diego	1970
H. Mason Hohl, Beverly Hills	1970

* * *

Judicial COMMISSION

Floyd O. Due, Oakland	1968
Sidney J. Shipman, San Francisco	1968
Francis E. West, San Diego (Chmn.)	1968
George C. Andersen, Hermosa Beach	1969
Douglas G. Campbell, San Francisco	1969
W. Philip Corr, Riverside (Secty.)	1969
Albert E. Long, Daly City	1970
Donald Lum, Alameda	1970
William F. Quinn, Los Angeles	1970

* * *

COMMISSION ON *Hospital Affairs*

William L. Argo, Fresno (Secty.)
James C. Doyle, Beverly Hills
Bert L. Halter, San Francisco
John C. Lungren, Long Beach
Glenn A. Pope, Sacramento
John T. Saidy, San Mateo
Joseph W. Telford, San Diego
Frederic N. Tyroler, Los Angeles
James C. MacLaggan, San Diego (Cbmn.)

Medical Staff Survey Committee

Robert M. Adams, San Leandro
Richard H. Anderson, Santa Cruz
Samuel T. D. Anderson, San Rafael
William L. Argo, Fresno
Myron Arrick, San Francisco
Ernest R. Barnett, Alhambra
John J. Bazzano, San Francisco
Daniel W. Black, Hayward
Rush M. Blodgett, Jr., Redding
Wayne O. Buck, San Jose
Robert Buckley, Santa Monica
Melvin A. Casberg, Long Beach
G. Cameron Cray, Palo Alto
Paul H. Deeb, Redlands
Fred H. Draper, San Jose

(Continued on Page 12)

Officers of Scientific Sessions

Members who contemplate presentation of scientific papers should promptly address the secretary of the proper section at the address shown below.

Allergy

Leo N. Meleyco.......Chairman
2040 Forest Avenue, San Jose 95128

Ernest J. Saslow.......Secretary
2011-18th Street, Bakersfield 93301

Samuel Ross.......Assistant Secretary
2946 Fresno Street, Fresno 93706

Anesthesiology

Thomas W. McIntosh.......Chairman
686 East Union St., Pasadena 91101

Donald B. Dose.......Secretary
5625 Linfield Ave., San Diego 92120

Donald A. Walker.......Assistant Secretary
35 Spring Lane, Tiburon 94920

Dermatology

Robert G. Walton.......Chairman
1700 McHenry Village Way, Modesto 95350

Samuel Ayres, III.......Secretary
405 N. Bedford Rd., Beverly Hills 90210

George T. Wilson.......Assistant Secretary
Welch and Pasteur Rd., Stanford Medical Plaza
Palo Alto 94304

General Practice

J. Blair Pace.......Chairman
408 Cassidy St., Oceanside 92054

Alfred F. Kandlbinder.......Secretary
302 Via Paraiso St., Monterey 93940

Allen F. Sterling.......Assistant Secretary
1285 D St., San Bernardino 92045

General Surgery

Seibert Pearson.......Chairman
2865 Atlantic Ave., Long Beach 90807

Albert G. Clark.......Secretary
450 Sutter St., San Francisco 94108

Robert T. Hood, Jr.......Assistant Secretary
2010 Wilshire Blvd., Los Angeles 90057

Industrial Medicine and Surgery

Leon R. Rudnick.......Chairman
15921 E. 14th St., San Leandro 94578

Raymond O. Oja.......Secretary
525 Golden Gate Ave., San Francisco 94102

James G. Forgerson.......Assistant Secretary
Metropolitan Life Insurance Company
600 Stockton St., San Francisco 94108

Internal Medicine

Robert L. Paver.......Chairman
384 Post St., San Francisco 94108

John L. Benton, Jr.......Secretary
1216 Wilshire Blvd., Los Angeles 90017

Edgar Rosen.......Assistant Secretary
1340 K St., Sacramento 95814

Obstetrics and Gynecology

Lester T. Hibbard.......Chairman
142 S. Windsor Blvd., Los Angeles 90004

Warren E. Jones.......Secretary
1380-45th Street, Sacramento 95819

James W. Ravenscroft.......Assistant Secretary
2330 First Ave., San Diego 92101

Ophthalmology

Victor G. Fellows.......Chairman
595 Buckingham Way, San Francisco 94117

Wendell C. Irvine.......Secretary
3875 Wilshire Blvd., Los Angeles 90005

Robert E. Hopkins.......Assistant Secretary
4255 Pacific Ave., Stockton 95207

Orthopedics

David C. Monsen.......Chairman
2300 S. Hope St., Los Angeles 90007

Arthur R. Hartwig.......Secretary
490 Post St., San Francisco 94102

Vernon L. Smythe.......Assistant Secretary
3875 Wilshire Blvd., Los Angeles 90005

Otolaryngology

G. Howard Gottschalk.......Chairman
8618 S. Sepulveda Blvd., Los Angeles 90045

Charles P. Lebo.......Secretary
490 Post St., San Francisco 94102

Irving L. White.......Assistant Secretary
911 San Antonio Dr., Long Beach 90807

Pathology

Frank R. Dutra.......Chairman
20103 Lake Chabot Rd., Castro Valley 94546

William H. Kern.......Secretary
679 S. Westlake Ave., Los Angeles 90057

Donald L. Bittner.......Assistant Secretary
Bay Area Laboratories, 2245 Post St.,
San Francisco 94115

Pediatrics

Richard L. Anderson.......Chairman
636 Harris St., Eureka 95501

Sherrod C. Swift.......Secretary
14619 Whittier Blvd., Whittier 90605

Robert J. Harvey.......Assistant Secretary
909 Hyde St., San Francisco 94109

Physical Medicine

Rene Cailliet.......Chairman
4900 Sunset Blvd., Los Angeles 90027

Anton A. Tratar.......Secretary
2360 Clay St., San Francisco 94115

Elizabeth Austin.......Assistant Secretary
Los Angeles County Hospital
1200 N. State St., Los Angeles 90033

Preventive Medicine

Henrik L. Blum.......Chairman
School of Public Health, U.C., Room 409, Earl Warren Hall, Berkeley 94720

Maxwell Rosenblatt.......Secretary
Los Angeles County Health Department,
220 N. Broadway, Los Angeles 90012

Russell B. Watson.......Assistant Secretary
Orange County Health Department, Santa Ana 92701

Psychiatry and Neurology

Werner M. Mendel.......Chairman
1934 Hospital Place, Los Angeles 90033

Ames Fischer.......Secretary
Langley Porter Inst., 401 Parnassus Ave.,
San Francisco 94122

Howard S. Barrows.......Assistant Secretary
2025 Zonal Ave., Los Angeles 90033

Radiology

Victor G. Mikity.......Chairman
1200 State St., Los Angeles 90033

Vernon R. Gee.......Secretary
2020 Court St., Redding 96001

Richard P. Storrs.......Assistant Secretary
2131 West 3rd St., Los Angeles 90057

Urology

Carl K. Pearlman.......Chairman
1518 N. Sycamore, Santa Ana 92701

James L. Goebel.......Secretary
51 Loma Linda, Ross 94957

Robert O. Pearman.......Assistant Secretary
5353 Balboa Blvd., Encino 91316

Delegates and Alternates

TO THE AMERICAN MEDICAL ASSOCIATION

DELEGATES		ALTERNATES
Francis E. West, San Diego	(1966-1967)	Vincent P. Carroll, Laguna Beach
Samuel R. Sherman, San Francisco	(1966-1967)	Albert G. Miller, San Mateo
Henry Gibbons, III, San Francisco	(1966-1967)	A. B. Sirbu, San Francisco
John M. Rumsey, San Diego	(1966-1967)	Carl E. Anderson, Santa Rosa
Eugene F. Hoffman, Los Angeles	(1966-1967)	Joseph F. Boyle, Los Angeles
Warren L. Bostick, Los Angeles	(1966-1967)	Walter H. Brignoli, St. Helena
J. B. Price, Santa Ana	(1966-1967)	Donald C. Dodds, Oakland
Ralph C. Teall, Sacramento	(1966-1967)	Robb Smith, Orange Cove
James C. Doyle, Beverly Hills	(1966-1967)	Herman H. Stone, Riverside
Wilbur G. Rogers, Glendale	(1966-1967)	Carl M. Hadley, San Bernardino
Charles B. Hudson, Oakland	(1966-1967)	Robert L. Watson, Jr., Los Angeles
John G. Morrison, San Leandro	(1967-1968)	Frederick Ackerman, Pleasant Hills
William F. Quinn, Los Angeles	(1967-1968)	Dudley M. Cobb, Los Angeles
Robert C. Combs, San Francisco	(1967-1968)	Emmett Rixford, San Francisco
Homer C. Pheasant, Los Angeles	(1967-1968)	James C. MacLaggan, San Diego
Alfred J. Murrieta, Los Angeles	(1967-1968)	Harold Kay, Oakland
Burt L. Davis, Palo Alto	(1967-1968)	Leon P. Fox, San Jose
Arlo A. Morrison, Ventura	(1967-1968)	Henry Brown, San Mateo
James E. Feldmayer, Exeter	(1967-1968)	Charles Grayson, Sacramento
O. W. Wheeler, Riverside	(1967-1968)	Wilbur Bailey, Los Angeles
Malcolm C. Todd, Long Beach	(1967-1968)	Neil C. Hamel, Encino
J. E. Vaughan, Bakersfield	(1967-1968)	Joseph P. O'Connor, Pasadena
Carl E. Anderson, Santa Rosa	(1967-1968)	Jean F. Crum, Downey

(Delegates and Alternates to the AMA are elected for terms of two calendar years. Each office is scheduled for election at the Annual Session of the second year shown above.)

Other Organizations and Medical Schools

Board of Medical Examiners of the State of California

Secretary.......Shelby M. Hicks, M.D.
1021 O Street, Room A202, Sacramento 95814
San Francisco.......30 Van Ness Ave., 94102
Room 2100
Los Angeles.......107 South Broadway 90012
Sacramento.......1021 O Street, Room A547 95814

The Public Health League of California

Executive Secretary.......Ben H. Read
955 South Western Ave., Los Angeles 90006
Assistant Executive Secretary.......Al Paul Putnam
693 Sutter Street, San Francisco 94102
San Francisco.......693 Sutter Street 94102
415 474-1061
Los Angeles.......955 South Western Ave. 90006
213 731-6397
Sacramento.......1127-11th Street, 95814
916 444-7496

Department of Public Health of the State of California

Director.......Lester Breslow, M.D.
2151 Berkeley Way, Berkeley 94704
Berkeley.......2151 Berkeley Way 94704
415 843-7900
Sacramento.......1927-13th Street 95814
916 445-2264
Los Angeles.......417 South Hill Street 90013
213 620-2900

Medical Schools in California

University of California School of Medicine, Medical Center, San Francisco 94122. Dean: Stuart C. Cullen, M.D.

Stanford University School of Medicine, 300 Pasteur Drive, Palo Alto 94304. Dean: Robert J. Glaser, M.D.

University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 90033. Dean: Roger O. Egeberg, M.D.

Loma Linda University School of Medicine, Loma Linda 92354. Dean: David Burdg Hinshaw, M.D.

University of California at Los Angeles, School of Medicine, Hilgard Avenue, Los Angeles 90024. Dean: Sherman M. Mellinkoff, M.D.

University of California, California College of Medicine, 1721 Griffin Avenue, Los Angeles 90031. Dean: Warren L. Bostick, M.D.

University of California, San Diego, School of Medicine, La Jolla 92038. Acting Dean: Robert D. Tschirgi, M.D.

University of California, Davis, School of Medicine, Davis 95616. Dean: Charles John Tupper, M.D.

California Medical Education and Research Foundation

Directors: Ralph C. Teall, M.D., President; Carl E. Anderson, M.D., Vice-President; Howard Hassard, Secretary; James C. MacLaggan, M.D.; Harold Kay, M.D.; William F. Quinn, M.D.; E. B. Shaw, M.D.

Roster — CALIFORNIA COMPONENT MEDICAL SOCIETIES

Society secretaries are requested to notify California Medicine promptly when changes are indicated in their roster information

ALAMEDA-CONTRA COSTA Medical Assn., 6230 Claremont Ave., Oakland 94618. Meets quarterly.
Frederick W. Ackerman..........President
2100 Monument Blvd., Pleasant Hill 93542
Herbert A. Holden..........Secretary
1436 Clarke St., San Leandro 94577

BUTTE-GLENN Medical Society, P. O. Box 1008, Chico 95927. Meets Fourth Thursday.
Walter S. Mazen..........President
1617 Esplanade, Chico 95926
John B. Copeland..........Secretary
1423 Magnolia, Chico 95926

FORTY FIRST Medical Society, Suite 821, 1515 N. Vermont Ave., Los Angeles 90027
Paul D. Yates..........President
844 Hermosa Ave., Hermosa Beach 90254
Jordan M. Phillips..........Secretary
11239 S. Lakewood Blvd., Downey 90241

FRESNO County Medical Society, 1118 Divisadero St., Fresno 93721. Meets Second Tuesday, 6:30 p.m., California Hotel.
Theodore Steinberg..........President
155 N. Thesta St., Fresno 93701
Jack Schiff..........Secretary
167 N. Thesta St., Fresno 93701

HUMBOLDT-DEL NORTE County Medical Society. Meets Second Thursday.
Halvor J. Braafladt..........President
3115 G St., Eureka 95501
Max Todd..........Secretary
616-14th St., Arcata 95521

IMPERIAL County Medical Society, P. O. Box 518, Imperial 92251. Meets Second Tuesday, 8 p.m., Pioneer Memorial Hospital, Brawley.
O. G. Fais..........President
1745 S. Imperial Ave., El Centro 92243
Elmer C. Werner..........Secretary
1745 S. Imperial Ave., El Centro 92243

INYO-MONO County Medical Society. Meets Fourth Tuesday except December, January, February.
Milton R. Jones, Jr..........President
Box 38, Lone Pine 93545
Robert W. Denton..........Secretary
611 W. Line St., Bishop 93514

KERN County Medical Society, 2603 G Street, Bakersfield 93301. Meets Third Tuesday, 7:30 p.m., Society Office, 2603 G Street, except June, July, August.
Keith W. Spaulding..........President
2521 "G" St., Bakersfield 93301
James R. Friend..........Secretary
P. O. Box 2087, Bakersfield 93303

KINGS County Medical Society, P. O. Box 1003, Hanford 93230. Meets Second Thursday, 7:00 p.m. Place to be announced.
James Dean..........President
912 N. Douty St., Hanford 93230
Willard S. Bridwell..........Secretary
1507 N. 11th Ave., Hanford 93230

LASSEN-PLUMAS-MODOC-SIERRA County Medical Society. Meets on call.
James M. McCullough..........President
50 N. McDow St., Susanville 96130
Charles W. Brown..........Secretary
Western Pacific Hospital, Portola 96122

LOS ANGELES County Medical Assn., 1925 Wilshire Blvd., Los Angeles 90057.
Joseph F. Boyle..........President
1136 W. Sixth St., Los Angeles 90017
Homer C. Pheasant..........Secretary
2300 S. Hope St., Suite 300,
Los Angeles 90007

MARIN Medical Society, 1601 Second St., Suite 106, San Rafael 94901. Meets First Thursday, 7:00 p.m.
Robert Mills..........President
711 D St., San Rafael 94901
William A. Galeno..........Secretary
712 D St., San Rafael 94901

MENDOCINO-LAKE County Medical Society.
Frank H. Dailey..........President
719 S. Dora, Ukiah 95482
Patrick R. Allanson..........Secretary
765 S. Dora St., Ukiah 95482

MERCED-MARIPOSA Medical Society, P. O. Box 586, Merced 95340. Meets Fourth Thursday; meeting place changes.
Patrick J. Maloney..........President
P. O. Box 586, Merced 95340
Robert J. Schiffler..........Secretary
P. O. Box 586, Merced 95340

MONTEREY County Medical Society, P. O. Box 308, Salinas 93903. Meets First Thursday.
Joseph E. Turner..........President
1010 Cass St., Monterey 93940
Thomas S. Elliott..........Secretary
101 Maple St., Greenfield 93927

NAPA County Medical Society, P. O. Box 2158, Napa 94559. Meets Second Wednesday.
Richard W. Neil..........President
1375 Main St., St. Helena 94574
Victor J. Bouillon..........Secretary
3020 Beard Rd., Napa 94558

ORANGE County Medical Association, 300 S. Flower, Orange 92666. Meets First Tuesday, 7:00 p.m.
Wallace A. Gerrie..........President
1901 Westcliff Dr., Newport Beach 92660
David I. Nielsen..........Secretary
400 Westminster Blvd., Newport Beach 92660

PLACER-NEVADA County Medical Society. Meets Second Wednesday.
F. Harold Johnson..........President
305 Oak Ridge Dr., Roseville 95678
Arthur R. Weaver..........Secretary
1212 High St., Suite 106, Auburn 95603

RIVERSIDE County Medical Association, 4175 Brockton Ave., Riverside 92501. Meets Second Monday, 7:30 p.m., Spanish Arts Gallery, Mission Inn.
Roger W. Ridley..........President
6969 Brockton Ave., Riverside 92506
Richard W. Trotter..........Secretary
6876 Magnolia Ave., Riverside 92506

SACRAMENTO County Medical Society, 5380 Elvas Ave., Sacramento 95819. Meets Third Tuesday, 8:30 p.m. Location of meetings varies.
James W. Martin..........President
2811 L St., Sacramento 95816
James O. Farley..........Secretary
3428 Watt Ave., Sacramento 95821

SAN BENITO County Medical Society. Meets once a month, except July and August. Time and place to be announced.
Robert D. Quinn..........President
930 Sunset Dr., Hollister 95023
Roderick L. Yip..........Secretary
891 Sunset Dr., Hollister 95023

SAN BERNARDINO MEDICAL SOCIETY, 369 Court St., San Bernardino 92401. Meets First Tuesday, 7:30 p.m., Harold's Club, Fontana.
Richard W. Judd..........President
1700 Waterman Ave., San Bernardino 92404
Daniel J. Rothenberger..........Secretary
321 Cajon St., Redlands 92373

SAN DIEGO County Medical Society, 3427-4th Ave., San Diego 92103. Meets Second Tuesday. Location of meetings varies.
John Wanless..........President
3427 Fourth Ave., San Diego 92103
W. C. Herrick..........Secretary
233 A St., San Diego 92101

SAN FRANCISCO Medical Society, 250 Masonic Ave., San Francisco 94118.
Albert G. Clark..........President
450 Sutter St., San Francisco 94108
Harry W. Sawyer..........Secretary
909 Hyde St., San Francisco 94109

SAN JOAQUIN County Medical Society, 445 W. Acacia St., P. O. Box 230, Stockton 95201. Meets First Thursday, 8:15 p.m.
Thomas Huff..........President
512 W. Harding Way, Stockton 95204
Dora A. Lee..........Secretary
2420 N. California St., Stockton 95204

SAN LUIS OBISPO County Medical Society, P.O. Box 319, San Luis Obispo 93402. Meets Third Saturday, 7:00 p.m., Madonna Inn, San Luis Obispo 93402.
Robert Buck..........President
P. O. Box 319, San Luis Obispo 93402
Billy W. Mounts..........Secretary
P. O. Box 319, San Luis Obispo 93402

SAN MATEO County Medical Society, 122 El Camino Real, San Mateo 94401. Meets Third Tuesday.
Stanford B. Rossiter..........President
Sequoia Hospital, Redwood City 94602
Eldon E. Ellis..........Secretary
2900 Whipple Ave., Redwood City 94602

SANTA BARBARA County Medical Society, 300 W. Pueblo St., Santa Barbara 93105. Meets Second Monday of the month, University Club, 1332 Santa Barbara St., Santa Barbara.
T. K. Hill..........President
1421 State St., Santa Barbara 93105
Casimir Domz..........Secretary
317 W. Pueblo St., Santa Barbara 93105

SANTA CLARA County Medical Society, 700 Empey Way, San Jose 95128. Meets First Monday.
Gerald Besson..........President
700 Empey Way, San Jose 95128
Alexander MacKenzie..........Secretary
700 Empey Way, San Jose 95128

SANTA CRUZ County Medical Society. Meets every Second Month, Second Tuesday. Time, place to be announced.
W. W. Hoskins..........President
30 Brennan St., Watsonville 95076
William Blade..........Secretary
1510 Seabright Ave., Santa Cruz 95062

SHASTA-TRINITY County Medical Society. Meets Second Monday of the month. Place to be announced.
Joseph T. DeWitte..........President
1129 Hartnell Ave., Redding 96001
Eugene A. Nutley..........Secretary
2625 Edith Ave., Redding 96001

SISKIYOU County Medical Society. Meets on call of President.
Robert L. Anderson..........President
504 Hillcrest Dr., Yreka 96097
John P. Geyman..........Secretary
705 S. A St., Mt. Shasta 96067

SOLANO County Medical Society, 631 Tennessee St., Vallejo 94599.
Tuesday, 8:00 p.m., at different meeting places.
Robert L. Garrett..........President
1512 Napa St., Vallejo 94590
Keith E. Vincent..........Secretary
2121 Redwood St., Vallejo 94594

SONOMA County Medical Society, 1625 Franklin Ave., Santa Rosa 95404. Meets Second Thursday.
John S. Dunn..........President
576 B St., Santa Rosa 95401
Elizabeth Buckley..........Secretary
1140 Sonoma Ave., Santa Rosa 95405

STANISLAUS County Medical Society, 1125-13th St., Modesto 95354. Meets Third Tuesday of the month, 7 p.m., Hotel Covell, Modesto.
Howard Kuhl..........President
1700 McHenry Ave., Modesto 95350
Jeanne I. Miller..........Secretary
709-18th St., Modesto 95354

TEHAMA County Medical Society. Meets at call of President.
W. Scott Davis..........President
P. O. Box 118, Los Molinos 96055
M. Blair McGee..........Secretary
217 Corona Ave., Corning 96021

TULARE County Medical Society, 1640 West Mineral King. P. O. Box 16, Visalia 93278.
Irving M. Schor..........President
P. O. Box 1151, Visalia 93277
George Tiss..........Secretary
P. O. Box 7, Visalia 93277

VENTURA County Medical Society, 3291 Loma Vista Rd., Room 40, Ventura 93003. Meets Second Tuesday each month, 7:30 p.m., Los Posas Country Club, Camarillo.
Joseph F. Gstettenbauer..........President
3003 Loma Vista Rd., Ventura 93003
Franklin K. Helbling..........Secretary
3291 Loma Vista Rd., Room 40, Ventura 93003

YOLO County Medical Society. Meets First Wednesday.
Benjamin R. Robinson..........President
650 Third St., Woodland 95695
Corbin Young..........Secretary
101 Main St., Winters 95694

YUBA-SUTTER-COLUSA County Medical Society.
John R. Heckman..........President
714-4th St., Marysville 95901
Norman K. Noordhoff..........Secretary
320 G St., Marysville 95901

Officers—CALIFORNIA PHYSICIANS' SERVICE—1966-67

BLUE SHIELD

Officers:

Chairman of the Board	Richard S. Wilbur
Vice-Chairman of the Board	Milo A. Youel
Secretary	Wilbur G. Rogers
Treasurer	Bert L. Halter

BOARD OF *Trustees:*

Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Roberta F. Fenlon*	San Francisco
Donald R. Fitch	Glendale
Mr. Thomas Hadfield	San Francisco
Bert L. Halter	San Francisco
Donald C. Harrington	Stockton
M. M. Haskell	Long Beach
Carl Goetsch	Berkeley
Mr. Philip S. Magruder	Flintridge
Gregory C. Murray	Los Angeles
Wilbur G. Rogers*	Glendale
Mr. Paul Schrade	Los Angeles
William H. Thompson	San Mateo
Richard S. Wilbur*	Palo Alto
Milo A. Youel	San Diego

---

*Appointed by CMA Council

BOARD COMMITTEES:

Executive Committee

Richard S. Wilbur, Chairman	Palo Alto
Carl Goetsch	Berkeley
Bert L. Halter	San Francisco
Mr. Philip S. Magruder	Flintridge
Wilbur G. Rogers	Glendale
William H. Thompson	San Mateo
Milo A. Youel	San Diego

Finance Committee

Bert L. Halter, Chairman	San Francisco
Mr. Ransom Cook	San Francisco
Mr. John Cowee	Berkeley
Burt L. Davis	Palo Alto
Donald R. Fitch	Glendale
Mr. Philip S. Magruder	Flintridge

Medical Policy Committee

Wilbur G. Rogers, Chairman	Glendale
Richard F. Altman	Newport Beach
Carl E. Anderson	Santa Rosa
R. W. Burnett	Bakersfield
Roberta F. Fenlon	San Francisco
Carl Goetsch	Berkeley
Mr. Thomas Hadfield	San Francisco
M. M. Haskell	Long Beach
Donald C. Harrington	Stockton
Gregory C. Murray	Los Angeles

"COCA-COLA" AND "COKE" ARE REGISTERED TRADE-MARKS WHICH IDENTIFY ONLY THE PRODUCT OF THE COCA-COLA COMPANY.

Coke
Coca-Cola
For the taste
you never
get tired of.

helps solve "the other problem" in venereal disease

The "other problem" in venereal disease is the sensitivity of many patients to penicillin and the increased resistance of the gonococcus.

Regarding Increased Resistance—During the last eight years, 5700 strains of *N. gonorrhoeae* have been isolated and tested for sensitivity to penicillin and sulfadiazine in the Public Health Laboratory (Toronto). (1) In the six-month period of January to June, 1966, no less than 18.8 per cent of the *N. gonorrhoeae* strains isolated required 1.0 unit of penicillin per ml to inhibit their growth; and 8.6 per cent required more than 1.0 unit. In contrast, only eight years ago, 98 per cent of the isolates were sensitive to 0.1 unit of penicillin or less.

Regarding Sensitivity—It has been reported that approximately 15 per cent of all patients admitted to a large hospital have a history of being allergic or hypersensitive to penicillin. It likewise has been stated that conventional skin testing with penicillin is not reliable and that more elaborate testing for sensitivity is not readily available. (2)

Regarding DECLOMYCIN—Excellent results have been achieved with DECLOMYCIN as a therapeutic alternative in a series of studies (3-8) representing a cross-section of national experience (Los Angeles, California; Columbia, So. Carolina; Houston, Texas; New York, New York; Boston, Massachusetts and Washington, D.C.). 1931 patients received DECLOMYCIN for treatment of acute gonorrheal urethritis. *The overall cure rate achieved was 89 per cent!**

In syphilis, dosage schedules of a total of 12 to 18 Gm given in equally divided doses over a period of 10 to 15 days should be followed.

*The above studies utilized DECLOMYCIN in a variety of dosage schedules. The recommended adult dosage of DECLOMYCIN is 600 mg divided into two or four doses daily.

1. Amies, C. R.: Development of Resistance of Gonococci to Penicillin: An Eight-Year Study. *Canad. Med. Ass. J.* 96(1):33 (Jan.7) 1967. **2.** Garagusi, V. F.: Antibiotic Review. *Amer. Fam. Phys.* 11:61 (Nov.) 1966. **3.** Sokoloff, B.: Demethylchlortetracycline Therapy in Acute Gonococcal Urethritis. *Clin. Pharm. Ther.* 6:350 (May-June) 1965. **4.** Allison, J. R., Jr.: Demethylchlortetracycline Hydrochloride in the Treatment of 267 Patients with Acute Gonorrhea: Results and Evaluation. *Antibiot. Chemother.* 11:454 (July) 1961. **5.** Vanderstoep, E. M.; Matheson, T. E.; Moore, M. B.; Short, D. H., and Knox, J. M.: A Comparison of Penicillin and Demethylchlortetracycline in the Treatment of Acute Gonorrheal Urethritis in the Male. *Southern Med. J.* 57:201 (Feb.) 1964. **6.** Marmell, M. and Prigot, A.: The Therapeutic Value of Demethylchlortetracycline in Gonorrhea, Lymphogranuloma Venereum, and Donovanosis. *Antibiotics Annual,* 1959-1960, p. 457. **7.** Pochi, P. E. and Strauss, J. S.: The Single Dose Treatment of Acute Gonococcal Urethritis with Demethylchlortetracycline. *Antibiot. Med.* 8:75 (Feb.) 1961. **8.** Greaves, A. B.: Demethylchlortetracycline in the Treatment of Venereal Disease. Unpublished data on file, Medical Research Section, Lederle Laboratories. 439-6—4797

DECLOMYCIN®

DEMETHYLCHLORTETRACYCLINE

Lederle

prescribing information ▶
on next page

helps solve
"the other problem"
in venereal disease

Effective in a wide range of everyday infections—respiratory, urinary tract and others—in the young and aged—the acutely or chronically ill—when the offending organisms are tetracycline-sensitive.

Contraindication: History of hypersensitivity to demethylchlortetracycline.

Warning—In renal impairment, usual doses may lead to excessive accumulation and liver toxicity. Under such conditions, lower than usual doses are indicated, and, if therapy is prolonged, serum level determinations may be advisable. A photodynamic reaction to natural or artificial sunlight has been observed. Small amounts of drug and short exposure may produce an exaggerated sunburn reaction which may range from erythema to severe skin manifestations. In a smaller proportion, photoallergic reactions have been reported. Patients should avoid direct exposure to sunlight and discontinue drug at the first evidence of skin discomfort. Necessary subsequent courses of treatment with tetracyclines should be carefully observed.

Precautions—Overgrowth of nonsusceptible organisms may occur. Constant observation is essential. If new infections appear, appropriate measures should be taken. In infants, increased intracranial pressure with bulging fontanels has been observed. All signs and symptoms have disappeared rapidly upon cessation of treatment.

Side Effects—Gastrointestinal system—anorexia, nausea, vomiting, diarrhea, stomatitis, glossitis, enterocolitis, pruritus ani. Skin—maculopapular and erythematous rashes. A rare case of exfoliative dermatitis has been reported. Photosensitivity; onycholysis and discoloration of the nails (rare). Kidney—rise in BUN, apparently dose related. Hypersensitivity reactions—urticaria, angioneurotic edema, anaphylaxis. Teeth—dental staining (yellow-brown) in children of mothers given this drug during the latter half of pregnancy, and in children given the drug during the neonatal period, infancy and early childhood (up to 12 years). Enamel hypoplasia has been seen in a few children. If adverse reaction or idiosyncrasy occurs, discontinue medication and institute appropriate therapy.

Average Adult Daily Dosage: 150 mg q.i.d. or 300 mg b.i.d. Should be given 1 hour before or 2 hours after meals, since absorption is impaired by the concomitant administration of high calcium content drugs, foods and some dairy products.

Capsules: 150 mg; *Tablets:* film coated, 300 mg, 150 mg, and 75 mg of demethylchlortetracycline HCl.

DECLOMYCIN®
DEMETHYLCHLORTETRACYCLINE

Lederle
LEDERLE LABORATORIES, A Division of
American Cyanamid Company,
Pearl River, New York

OFFICERS

OF THE CALIFORNIA MEDICAL ASSOCIATION

(Continued from Page 5)

James O. Farley, Sacramento
Sanford Feldman, San Francisco
Jacob J. Foster, Salinas
Menno S. Gaede, Reedley
Norman Graff, San Mateo
Bert L. Halter, San Francisco (Chmn.)
Alexander Hatoff, Oakland
John R. Heckman, Marysville
Merlin A. Hendrickson, Rialto
J. Ralph Hughes, Bridgeport
Robert E. James, Belmont
William K. Jennings, Santa Barbara
Donald O. Lagerlof, Los Angeles
Jack B. Lomas, Los Angeles
Rolland C. Lowe, San Francisco
Andrew D. Lucine, San Jose
John C. Lungren, Long Beach
Arthur Lyons, San Francisco
Richard H. McDonald, Garden Grove
John McGee, San Anselmo
James A. Mattison, Salinas
Harold M. Messenger, San Diego
William A. Newsom, San Francisco
Paul Newton, Oakland
Shepard F. Palitz, Bakersfield
Calvin Plumhof, Kentfield
A. John Quinn, Sacramento
E. William Rector, Sacramento
James L. Rhee, Oakland
Pierce A. Rooney, Sacramento
Ray Seavers, San Bernardino
Gerald W. Shaw, Santa Monica
Stanley Smiley, Sacramento
Wilfred J. Snodgrass, Santa Monica
William T. Soldmann, Jr., La Mesa
Frank Solomon, San Francisco
William H. Stephenson, Santa Barbara
William L. Sumption, Santa Monica
Richard L. Sutherland, Oakland
Coe Tanner Swift, Madera
Donald F. Taugher, Monterey
Joseph W. Telford, San Diego
Rudolph B. Toller, Stockton
William W. Tucker, Sacramento
B. G. Wagner, Sacramento
William B. Wenner, Monterey
Ross Willoughby, Fresno
Robert M. Zweig, Riverside

Committee on Health Facilities Planning

Jean F. Crum, Downey	1968
Glenn A. Pope, Sacramento (Chmn.)	1968
Richard L. Taw, Los Angeles	1968
James W. Dalton, Santa Barbara	1969
Paul C. Doehring, Glendale	1969
Pierre Salmon, San Mateo	1969
Laurence A. Mosier, Garden Grove	1970
Joseph W. Telford, San Diego	1970
Donald M. Gallagher, San Francisco	1970
Charles A. Webster, Oakland	1970

CONSULTANTS: Jack B. Lomas, Los Angeles; Robert F. Schell, San Rafael.

* * *

COUNCIL COMMITTEES

Finance Committee

Harold Kay, Oakland; Frank C. Melone, Ontario; Elmer F. Gooel, Beverly Hills; Henry V. Eastman, Tustin; Marvin J. Shapiro, Encino.

Committee on Legislation

Dan O. Kilroy, Sacramento (Chmn.); Stuart C. Knox, Los Angeles; Harold E. Wilkins, Downey; John Rumsey, San Diego; Samuel R. Sherman, San Francisco.

EX-OFFICIO: John G. Morrison, San Leandro; Malcolm C. Todd, Long Beach; William F. Quinn, Los Angeles; Albert G. Miller, San Mateo.

Advisory Board to Woman's Auxiliary

John G. Morrison, San Leandro (Chmn.); Malcolm C. Todd, Long Beach; Ralph C. Teall, Sacramento; James C. MacLaggan, San Diego; Fred Ackerman, Pleasant Hill.

Physicians Benevolence Fund Operating Committee

Clyde L. Boice, Palo Alto (Chmn.); Alexander Fraser, San Francisco; Elizabeth Mason Hohl, Los Angeles; Don C. Musser, San Francisco; George Wolf, Fresno; Dudley M. Cobb, Jr., Los Angeles; Frank C. Melone, Ontario.

Worldwide clinical experience confirms the predictable therapeutic potential of Synalar

It is particularly gratifying that the promise of the advanced chemical design and high order of bioassay activity of Synalar (fluocinolone acetonide) has been confirmed by widespread therapeutic application. Indeed, the impressive clinical response rate of Synalar has been documented in no fewer than 232 papers from 22 countries.

Representative Clinical Results with Synalar*

Efficacy Documented in over 4,000 Patients

Condition	Number of Publications	Number of Patients	Significant Improvement†
Contact Dermatitis	27	750	713
Eczematous Dermatitis	21	472	409
Seborrheic Dermatitis	18	442	426
Atopic Dermatitis	24	460	426
Psoriasis	36	1,699	1,510
Neurodermatitis	18	351	324
Total	144	4,174	3,808

*Complete bibliography on request.

†Expressed by the authors as excellent, very good, good, complete remission of inflammation, etc.

Prescribing Information

For initiation of therapy: Cream 0.025%, 5 and 15 Gm. tubes, 425 Gm. jars; *for emollient effect:* Ointment 0.025%, 15 Gm. tubes; *for maintenance therapy:* Cream 0.01%, 15 and 45 Gm. tubes, 120 Gm. jars; *for intertriginous or hairy sites:* Solution 0.01%, 20 cc. and 60 cc. plastic squeeze bottles; *for infected inflammatory dermatoses:* Neo-Synalar® Cream (0.025% fluocinolone acetonide, neomycin sulfate, equivalent to 0.35% neomycin base), 5 and 15 Gm. tubes.

Contraindications: Tuberculous, fungal, and most viral lesions of the skin, (including herpes simplex, vaccinia, and varicella). Not for ophthalmic use. Contraindicated in individuals with a history of hypersensitivity to any of the components. Precautions: Synalar preparations are virtually nonsensitizing and nonirritating. However, the solution may produce burning or stinging when applied to denuded or fissured areas. In some patients with dry lesions, the solution may increase dryness, scaling or itching. While topical steroids have not been reported to have an adverse effect on pregnancy, the safety of their use on pregnant females has not absolutely been established. Therefore, they should not be used extensively on pregnant patients, in large amounts, or for prolonged periods of time. Prolonged use of any antibiotic may result in overgrowth of nonsusceptible organisms; if this occurs, appropriate therapy should be instituted. When severe local infection or systemic infection exists, the use of systemic antibiotics should be considered, based on susceptibility testing. Side Effects: Side effects are not ordinarily encountered with topically applied corticosteroids. As with all drugs, however, a few patients may react unfavorably to Synalar under certain conditions. The neomycin in Neo-Synalar Cream rarely produces allergic reactions.

References: 1. Lerner, L. J., Bianchi, A., Turkheimer, A. R., Singer, F. M., and Borman, A.: Anti-inflammatory steroids: potency, duration and modification of activities. Ann NY Acad Sci 116:1071 (Aug. 27) 1964. 2. Idem: Comparison of anti-granuloma, thymolytic and glucocorticoid activities of anti-inflammatory steroids. Proc Soc Exp Biol Med 116:385 (June) 1964. 3. Ringler, A.: Activities of adrenocorticosteroids in experimental animals and man, in Dorfman, R. I.: Methods of hormone research, New York, Academic Press, 1964. vol. III. pp. 234-280. 4. Gubersky, V. R.: To be published.

fluocinolone acetonide — an original steroid from
SYNTEX
LABORATORIES INC., PALO ALTO, CALIF.

For inflammatory dermatoses... by any measure a topical corticosteroid of choice

Synalar® (fluocinolone acetonide)

Milligram for milligram one of the most active topical corticosteroids available

Rapid and predictable in antiinflammatory and antipruritic activity

Results often comparable to those of systemic corticosteroids with fewer hazards

What can be done for Susan Jane To stop the runs and crampy pain?

Parepectolin for quick relief of acute diarrhea
...soothes colicky pain with paregoric
...consolidates fluid stools with pectin
...adsorbs irritants with kaolin, and protects intestinal mucosa

In children, Parepectolin may be used to control diarrhea promptly and prevent dehydration, until etiology has been determined. In some cases, Parepectolin may be all the therapy necessary.

Parepectolin®

Each fluid ounce of creamy white suspension contains:
Paregoric (equivalent)........(1.0 dram) 3.7 ml.
Contains opium (¼ grain) 15 mg. per fluid ounce.
warning: may be habit forming
Pectin .... (2½ grains) 162 mg.
Kaolin (specially purified)....(85 grains) 5.5 Gm.
(alcohol 0.69%)
Usual Children's Dose: One or two teaspoonfuls three times daily.

RORER
WILLIAM H. RORER, INC.
Fort Washington, Pa.

Physicians Join Clergymen In Communications Exchange

Interested physicians now have the opportunity to take part in projects designed to improve the relationship between a patient's physician and his clergyman.

Pilot projects designed by the AMA Department of Medicine and Religion, are being conducted in 17 theological seminaries across the nation in cooperation with 17 county medical societies in these areas.

These projects are designed to help seminary students understand their role in American medicine. "Medicine and religion is a two-way road," Robert Etheridge, Department of Medicine and Religion, explains. "The physician should learn to work with the minister in treating a patient and the minister should be able to discuss a patient's problems with the physician."

Some of the topics considered in the projects include what a minister should know regarding his relationship to a patient and a patient's family; what areas of medical knowledge a clergyman needs to understand in order to properly counsel a patient and to work with the physician—medical nomenclature, venereal disease, sex education and premarital preparation, for example; and what a minister should know of hospital procedures; his relationship with staff members and the proper conduct in an intensive care unit, the recovery room or surgery.

The pilot programs will not only help participating students, but will provide the basic information for a book to be published by the AMA later this year.

The book will contain a full report of the results of the pilot studies from the viewpoint of students, faculty and the medical societies.

These reports will be prepared by participants at the end of the present school semester and presented to the AMA for review and use in the book. The result will be a guide of subjects and procedures to help county medical societies set up individual programs with seminaries in their own area.

Physicians desiring to participate in these worthwhile projects should contact the committee on medicine and religion of their county medical society. Physicians are needed to lecture, direct roundtable discussions with senior seminary students and participate on seminar panels. In addition, physicians are invited to audit these classes and sessions simply for their own benefit.

A theological Seminary will be held in Berkeley. Participants will be the Pacific School of Theology and the Alameda-Contra Costa County Medical Association.

in many types of otitis externa—of varying etiology—as part of the treatment regimen

Coly▲Mycin®S Otic
with Neomycin and Hydrocortisone
Each ml. provides:

Colistin base activity (as the sulfate)
3.0 mg.
Neomycin base activity (as the sulfate)
3.3 mg.
for complementary broad-spectrum bactericidal action

Hydrocortisone acetate
10.0 mg. (1%)
for anti-inflammatory/antipruritic benefits

Thonzonium bromide
0.5 mg. (0.05%)
for more rapid dispersion and penetration

It also contains polysorbate 80, acetic acid and sodium acetate in a buffered (pH 5) aqueous vehicle and thimerosal 0.002% as a preservative.
Buffered to pH 5 to reduce the abnormal alkaline pH which may be favorable to bacterial growth, and to help restore the normal acid pH of the ear canal.[8,9]
combats the bacterial cause
relieves the dermatologic manifestations
Recommended Dosage: Therapeutic—4 drops t.i.d. Prophylactic—4 drops before and after swimming.
Contraindication: A history of sensitivity to any of the components, or in tubercular, fungal and most viral lesions; especially herpes simplex, vaccinia and varicella.
Precautions: If sensitivity or irritation occurs, medication should be discontinued promptly. Overgrowth of resistant organisms is possible. Use with care in cases with perforated eardrum or in long-standing otitis media because of the possibility of ototoxicity caused by neomycin. Increased skin sensitivity to neomycin has been noted in current medical literature.
Adverse Reactions: A low incidence of mild burning or painful sensation in the ear has been reported. Such local effects do not usually require discontinuance of medication. Sensitivity reactions were reported in a few instances.
Full information is available on request.
References: 1. McLaurin, J. W.: J.A.M.A. *154*: 207, 1954. 2. Sulzberger, M. B., *et al.*: Dermatology: Diagnosis and Treatment, ed. 2, Chicago, The Year Book Publishers, Inc., 1961, p. 595. 3. Stewart, J. P.: J. Laryng. & Otol. *65*:24, 1951. 4. Elia, J. C.: Southwestern Med. *42*:405, 1961. 5. McLaurin, J. W.; Raggio, T. P., and Simmons, M.: Laryngoscope *75*:1699, 1965. 6. Mann, P. H.: Current Therap. Res. *6*:101, 1964. 7. Saltzman, M.: Clin. Med. *70*:559, 1963. 8. Ettenger, M. S.: Pennsylvania M. J.: *66*:35, 1963. 9. Pullen, F. W. II: Eye Ear Nose & Throat Month. *42*: 38, 1963. 10. Jones, E. H., and McLain, P. G.: Eye Ear Nose & Throat Month. *43*:52, 1964.

WARNER-CHILCOTT
CO-GP-72 R-1
Morris Plains, N.J.

Medical Motion Pictures, Color TV To Again Be Features at AMA Annual Convention

Medical motion pictures and color television will be a feature of the Annual Convention of the American Medical Association again this year.

The Convention is to be held in Atlantic City June 18 to 22; the Scientific Program at Convention Hall and nearby hotels and the House of Delegates at the Chalfonte-Haddon Hall Hotel.

Medical motion pictures have become an integral part of the Annual Convention program. Movies are carefully screened and selected for quality, content and diversity of subject matter. Some are chosen from the AMA library of medical motion pictures while others are picked from among films just completed. Several new films are usually shown for the first time at the Annual Convention. The total movie program is thus planned to achieve both variety and currency.

Medical motion pictures will be presented daily. At least five color television programs will be presented live, on a closed circuit from a Philadelphia hospital in cooperation with the University of Pennsylvania School of Medicine.

Several of the Scientific Sections will participate in this year's color television program.

The entire Scientific Program for the 1967 Annual Convention was published in the May 8 issue of the *Journal of the AMA*.

★
Specialized Service
IN
PROFESSIONAL LIABILITY
INSURANCE
is a high mark
of distinction

THE
MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA
Professional Protection Exclusively since 1899

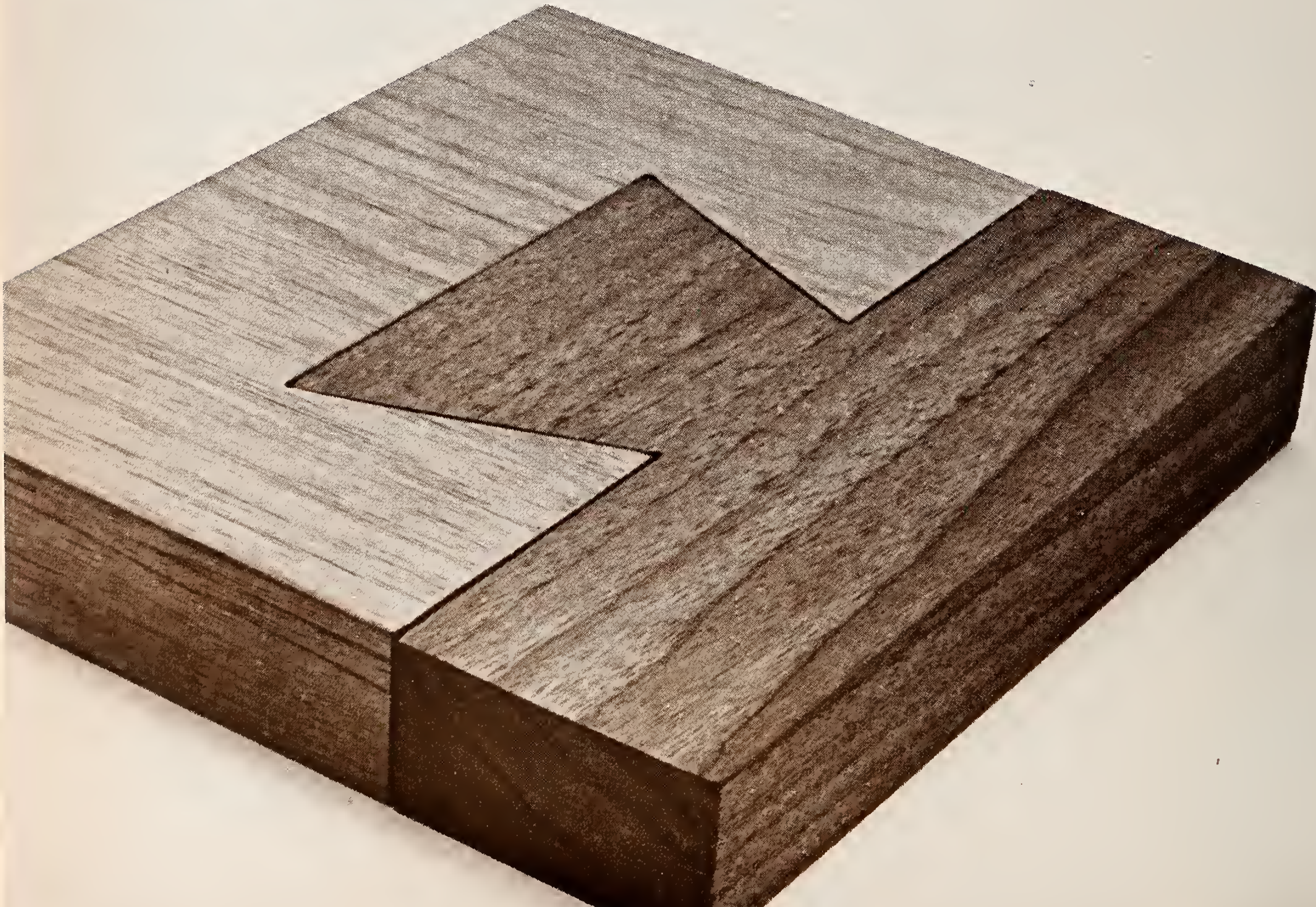
NORTHERN CALIFORNIA OFFICE:
Gordon C. Jones and John K. Galloway, Representatives
1518 Fifth Avenue, San Rafael, Telephone: 415-453-5140
Mailing Address: P.O. Box 1079, San Rafael 94902
SOUTHERN CALIFORNIA OFFICE:
Davis S. Spencer and Grady T. Dyer, Representatives
Suite 3, 33 East Huntington Drive, Arcadia, Tel. 213-681-5239
Mailing Address: P.O. Box 543, Arcadia 91008

even better together
in mild hypertension

Serpasil®-Esidrix®

(reserpine and hydrochlorothiazide CIBA)

Combination Tablets



California
Medicine

Accomplishments and Frustrations

A Report of Two Years in the Life of the California Medical Association

JAMES C. MAC LAGGAN, M.D., *San Diego*

It is my privilege to bring you a report of the accomplishments and frustrations our Association has experienced during the past two years. In reality, this is the story of basic evolutionary changes in attitude and relationships of medicine over a long span of years. It is the story of the gradual involvement of the medical profession in health matters beyond the actual delivery of professional services.

It has not always been an easy or popular sequence of events. Not all the members of our Association are necessarily pleased with the direction in which the recent activities have pointed. However, let me assure you that at no time have your elected leaders failed to consider every possibility before a course of action was pursued.

One George Herzog puts it this way: This has been Chinese year 4665, the Jewish year 5726, the Buddhist year 2509, the 900th anniversary of the Battle of Hastings, but to doctors it has been, above all, Medicare year 1.

This involvement has been a long time coming.

One example is the California Workmen's Compensation law passed originally in 1912. Medicine at that time did not wish to become involved with government in any way; so they did not advise, or participate in the writing or implementation of the law. As a consequence, the law was not realistic; it was difficult to work under; and it was hated. We did, however, live under that law for more than 50 years before basic changes were made. This is a classic example of what happens when an interested party refuses to become involved in laws that affect his livelihood.

As time passed, opinions and people changed! This House, at one time in the thirties, actually passed a resolution calling on California to set up a system of state medicine for its citizens—fortunately this was rescinded. It was at this time that realistic involvement in the problems of provision of medical care began.

One step was the formation of California Physicians' Service (now Blue Shield) to help persons of low income to meet their medical expenses. Other states recognized the same need, and the Blue Shield concept spread throughout the country.

As the prepayment concept gained momentum, commercial carriers entered the field. The physician's attitude was that this was a contract between an insurance agency and an individual or a group, and of little interest to him. So minimal was the concern by physicians that chaos resulted. We all know the problems that arose from unrealistic fee schedules, restrictive clauses, and unreliable promises. These problems continued until after World War II.

Then came Big Government, with proposals, bills, grants and institutes, until every medical school and most physicians were somehow participating in a government program. Involvement was sometimes limited to paying taxes to support it all. However, advice was being given; funds were being accepted; involvement was here to stay.

This is the valedictory address of Dr. MacLaggan at the end of his term as President of the California Medical Association. It was presented before the House of Delegates, California Medical Association, Los Angeles, 15 April 1967.

Reprint requests to: 525 Hawthorne Street, San Diego 92101.

But the past is prelude, and it is our status today that interests us. We are, and have been for some time, faced with the fact that government is heavily involved in medical care, and, in all likelihood, will become more involved in the future. A clear choice confronts Medicine. Either we ignore the whole thing, as had been done in the past, or we try to shape the future. We can promote realistic programs beneficial to the people of California, and acceptable to physicians, or we can remain aloof and fight to the end social trends that seem inevitable.

We chose to become involved. This involvement has taken many forms—sponsoring legislative action in our own state, participating in the writing of regulations to implement federal laws, sponsorship of new concepts; and, perhaps most important, a new relationship with allied professional groups.

I will try to give you a few examples of what this realistic involvement has accomplished. From a small start a few years ago, with a joint committee meeting once a year, our relationship with the California Hospital Association has grown in stature to an attitude of mutual trust and dependence. We have several joint ventures and frequent consultations.

Each group talks with the other before embarking on a legislative venture, or before developing a program that would involve the other.

Similar liaison has been established with other health related organizations.

The deans of all medical schools are invited to each Council meeting. State governmental agency heads meet regularly with our Council and committees to consult on their activities.

What has been accomplished by this? Let's review some of the events of the past two years.

Title 19 and its California implementation is of primary interest. The willingness of CMA committee members, officers and staff to become involved gave us opportunities to: Advise and consult with the Legislature and various bureaus of state government; guide both the legislation and the regulations in a direction which we believe is the best for the people of California and that the physicians of California can generally accept. The situation here is different from other states, especially in New York, where there has been less physician involvement in planning for Title 19.

The efforts of California physicians in the implementation of Title 18 is another example. Sam Sherman, John Rumsey, Roberta Fenlon, to name but a few—contributed immensely to the development of regulations that, cumbersome as they may seem, are far superior to those written prior to physician consultation and guidance.

Other federal legislation has directly affected the activities of the California Medical Association during the recent months. A major project came with the Cancer and Stroke law. Our concern with this started before the bill was passed, with a meeting called by the Bureau of Research and Planning. Invited to this meeting were deans of medical schools in California, California Hospital Association, representatives of the federal government, including the now Surgeon General, William Stewart, and others. Subsequently, a California Coordinating Council was formed with representation from CMA, CHA, medical schools, the Department of Public Health, and voluntary agencies for heart and cancer. This group, now known as the California Coordinating Committee for Regional Medical Programs, has received a planning grant, administered by our California Medical Education and Research Foundation. This Foundation will be responsible for all grants in California under the program. CMA has a grant to study the effectiveness of current continuing education courses, to take a census of courses now available and to study the needs for courses in heart, cancer and stroke. From the beginning we have been represented by Ralph Teall as vice chairman of the coordinating committee, Jack Morrison and me.

Another example of direct involvement of CMA: Many months ago the CHA became conscious of the manpower shortage in the health fields, and initiated plans to alleviate the problem. At about the same time, the California statutory Health Facilities and Services Planning Committee started to think about health personnel shortages. A health manpower conference was held with 60 professional and health groups to evaluate the needs. It was decided to set up a voluntary Health Manpower Council to devise means of improving the situation. A second conference was held in February, when the Council was formally established. It includes representation from CMA, CHA, nursing, nursing homes, dentistry and State departments of Public Health, Education and Employment. Thirteen other members are chosen from remaining representatives to the conference (which had grown to over 80 at the second meeting). This council will be financed voluntarily by the groups involved. Our representatives are Frank Melone, Gerald Scarborough and Harold Kay.

What else has CMA been involved in this year? CMA Accomplishments Report, intended for distribution to opinion leaders, describes our activities in many fields—maternal and infant mortality studies, student health programs, our active campaign to reduce cigarette smoking, evaluation of extended care facilities, health information distributed to hundreds of newspapers and magazines, radio and TV spots, the Doctors at Work series of films now being used in several other states in addition to wide distribution in California, the medical staff survey program that has now surveyed nearly five hundred hospitals.

All of these, and others, are public or community service projects supported by our Association. We should look on them with pride.

What about direct benefits to CMA members and county societies?

The Workmen's Compensation laws have been modified so that there is now a suitable committee structure, with an appeals board, physician representation and a more realistic fee schedule. The Medi-Cal program is more realistic in its approach to patient care, and pays reasonable charges. The Medical Practice Act was amended to make medical discipline more equitable. For example, it is now possible to impose restrictions on practice, or mandatory reeducation, instead of revocation of license, when this penalty seems too harsh. The Military Dependents Program has been extended to dependents of retired personnel, and usual and customary fees will start in July. California Blue Shield has been encouraged to write usual fee programs and by mid-year nearly 50 per cent of Blue Shield programs will be written on this concept.

If nothing else has impressed me during the past two years, one fact stands out very clearly: That changes in patterns of practice and involvement with governmental agencies and other groups have just begun. The impact of legislation, either recently passed or now under consideration, will create tremendous pressure for change within the medical profession in California. We must be flexible in our committee structure, alert for opportunities to stay ahead of legislative and social forces that bring about change. It is my fervent hope that our three planning committees will keep CMA abreast or ahead of the times and that our "action committees" will continue the standards of excellence which have marked their activities in the past.

Involved? Yes you are! Involvement? Yes we have! Eugene V. Debs said, "I realize that there are certain limitations placed upon the right of free speech. I may not be able to say all I think, but I am not going to say anything I do not think."

The activation of the thinking of its members, the stimulation of interest in our affairs, the answering of many practicing physicians' queries and complaints is a monumental accomplishment of the dedicated staff of CMA, your Council, your House of Delegates, your elected officers, executive secretaries and the members of the Woman's Auxiliary.

I extend my sincere appreciation:

To Carl Anderson, Chairman of our Council.

To Ralph Teall, el más grande de profesores, the greatest of teachers and your devoted past president.

To John Morrison, a man whom I admire and one who represents you so ably on a state and national level. To three chores—beyond his service to you—he has to loan his hardy strength: Sleeping on planes and hotel mattresses, breathing a variety of air or smog and eating on the roast beef circuit.

To the members of committees and commissions and their chairmen. Someone has said, "When a large group of the incapable appoints a smaller group of the unwilling to do the unnecessary, this constitutes a committee." I categorically deny that this is true in California. We strike out two letters, and we have: Capable, willing and necessary! I repeat, capable, willing and necessary!

To Howard Hassard and his staff a commendation for coordinating skilled professional people with talents and abilities we so greatly appreciate and need. Hope is a species of happiness, and I know we can depend on our staff for continuity throughout the year and from annual session to annual session. I am sorry I cannot mention each one personally but I do extend my gratitude to all of them.

In my thousands of miles of travel and my visits with you and others, I have been taught some lessons:

1. We must not assume that a critic is an enemy.

2. We must keep politics in the open, insofar as possible. The airing of our differences, with

ultimate resolution of problems will quiet criticism.

3. We must understand the reason for giving man status. Age should not have its face lifted, but rather teach the world to admire wrinkles.

4. We must learn to live with the mass media of communication and to exert our influence through these channels.

5. We must preserve the environmental peace and quiet that comes to the individual to stimulate independence and creative thinking.

In all of our future challenges of involvement, let us remember this thought:

"God grant me the serenity to accept things I cannot change, courage to change things I can, and wisdom to know the difference."

CORRECTION

In the article, "Blood Coagulation and the Coumarin Anticoagulant Drugs," which appeared in the April 1967 issue of CALIFORNIA MEDICINE, an error was made in the legend for Figure 15 on page 264. The legend read: "Figure 15. The 'cascade' or 'false waterfall' theory of the mechanism of blood coagulation . . ." It should have read: The "cascade" or "waterfall" theory.

tion. On the other hand, I have investigated the problem to some limited degree and have the following comments.

Nitrous oxide is used extensively as a cream whip propellent and, as such, is sold without prescription and without any specific controls. In addition, it is sold for the reduction of preignition in racing automobile engines and this also is an uncontrolled sale. One very large manufacturer in the city told me that they had reports of a large number of thefts of small cylinders in the San Fernando area in particular, and, indeed, these cylinders were being returned to them through the police force. The manufacturer also indicated that the probabilities are that nitrous oxide could be purchased without prescription from a variety of distributor sources.

While it would appear that the control of nitrous oxide is less than satisfactory, I must say that in recent times your letter is the first indication of the promiscuous use of this agent. I am enclosing for your information a copy of a notice that appeared in a recent journal which bears on this problem, but I would have you note that the date of this was 1799 (Science, 155:266, 20 January 1967).

I believe that this young man is certainly risking his life very foolishly and I think that your admonition to him that he has had, at least temporarily, the total destruction of the faculty of making logical judgments was entirely correct. This arises from two possible sources: (1) From the fact that he is using nitrous oxide without supplemental oxygen and therefore possibly suffering from oxygen lack and (2) as a direct result of the effects of the nitrous oxide itself. It is theoretically possible that the effects of the oxygen lack might be cumulative, but there is no evidence that I know of that would indicate that the effects of the nitrous oxide would be cumulative. I can well believe that the story he told you raised the hair on the back of your neck. I must say that I had an immediate mental image of someone precipitating himself into infinity in a most inglorious manner.

I seriously believe that this is probably a matter that should be given some official notice and I would believe that the notification of the Highway Patrol of the potentiality of this source of accident is certainly indicated. With your permission I will notify them, although it would seem to me that since this young man cannot be involved after the fact, you certainly would have no restraints in doing it. I would also like your permission to use the material in your letter for publication in a medical journal, obviously without the use of names, for if these cylinders are being stolen and used in the manner that they apparently are, it would indicate that hospitals and suppliers should become much more careful of their cylinder control. I believe, as I stated before, that the reason they have not in the past is because this has not been a problem to the best of my knowledge until the present time.

Again, may I thank you for bringing this extremely interesting and yet frightening matter to my attention. I trust that you will see it clear to allow me to at least use this as a matter of medical reporting and that you will find it possible, yourself, to notify the Highway Patrol and the police of the possibility of this source of accidents so that they will have their eyes open, or that you will let me do so if you feel that you should not.

I would very much like any follow-up on this that you may have, hopefully to the end that this individual will give up his practice, but, possibly, to the horrible conclusion that may result. Believe me, I sympathize with your responsibility in this matter.

Sincerely,

John B. Dillon, M.D.

27 February 1967

Dear Doctor Dillon:

Your letter of 17 February was gracious, as I knew it would be.

I am writing this day letters to the Police and Sheriff's departments and to the State Highway Patrol concerning the urgency of specific knowledge on the part of their personnel as to the meaning of the presence of a nitrous oxide cylinder in a private vehicle.

But the important thing is to get legislation to control the availability of this substance to the general public. The apparent impossibility of providing evidence of use poses a problem that you, as a scientist, would be better equipped to discuss than I. My lad tells me that a gulp of fresh air brings immediate and total recovery from the effects of direct breathing from the cylinder! His self-description may be on the subjective side; but obvioutly his recovery after an accident in "total black-out" was such in every case as to prevent investigating officers from questioning his ability to operate a vehicle. Knowing how hard it is for the police to enforce narcotics laws, one wonders how they could get a conviction of using this gas, even if tough legislation were enacted.

In this letter I would ask that you send a note to the law enforcement agencies, couched in more scientific language the information might be more impressive.

I hasten to assure you that you may feel free to use the matter in these letters in any way you wish, doctor. I read your letter to the young man and he agrees that if the information can help others, it should be used.

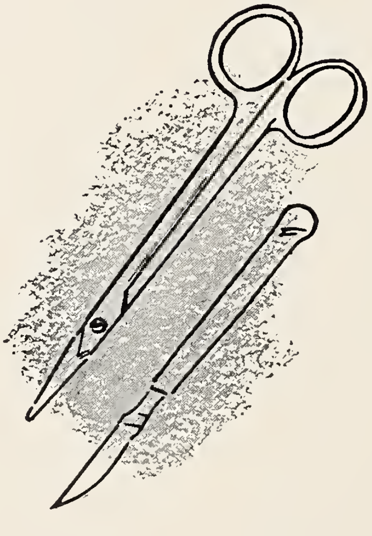
I failed to tell you (because I thought it impertinent in a layman) that the lad in question has great hopes that his findings in the field of nitrous oxide research might lead to its beneficial use in the treatment of retarded children. He was put out to discover that I did not refer to this facet of the matter, and I made bold to tell him that I considered this reference on his part to be a subconscious device to clear his conscience of a sense of guilt upon the continued use of the chemical. Naturally he was piqued by this analysis: He did not realize that I was not questioning the sincerity of his good intention. Further, I was confident that if there were any possibilities for the beneficial use of nitrous oxide in this direction, research people would surely have pursued the matter exhaustively.

I will close with a "mot" that would be "bon" were it not so tragic. My friend has a relative who at one time was addicted to heroin. At his suggestion she tried the nitrous oxide for purposes of comparison and insisted that its effects were far better (better?) than she had ever experienced from heroin! I asked the obvious question: Why not a massive conversion from heroin and its related narcotics to the gas? No addiction (says he), consequently no withdrawal terrors; "better" results; instant recovery when necessary (if you're not dead); not illegal to buy or sell or possess; and no danger of conviction on charges of use without prescription, since recovery is so sudden and complete, and use cannot be proven post factum. Oh, and on the whole, cheaper!

His answer was, I thought, quite valid. He thinks that such a conversion would take place were it not for fear generated in the average person by a gas cylinder!

I am very gratefully yours.

Rev. Father ...., Pastor



Poikilocytosis in Cancer Patients

DONALD I. GALEN* AND WILLIAM H. KERN, M.D., *Los Angeles*

■ *The incidence of poikilocytosis in 100 patients with cancer of various organ systems was found to be 12 per cent. Poikilocytosis was more frequently seen in patients with adenocarcinoma of the gastrointestinal tract and with inoperable metastatic carcinoma than in patients with other malignant lesions. Anemia was associated in three cases, but in nine instances the patient was not significantly anemic at admission. Two of these patients may have been dehydrated. Uremia was not a factor in any of the cases.*

Fifty per cent of the patients exhibiting poikilocytosis died or were in a terminal condition within one month after the observation was made, as compared with 21 per cent of the patients who did not have evidence of poikilocytosis. These findings show that poikilocytosis may occur in cancer patients without anemia or uremia; and they indicate that its presence is an unfavorable prognosis sign.

POIKILOCYTOSIS IS A major deviation from the normal shape of erythrocytes[4] that is evidential of an increased rate of blood destruction.[7] It is particularly striking in pernicious anemia and some of the hemolytic anemias,[4] but is also characteristically found in a variety of other conditions. The occurrence of poikilocytosis of red blood cells in patients with acute renal failure and uremia has been well known for many years.[2] More recently, the occurrence of poikilocytosis has also been described in patients with cancer,[1,6] but this is still considered as unusual and is not noted in the standard textbooks of hematology.[4,7] In a recent publication, Forshaw and Harwood[3] reviewed a large number of peripheral blood smears and found that after exclusion of patients with uremia and specific hematologic diseases, 17 of 27 patients with poikilocytosis of the peripheral blood had carcinoma. Fourteen of these patients died only a few weeks after this abnormality of the blood was first observed, and in these cases unlike those reported previously, thrombocytopenia and signs of hemolysis were only rarely present. Forshaw and Harwood concluded that the presence of decided poikilocytosis is of considerable diagnostic significance and indicates a grave prognosis. It is the purpose of this study to review the incidence of poikilocytosis in a group of consecutive cancer patients and to evaluate its prognostic significance.

Material and Methods

The peripheral blood smears and various laboratory findings of 100 patients with histologically diagnosed cancer were reviewed. The material was from consecutive cases during a two-month period in the files of the Department of Pathology of the Hospital of The Good Samaritan. Excluded from the review were cases of in situ carcinoma, basal cell carcinoma, other superficial carcinomas of the

---

From the Department of Pathology, the Hospital of the Good Samaritan Medical Center and the University of Southern California School of Medicine, Los Angeles.

This study was supported by a grant from the U.S. Public Health Service, Grant No. FR-5356-05, through the USC School of Medicine.

Submitted 2 September 1966.

Reprint requests to: Hospital of the Good Samaritan Medical Center, 1212 Shatto Street, Los Angeles 90017 (Dr. Kern).

*Junior student at USC School of Medicine.

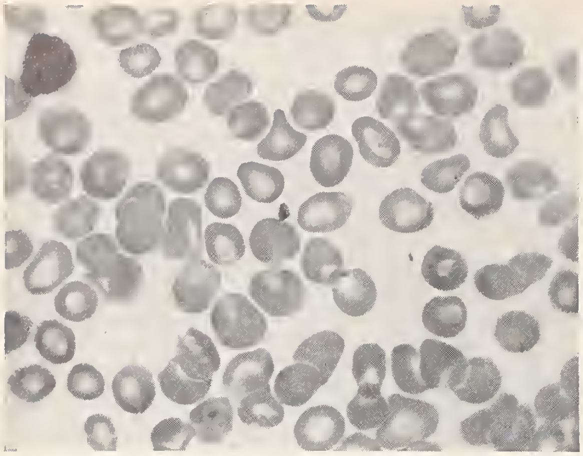


Figure 1.—Poikilocytosis, 3+, in patient with invasive adenocarcinoma of the endometrium (×1,200).

skin and leukemia. Also excluded were cases of primary hematologic disorders and those in which blood urea nitrogen exceeded 40 mg per 100 ml.

For the purpose of this study, the term poikilocytosis was defined as indicating the presence of erythrocytes with abnormal shapes, including oval, burr, sickle, oat-shaped, pencil-form, pear-shaped, tailed or fragmented forms.[5] The smears were reviewed by one author (DG) and were classified as 0 if fewer than 5 per cent of the erythrocytes were poikilocytes, 1+ if more than 25 per cent, 2+ if the proportion of poikilocytes was between 25 and 50 per cent and 3+ if more than 50 per cent. (See Figure 1.) All findings were based on laboratory work performed at the time of admission.

From review of the charts, the clinical condition of all patients was classified at a period from one to three months after admission. They were considered "improved" when, after surgical or other therapy, they were discharged from the hospital with a statement of the attending physician that their condition was improved; as "unimproved" when they were still inpatients at the hospital with disseminated disease or in a terminal state, or had been discharged as "unimproved"; or they were included in the group of fatalities if death occurred during the period of this study.

Results

The findings of the review are shown in Table 1. Carcinoma of the breast was the most common kind of cancer among the 100 patients, adenocarcinoma of the prostate was second and adenocarcinoma of the gastrointestinal tract third. In a large group of patients, metastatic carcinoma was proved by biopsy and no attempt at surgical resection was made. The average age of patients with one kind of cancer was about the same as for all others, except for patients with adenocarcinoma of the prostate, whose average age was considerably higher.

The relatively normal average hemoglobin level at the time of the admission laboratory examination is of particular interest since a higher incidence of poikilocytosis is to be expected in anemic patients. In the entire group, there were six patients who were distinctly anemic, with a hemoglobin level from 7.2 to 10.1 gm per 100 ml. Three of these six patients (with hemoglobin levels of 7.6, 7.4, and 10.1 gm per 100 ml) had poikilocytosis, from 1+ to 3+. The hemoglobin levels of the remaining nine patients with poikilocytosis were not significantly decreased, but review of the

TABLE 1.—*Data on Poikilocytosis Related to Prognosis in 100 Patients with Cancer*

Type of Cancer	*Number of Cases*	*Sex*		*Average Age (Years)*	*Average Hb (gm per 100 ml)*	*Poikilocystosis*	*Short-Term Follow-Up*	
		M	*F*				*Discharged Improved*	*Unimproved or Died*
Carcinoma of the breast	20	0	20	62.1	13.9	2	20	0
Adenocarcinoma of the prostate	15	15	0	69.0	14.2	0	15	0
Adenocarcinoma of the gastrointestinal tract	12	9	3	64.2	13.6	3	10	2
Carcinoma of the uterus (cervix and/or endometrium)	8	0	8	56.1	13.2	1	7	1
Carcinoma of the lung	5	3	2	55.8	13.7	0	4	1
Metastatic cancer, as proved by biopsy	21	12	9	60.8	13.0	4*	8	13
Other cancers	19	9	10	63.9	13.9	2†	11	8

---

*Includes two metastatic adenocarcinomas in omentum, one carcinomatosis from duct carcinoma of breast, and one undifferentiated carcinoma in mediastinal lymph node.

†Includes malignant lymphoma, primary in lung, and a papillary carcinoma of the thyroid.

clinical history and findings and the available laboratory data indicates that two of the patients, one with a hemoglobin level of 11.9 gm and the other with hematocrit of 35 per cent, were probably dehydrated. There is no indication of decreased plasma volume in the clinical histories of the remaining non-anemic patients. None of the patients with poikilocytosis were uremic, the blood urea nitrogen ranging from 12 to 22 mg per 100 ml.

Because of the possible morphologic influence of jaundice on red cells, the levels of bilirubin were also reviewed in the cases in which this determination had been done. Three patients had decidedly increased bilirubin levels (24.6, 20.4 and 9.8 mg per 100 ml) but only one of them, the one with bilirubin of 24.6 mg, had poikilocytosis, and in that case it was 1+.

The incidence of poikilocytosis was highest in patients with adenocarcinoma of the gastrointestinal tract and next highest in those with metastatic carcinoma proved by biopsy. A high incidence of poikilocytosis in patients with carcinoma of the gastrointestinal tract and abdominal metastasis was also noted by Schwartz and Motto[6] and by Forshaw and Harwood.[3]

A comparison of the short-term follow-up of all patients as related to the presence or absence of poikilocytosis is shown in Table 2. Forshaw and Harwood[3] reported that 14 of 17 cancer patients who showed poikilocytosis died within a few weeks and suggested that poikilocytosis indicates a grave prognosis. The immediate postoperative course in cancer patients with poikilocytosis was similarly poor in the present study: 50 per cent of the patients with this phenomenon either died or were in terminal condition at the conclusion of this study, as compared with 21 per cent of those who did not have poikilocytosis. Even more striking is the fact that three of the twelve patients with poikilocytosis died, as compared with four deaths in the large group of 88 patients without poikilocytosis.

TABLE 2.—*Summary of Short-term Follow-up Related to Poikilocytosis in 100 Patients with Cancer*

	Number of Cases	*Improved*	*Not Improved*	*Died*	*Per Cent Not Improved*
Poikilocytosis present	12	6	3	3	50
No poikilocytosis	88	70	14	4	21

In the evaluation of these findings, it should be noted that all blood smears were examined by one of the authors (DG) without knowledge as to the patient's clinical course. This is important because during a routine examination of the peripheral blood smears, even by experienced medical technologists, a considerable variation in interpretation must be expected. Of the 12 cases of poikilocytosis in this series, only three had been so classified during the routine examination originally, whereas in one additional case poikilocytosis had been reported, but no evidence of it could be demonstrated on reexamination.

REFERENCES

1. Brain, M. C., Dacie, J. C., and Hourihane, D. O'B.: Microangiopathic haemolytic anaemia: The possible role of vascular lesions in pathogenesis, Brit. J. Haematology, 8:358, 1962.
2. De Gruchy, G. C.: Clinical Haematology in Medical Practice, ed. 2, Oxford: Blackwell Scientific Publications, 1960.
3. Forshaw, J., and Harwood, L.: Poikilocytosis associated with carcinoma, Arch Int. Med., 117:203, February 1966.
4. Miale, J. B.: Laboratory Medicine—Hematology, p. 416, C. V. Murphy Co., St. Louis, 1962.
5. Page, L. B., and Culver, P. J.: A Syllabus of Laboratory Examinations in Clinical Diagnosis, Harvard University Press, Revised Edition, 1961, p. 60.
6. Schwartz, S. O., and Motto, S. A.: The diagnostic significance of "burr" red blood cells, Amer. J. Med. Sci., 218:563, 1949.
7. Wintrobe, M. M.: Clinical Hematology, Lea and Febiger, Philadelphia, ed. 5, 1961.

CLINICAL NOTE

THE "HIP-POINTER"

A Term to Describe a Specific Kind of Athletic Injury

MARTIN E. BLAZINA, M.D., *Los Angeles*

A TERM COMMONLY USED in athletic injuries is *hip-pointer*. Unfortunately, the injury it is used to describe actually involves the iliac crest, not the hip; and the term has also been used to designate a variety of injuries occurring in the region of the iliac crest. In order to standardize the nomenclature of athletic injuries and preserve the integrity of this term in reporting and recording *hip-pointer* should be arbitrarily restricted to a contusion of the iliac crest.*

The strains involving the various muscles attaching to the iliac crest are not to be included in this meaning and must be recorded separately. The injury causing a hip-pointer is always a direct blow, whereas muscular strains or avulsion fractures are caused by rotatory bending or twisting movements. A fracture of the iliac crest resulting from a direct blow could be ascertained by x-ray examination and diagnosed separately.

In American football a hip-pointer is usually caused by an opponent's knee, head or shoulder, with inadequate or ill-fitting hip pads sometimes being a contributing factor. A person with this injury has severe pain on movement, especially on coughing, deep breathing, sneezing or running and there may be significant disability. Exquisite localized tenderness over the iliac crest is elicited on palpation and there may be ecchymosis and swelling.

If we reserve the usage of the term *hip-pointer* to imply a contusion of the iliac crest only, clarity of reporting is maintained and a commonly used vernacular term is perfectly acceptable in athletic medical terminology.

From the Department of Surgery/Orthopedics, UCLA School of Medicine, Los Angeles.

Submitted 10 October 1966.

Reprint requests to: Department of Surgery, UCLA School of Medicine, Los Angeles 90024 (Dr. Blazina).

*Standard Nomenclature of Athletic Injuries, prepared by the A.M.A. Subcommittee on Classification of Sports Injuries, American Medical Association, Chicago, 1966.

Recent Developments in the Management of Gastrointestinal Malformations

ERIC W. FONKALSRUD, M.D., *Los Angeles*

DURING THE PAST few years we have witnessed a steady increase in attention toward improving the lot of infants with congenital malformations. Dramatic advances have been made in the treatment of infants with cardiac anomalies. Similarly, many recent investigative and clinical studies have contributed greatly in the quest for a favorable outcome for babies with serious malformations of the gastrointestinal tract. Not only have surgical principles and monitoring techniques used in adults been modified appropriately for the newborn, but numerous operative procedures and physiologic concepts have been developed. In addition surgeons have developed a more precise understanding of the pre- and postoperative metabolic requirements of these small patients. This review will present some of the recent advances and current concepts in the management of 10 major anomalies of the gastrointestinal tract with particular attention to early infancy.

Esophageal Atresia and Tracheoesophageal Fistula

An increasing awareness of the early clinical signs and symptoms of tracheoesophageal fistula has led to earlier recognition. Combined with improved surgical techniques and postoperative care, this has resulted in an impressive increase in the survival rate during the past few years.

Complete repair of the defect under general anesthesia in the first 24 hours is advisable in full-term infants unless pneumonia is present. Preoperative fluid requirements are small and overhydration should be avoided. Antibiotics should be started preoperatively. Although convenient for postoperative feedings and dilatations, a gastrostomy does not appear appreciably to alter the mortality following repair.[45] Since gaseous overdistention of the stomach as a result of resuscitative measures may cause phrenic elevation or even rupture of the stomach, gastrostomy should be used early when abdominal distention is present.[78] The survival rate with operation by the extrapleural approach (76 per cent) is somewhat higher than when the transpleural approach is used (66 per cent). This is because of the appreciable difference in survival following the development of leakage after retropleural repair (60 per cent) as compared with transpleural (32 per cent).

The double-layer anastomosis described by Haight[37] should be used when sufficient tissue is available, since leaks are less common and more benign (if they do occur) than with anastomosis of other types. Strictures, however, are more likely to occur following any type of double-layer repair. Circumferential free autogenous grafts of pericardium placed around the site of anastomosis have been reported to provide considerable early and late strength to the suture line.[47] The primary repair may be facilitated by passing a Foley catheter into the stomach through the site of anastomosis, inflating the balloon and applying gentle traction to relieve tension while the initial sutures are tied.[41]

From the Department of Surgery, UCLA School of Medicine, Los Angeles.

Dr. Fonkalsrud is a John and Mary R. Markle Scholar in Academic Medicine.

Reprint requests to: Department of Surgery, UCLA School of Medicine, Los Angeles 90024.

Short delays in primary anastomosis (two to 10 days) may be advisable when severe pneumonia, hyperbilirubinemia, hyaline membrane syndrome or sepsis is present.[39] Since pulmonary complications are by far the leading cause of death following complete repair of the anomaly (62 per cent), the lungs should be in optimal condition before operation.[45] Tube gastrostomy under local anesthesia and constant suction on the upper pouch are necessary when a delayed course is taken. The Replogle double lumen sump catheter has been helpful in keeping the upper pouch well drained.[82]

Repair should be delayed for longer periods in premature infants or when the esophageal segments are widely separated as in atresia without a fistula.[46] The fistula should be divided and the lower esophageal segment inverted and closed with two layers of sutures. Cervical esophagostomy was usually the next step until Howard[49] demonstrated that the upper pouch could be elongated by repeated pressure with a large blunt tipped bougie and thus made suitable for an easy anastomosis without tension within a few weeks. The temporary tube pharyngostomy described by Talbert and Haller[102] provides an easily manageable method of draining the upper pouch while waiting to do the second stage of the repair.

Anatomical studies indicate that the lower esophagus in this malformation is not well supplied by blood vessels and that mobilization of the distal esophageal segment for anastomosis may reduce the supply to dangerous levels.[62] Even if the anastomosis is not complicated by leakage or stricture, subsequent esophageal motility is likely to be considerably decreased if the vascular supply is compromised. Lind and associates,[60] however, suggested that the abnormal motility is a congenital abnormality of function and is not related to the operative repair.

Significant anastomotic leaks should be treated by discontinuing oral feedings and placing the stomach and esophagus at rest by gastrostomy tube aspiration, while maintaining nutrition by feedings through a duodenal catheter.[28] Small leaks may be treated by calibrated gastrostomy feedings while the infant's head is kept elevated. Mediastinal abscesses require immediate external drainage.

Anastomotic stricture should be treated by early dilatation with Tucker dilators guided by a string extending from the oral cavity out through the gastrostomy wound.[10,29] Hurst mercury-weighted bougies may be used safely from above when a size 22 F. dilator passes through the stricture.

In infants under 18 months of age, dilatation procedures usually are performed satisfactorily without anesthesia and without keeping the patient in hospital. Recalcitrant strictures should be excised. Recurrent tracheoesophageal fistulae should be closed promptly if the infant's condition permits, since these communications rarely close spontaneously.

Precise diagnostic studies usually are necessary to demonstrate tracheoesophageal fistulae of unusual types. The septal type of esophageal atresia with fistula may be suspected if the upper pouch extends unusually low.[43,61] The fistula is high in such cases and the dissection should be extended cephalad as high as the cervical trachea if necessary. The web may be excised and the esophagotomy closed transversely, or an end-to-end anastomosis may be performed.

Tracheoesophageal fistula without esophageal atresia (H-type fistula) is often not demonstrated even after numerous attempts at diagnosis by conventional techniques. Cinefluorographic esophageal studies using thin contrast material, with the patient in a prone oblique position, may demonstrate the defect. Endoscopy using a cystoscope with or without blue dye in the esophagus has been used successfully to demonstrate this lesion by Winslow and associates.[112] Occasionally air bubbles may be seen passing through the fistula into the esophagus. When the H fistula is strongly suspected but cannot be demonstrated, surgical exploration of the neck has been recommended.[72]

Routine exploration of the mediastinum in the infant with a gasless abdomen and esophageal atresia is not recommended.[57] Howard's techniques of stretching the upper pouch may permit sufficient elongation in such patients to allow anastomosis of the upper and lower segments after two or three weeks.[44] The proved method of treatment for these patients is gastrostomy and cervical esophagostomy followed by colon interposition. Complications appear to be fewer with the retrosternally placed colon than when it is placed in the chest. If the stretching method proves successful, esophageal replacement may become obsolete. Although some investigators favor immediate interposition of the colon in the neonatal period,[7,57,80] most authorities recommend a delay until the infant has attained a weight of 20 to 25 pounds. Othersen and Clatworthy[77] have shown that the interposed colon does not act as an esophageal substitute, but only as a conduit. Gravity, not peristalsis, governs the flow of ingested material regardless of whether the colon is placed in an iso- or antiperistaltic position.

Diaphragmatic Hernia

Congenital intestinal herniation through the foramen of Bochdalek in the newborn provides one of the most challenging problems in pediatric surgery. The mortality rate in infants under 48 hours of age is over 50 per cent despite modern diagnostic and operative techniques.[94] Herniations through the esophageal hiatus and foramen of Morgagni will not be reviewed here since the operative repair is straightforward and the mortality rate is low.

The newborn with respiratory distress should immediately have a chest roentgenogram. A portable x-ray machine kept in the newborn nursery will facilitate immediate examination of any infant with serious malformations without movement to remote areas of the hospital. Dyspnea, cyanosis and dextrocardia in an infant with a "wasp waisted" abdomen and tracheal deviation should lead to a prompt diagnosis of diaphragmatic hernia. The characteristic appearance of gas-filled intestine in the chest is seen on radiologic examination. Gastroinestinal contrast roentgenograms are contraindicated.

Resuscitation should be different from that commonly used for most infants with respiratory distress. Immediately after diagnosis, continuous nasogastric suction should be used. Oxygen should be administered through an endotracheal tube since a face mask may cause ingestion of gas and fatal distention of the intestine within the thoracic cavity.

Immediate operation without preoperative medications is recommended for infants with diaphragmatic hernias. Because of the danger of alveolar rupture and tension pneumothorax, the anesthesiologist should avoid overinflating the inevitably hypoplastic lungs. Respirations of flutter type (90 to 100 per minute) are desirable since the tidal volume may be as low as 3 to 4 cc. A subcostal transabdominal repair is advised for left-sided defects since other gastrointestinal anomalies such as duodenal bands are encountered in over half of infants with diaphragmatic hernia. In addition a large ventral hernia may be created by closing only the skin over the viscera when they are retrieved from the chest.[69] If an abdominal hernia is not created, severe respiratory difficulty may occur when the bowel is returned to the underdeveloped peritoneal cavity and it elevates the diaphragm. The thoracoabdominal incision recommended by Bingham[11] appears unnecessarily extensive for satisfactory repair in most patients. Although the hernia repair may be accomplished with technical ease through a thoracotomy incision, morbidity and mortality are highest when this approach is used.[34] Hernias on the right may be repaired with greater technical ease and safety through a thoracic or thoracoabdominal approach, since manipulation of the liver from below may cause hepatic injury and fatal hemorrhage.

Although most Bochdalek defects have a circumferential rim of phrenic muscle which may be sutured together with minimal tension for a satisfactory repair, several modifications of this technique have been tried on difficult cases. Holcomb recommended the formation of a hinged flap from the anterior and lateral lower thoracic wall to take the place of the absent left diaphragm.[42] A flap of muscle, fascia and peritoneum from the anterior abdominal wall has been used successfully by Rosenkrantz and Cotton.[83] Dacron mesh has been employed with success by Shaffer[87] to close the hernial defect. As the patients are critically ill, in general the principle of simple and rapid operation should be followed, with a patch used only when the defect is such that closure would be difficult without it.

Gastrostomy is recommended to prevent vomiting and to vent swallowed air. A chest tube is connected to water seal drainage to allow expansion of the lung. Chest suction is condemned since it may cause alveolar rupture and pneumothorax.[69]

Postoperative ventilatory insufficiency is due to both abdominal distention and the incompletely developed or hypoplastic lung. Most lungs function effectively in three days if the intestines are removed from the chest promptly and if the infant is given vigorous supportive treatment.[94] Pulmonary complications such as atelectasis, tension pneumothorax, hypoplasia of the lung, pneumonia and hyaline membrane syndrome are the leading causes of death in the postoperative period.

Management should include techniques used in the treatment of hyaline membrane disease, including frequent blood gas analysis and determination of pH and administration of appropriate doses of sodium bicarbonate to correct metabolic acidosis. The importance of a high humidity environment, postural drainage, sedation and nasopharyngeal suction in the postoperative period has been emphasized by Allen and Thompson.[1] Antibiotics are recommended postoperatively.

Abnormalities of Intestinal Fixation

When the mesentery of the small intestine is attached solely to the superior mesenteric vessels without the usual broad posterior stabilization, the patient is vulnerable to midgut volvulus, the most serious complication of malrotation. Such forms of malrotation may occur in an otherwise normal infant or in patients with omphalocele or posterior diaphragmatic hernia. As emphasized by White,[111] volvulus may occur in patients with normal rotation if a rudimentary mesenteric attachment is present. When nonrotation occurs, the duodenum extends from the pylorus out along the superior mesenteric vessels as the jejunum. The ascending and proximal transverse colon extend along these vessels and are closely attached to the duodenum. The duodenum, colon and mesenteric vessels are bound by peritoneum into a single pedicle which includes the intervening small bowel. Duodenal obstruction frequently results from its attachment in the mesenteric pedicle. When volvulus occurs, blood is pooled in the mesenteric bed and bowel necrosis and shock ensue rapidly. Rectal hemorrhage may occur.[21] Clinical evidence of high intestinal obstruction is usually present. Abdominal roentgenograms and roentgenograms taken with a barium enema are helpful in establishing the diagnosis. Preoperative attention should be given to hydration and electrolyte balance. Laparotomy should follow promptly with counterclockwise derotation of the pedicle.

Several investigators have observed recurrent volvulus in patients treated by the conventional Ladd procedure.[2,54,59] Bill and Grauman[9] recommended that, in addition to the freeing of the upper small bowel and colon from the peritoneal covering, the mesentery be stabilized by fixation of the cecum and its mesentery to the left colon and suture of the duodenum to the right kidney. All duodenal bands must be completely released. Tube gastrostomy is advised in such cases. When intestinal viability is in doubt, injection of a local anesthetic about the mesenteric pedicle may cause improvement in vascularity. Intravenous low molecular weight dextran may bring about some improvement in blood flow to the midgut in such patients. Smith and associates[93] showed that hyperbaric oxygenation may reduce mortality 50 per cent in rats with simulated volvulus.

Chronic duodenal ileus, observed in patients beyond early infancy by Louw,[63] represents a mild form of duodenal obstruction due to peritoneal bands with or without rotational anomalies. It should be corrected by division of the bands.

Intestinal Duplications

Duplication is a rare anomaly of still undetermined embryologic origin which may arise at any point along the alimentary tract, although it is most common in the distal small intestine.[13] Duplications may be spherical or tubular, may or may not communicate with an adjacent segment of intestine or other viscus, and may vary widely in size. Spherical duplications are usually found along the esophagus and upper intestine, do not communicate, and cause symptoms by compression. Tubular duplications are longer and usually communicate at one or both ends. When there is only a proximal opening, the duplication may fill but be unable to empty and thus increase greatly in size, causing obstruction or pain.[70] Small intramural duplications may become the point of origin of intussusception.

Duplications lie within the mesentery and frequently have a common muscularis as well as blood supply with the adjacent bowel. Heterotopic mucosa is frequently present and may cause hemorrhage or ulceration. Other visceral anomalies and also anomalies of the vertebral column, are frequently associated with duplications. Multiple duplications are occasionally encountered.[13] Duplications of the primitive hindgut are much more complex and fortunately uncommon.[5] Gastric carcinoma may occasionally occur in the heterotopic mucosa of duplications.[71]

Almost all duplications should be excised,[48] although the surgical procedure should not be more radical than is necessary to eliminate the symptoms and prevent future complications. Usually, spherical duplications may be completely excised. For tubular duplications it may be necessary to excise the lesion together with adjacent bowel and perform an end-to-end anastomosis or Mikulicz enterostomy. Long tubular duplications may be treated by a distal "windowing" operation to establish continuity with the intestine, although future symptoms may result from the residual heterotopic mucosa. Mucosal stripping operations[113] and marsupialization, although more likely to relieve symptoms, are associated with higher morbidity and are not generally necessary.[13]

Intestinal Atresia

Congenital atresia of the intestine has been regarded as an embryonal malformation due to failure of recanalization of the gut at the end of the second month of fetal life. Recent embryologic and pathologic studies suggest rather that true plugging of the lumen must be a rare phenomenon confined to the duodenum, if it occurs at all.[14,73] Current opinion leans to the view that intussusception, injury or vascular accident to the fetal intestine are the probable etiologic factors.[4]

The three recognized types of atresia are the diaphragm occlusion, the blind proximal end without intestinal continuity, and the multiple cord-like segments. In the last two types there may be an associated gap in the mesentery.[85] Approximately 15 per cent of patients have multiple atresias.[20] Benson and associates[6] called attention to the considerably shortened length of bowel in infants with atresia.

Operative correction should be undertaken shortly after the diagnosis has been made. A search for multiple lesions should always be made. Intraluminal injections of methylene blue dye and mineral oil with observation of its distal progress will facilitate the search. End-to-side intestinal anastomosis is inadvisable because of subsequent development of blind loop segments and associated nutritional problems.[50] End-to-end or end-to-side anastomosis is recommended, with resection of the proximal 10 to 15 cm of dilated bowel.[64] A two-layer anastomosis with an outer row of silk and inner row of catgut is advised.[97] The anastomosis may be facilitated by clamping the distal collapsed segment 1 inch distal to the site of atresia with an atraumatic clamp and injecting air with a fine needle to produce distention.

The multi-staged Mikulicz resection is favored for premature infants and those with severe associated abnormalities. Tube gastrostomy is recommended for most infants with intestinal obstruction.[17] Gerrish[31] advocated decompression of the anastomotic site by a small polyethylene tube inserted in the bowel 10 cm proximal to the suture line. Ducharme[25] recommended a similar tube passed through the appendix in patients with ileal atresia. A favorable outcome may be anticipated in many of the patients despite the absence of large segments of small intestine.[18] Postoperative diarrhea may frequently be minimized by rigid restriction of fat intake.[38] Vagotomy or reversal of peristalsis in an isolated intestinal segment may be necessary for intractable diarrhea.

Meckel's Diverticulum

Meckel's diverticulum has been recognized as a pathologic entity for more than 150 years,[108] yet several features have been emphasized only recently in reviews on the subject. The 2 per cent incidence, most often in males, makes it the most frequent malformation of the gastrointestinal tract.[96] Most diverticula are 1 to 5 cm in length, 1 to 2 cm in diameter and occur within the 91 cm proximal to the ileocecal valve on the antimesenteric border.[108] Occasional diverticulae are found on the mesenteric side and may be difficult to identify.[96] The lesion is frequently found in association with other congenital malformations. Although the normal lining of the diverticulum is intestinal mucosa, heterotopic gastric mucosa is found in from 15 to 60 per cent of cases.[108]

Complications of Meckel's diverticulum, usually occurring beyond infancy, are a cause for surgical intervention in more than 22 per cent of cases.[107] The symptomatic diverticulum may become apparent in any of the following ways: Hemorrhage, persistent omphalomesenteric sinus which may result in volvulus, inclusion in a hernial sac, intussusception, inflammation which may result in perforation, intestinal obstruction and neoplasms of either benign or malignant behavior.[15,108] The heterotopic gastric mucosa is usually an underlying cause for both hemorrhage and inflammation. Recently attention has been directed to intestinal obstruction which may be caused by herniation around mesodiverticular bands.[84] Meckel's diverticulum must be considered as a possible cause of any intra-abdominal condition which cannot be readily diagnosed. Radiographic studies are frequently of little value in establishing the diagnosis.

Although this is not a universal opinion, many physicians recommend search for and removal of all Meckel's diverticula,[36,51] although not in conjunction with other major abdominal surgical procedures or during operations for removal of an acutely inflamed appendix.[81] Diverticula with a thin pedicle may be amputated transversely or obliquely, whereas larger lesions may be removed most safely by excising a segment of small intestine.

Meconium Ileus

Cystic fibrosis is a hereditary disorder which affects the exocrine glands, sweat glands and mucus-secreting glands of the bronchial tree. Increased electrolyte content in sweat, measured by pilocarpine iontophoresis, usually establishes the diagnosis. Amounts above 60 mEq per liter are usually diagnostic and those below 40 mEq usually exclude the diagnosis. Duodenal and stool trypsin analysis may be of some diagnostic value. Parkins and coworkers[79] recently advocated rectal biopsy with identification of characteristic dilated mucus-packed crypts as an accurate diagnostic technique. Shwachman[90] showed a characteristic increase in sodium and potassium concentration in nail clippings from patients with cystic fibrosis.

The most serious secondary manifestation of fibrocystic disease is meconium ileus, which occurs in 10 to 15 per cent of cases.[22,74] This form of obstruction may be associated with bowel atresia, stenosis, volvulus, meconium peritonitis or other congenital malformations.[76] Atresia of the bowel is associated with cystic fibrosis in about 25 per cent of cases.[3]

The development of meconium ileus appears to depend largely upon alterations of the mucosal glands in the small intestine. Pancreatic lesions seem to play only a secondary role if any at all.[105] Except for meconium ileus the other gastrointestinal manifestations of cystic fibrosis appear to depend primarily upon pancreatic achylia.

The clinical features of meconium ileus in the newborn are vomiting, or regurgitation of bilious material, abdominal distention and scantiness or complete absence of stools. The characteristic roentgenographic findings are a few distended loops of small intestine of various sizes with a generalized "ground glass" or "soap bubble" appearance which is produced by small bubbles of gas scattered throughout the meconium in the distal portion of the small intestine.[66] White[110] observed the absence of fluid levels on upright roentgenograms of the abdomen. If the diagnosis remains in doubt, roentgen studies after an enema of thinned barium will show an "unused" colon.

Clatworthy and Lloyd[17] indicated that operation may be inadvisable in a small proportion of infants who have a mild form of the disease. Enterotomy followed by irrigation and closure has been recommended by Olim and Ciuti[75] and is suitable for patients in whom the obstructed segment of the intestinal wall appears to be of normal thickness and vascularity. If the obstructed segment has been irreversibly damaged by perforation or by severe stenosis or atresia, Gross[35] and Welch[109] prefer the Mikulicz resection with double-barreled ileostomy, whereas Swenson[100] recommends primary resection with end-to-end ileoileostomy. Bishop and Koop[12] have used a Roux-en-Y ileoileostomy, exteriorizing the distal end of the loop as a mucous fistula for subsequent irrigation with Viokase® or hydrogen peroxide. This method necessitates a secondary laparotomy to remove the fistula but obviates the electrolyte imbalance which may occur after the procedure described by Gross and Welch. Recently Meeker and Kincannon[68] recommended Mucomyst® for irrigation of the small intestine impacted with meconium.

Complications after operation for meconium ileus include peritonitis, volvulus, intussusception and obstruction secondary to adhesions. Peritonitis usually results from contamination at the time of operation or leakage from the suture line or cutaneous enterostomy site. Welch[109] recommended that the intestine be sutured to the peritoneum at the site of ileostomy to guard against prolapse or retraction.

Gastrostomy is recommended to allow effective intestinal decompression. Broad spectrum antibiotics should be given for peritoneal contamination and to prevent respiratory complications. A high mist environment with frequent postural drainage treatments are necessary to minimize pulmonary infection. Instillation of pancreatic extracts by nasogastric tube or by enema may help to clear meconium from the intestine.

Malrotation with or without midgut volvulus has been observed to occur in one-third of infants with meconium ileus.[33] Meconium peritonitis, or in utero intestinal perforation without bacterial contamination, is caused by meconium ileus in 60 per cent of cases.[23] Intussusception may occur in older infants with cystic fibrosis, presumably because of the abnormal consistency of the stool.

Continued administration of pancreatic enzymes is usually necessary to prevent subsequent obturant obstruction and to facilitate intestinal absorption necessary for satisfactory growth and development.

Meconium Plug Syndrome

Transient obturation obstruction due to inability of the distal intestinal tract to rid itself spontaneously of mucoid anorectal plugs is not uncommon and is usually cured by gentle dilatation of the anal orifice. The obstruction caused by the meconium plug syndrome may be indistinguishable from other serious forms of distal obstruction in the newborn, and it is not relieved by anal dilatation.[16]

When low intestinal obstruction in the newborn is suspected, a barium enema should be cautiously administered, using a thin solution introduced under low pressure through a large soft-tipped catheter.[30] Characteristic roentgenographic findings of a radiolucent filling defect in the left colon with a distal microcolon have been described by Ellis and Clatworthy.[27] Differentiation from Hirschsprung's disease on the initial examination may be impossible. After the diagnostic enema, the procedure should be repeated, using a mixture of one part hydrogen peroxide and three parts of a dilute mixture of normal saline solution and barium.[75] This maneuver apparently breaks down the surface tension between the obturant mass of meconium and the mucosal surface of the colon by forming tiny bubbles of gas in this interface. The meconium plug usually passes spontaneously following this procedure and permanently relieves the obstruction. A recent report by Danis indicates that precaution is advised when hydrogen peroxide is used for enemas in infants, since gas embolism has been observed to occur.[19]

Laparotomy is rarely indicated and then only for the uncommon ileal plug which cannot be dislodged with repeated peroxide enemas. A transient ileostomy with manual removal of such plugs has been advocated by Ellis and Clatworthy.[27]

The cause of the meconium plug syndrome remains unknown, but no association with cystic fibrosis has been reported. Neonatal Hirschsprung's disease may mimic the syndrome and if subsequent bowel evacuation is not normal, a repetition of barium enema studies and possibly a rectal muscle biopsy are indicated to exclude the diagnosis of aganglionic megacolon.[32]

Hirschsprung's Disease

Congenital intestinal aganglionosis is confined to the rectum or descending colon in more than 90 per cent of patients and commonly produces symptoms of low intestinal obstruction beginning in early infancy.[40] The incidence is four times as high in males as in females. The aganglionic segment is likely to extend higher in females than in males. Total colonic aganglionosis is an uncommon but more lethal form of the disease.[40]

Megacolon in infancy and childhood may be due to a number of causes and the proper treatment depends upon the accuracy of the diagnosis. Kottmeier and Clatworthy[58] indicated that the conventional symptoms of distention, emesis, obstipation and diarrhea may be indistinguishable in patients with aganglionic and functional megacolon, and they also noted that barium enema is not uniformly diagnostic, especially in infants in whom the characteristic "pigtail" deformity may not be present. The time-tested method of accurately diagnosing Hirschsprung's disease is a rectal muscle biopsy performed under general anesthesia Smith[92] called attention to the progressive histologic changes in ganglion cells observed with maturation from the fetal state to the adult. In the diagnosis of Hirschsprung's disease the importance of employing a pediatric pathologist familiar with these changes has been emphasized by Shim and Swenson.[89] Dobbins and Bill[24] recently had success in diagnosing Hirschsprung's disease by suction biopsy of the rectal mucosa obtained without anesthesia. The procedure is painless, easy and safe, and it is an accurate method of demonstrating submucosal ganglion cells.

Enterocolitis is a common serious complication which is the leading cause of death in patients with this malformation and is almost never seen in patients with functional megacolon.[8] The pathogenesis of this condition is apparently related to partial obstruction of the intestine by the aganglionic segment. The disease is characterized by abdominal distention, pain, explosive watery diarrhea, fever and prostration from acute water and electrolyte loss. Enteric pathogens are rarely isolated from the stools of these patients. Shim and Swenson[89] recommend small rectal irrigations of saline solution at least three times daily to remove intestinal gas and liquid and to allow increased water absorption until the colon returns to normal caliber. Antibiotics are not advocated by these authorities.

Bill and Chapman[8] emphasized that the best treatment of enterocolitis is to prevent it by colostomy in infancy as soon as the diagnosis of Hirschsprung's disease is made. These investigators observed a 33 per cent mortality rate when colostomy was not performed until enterocolitis appeared, as contrasted to the 4 per cent mortality rate when colostomy was performed immediately after Hirschsprung's disease was diagnosed. Colostomy may not always cure enterocolitis once it is established.

Traditionally the treatment of an infant with symptomatic aganglionosis proved by rectal biopsy is a diverting colostomy with the stoma placed at the proximal end of the aganglionic segment. Accurate frozen section analysis is imperative if the stoma is to be placed in the proper position. This is followed by the second stage proctectomy and pull-through anastomosis when the infant reaches a weight of 20 to 25 pounds.[101] For small infants with aganglionosis who are not emaciated, Shim and Swenson[89] recently advocated primary resection without preparatory colostomy. Most pediatric surgeons continue to use the Swenson pull-through procedure for definitive treatment of the disease. Emphasis is placed on meticulous pelvic dissection, with care taken to confine the dissection to the rectal wall to avoid injury to the pelvic nerves. A 1 cm cuff of rectum is left posteriorly and 2 cm anteriorly. The anastomosis should not be performed under tension. In event of an anastomotic leak colostomy should be done immediately.[99] If enterocolitis occurs after resection, anal sphincterotomy and rectal irrigations are recommended.

Recent experience with the Duhamel modification of the Swenson operation has shown that serious complication is less likely and it is easier to perform technically.[26,91] The uncertainty of the fate of the rectal pouch is the main reservation to general acceptance of this now commonly performed procedure. The Duhamel modification has been modified further by Martin and Altemeier to remove the blind fecal reservoir.[67] Talbert and coworkers have shown that the Duhamel spur may be easily divided primarily with an anastomotic stapling instrument.[103] Another procedure which is gaining wider popularity is the Soave modification of the Rehbein rectal mucosa stripping and colonic pull-through procedure.[95] This operation may be performed rapidly and safely in infants of all sizes.[104] Rectal myomectomy has recently been used effectively without complications by Lynn[65] in patients who have had a poor response to low anterior resection or a pull-through procedure. It is also recommended in patients with short segment aganglionosis.

Imperforate Anus

Stimulated by the high incidence of incontinence following operation for correction of imperforate anus in infants, significant changes in management have evolved during the past few years.[56] A more accurate understanding of the anal sphincter mechanism in patients with this anomaly has come from the extensive dissections on infant cadavers by Stephens[98] and Kiesewetter.[53]

The critical factor in the diagnosis, treatment and prognosis of imperforate anus is the relationship of the rectum to the puborectalis sling which controls continence in these patients. When the rectal pouch descends below the pubococcygeal line as seen on Wangensteen-Rice roentgenograms,[106] the rectum usually has passed through the sling and a perineal anoplasty may be performed at birth with expectation that continence will be maintained in most instances. In males a rectoperineal fistula, perineal raphe, covered anus or anteriorly placed ectopic anus is usually found in association with low rectal pouches or so-called anal deformities. If meconium is found in the urine, even in low deformities, it is evidence that a rectourethral fistula is present; and for this the safest management is a preliminary transverse colostomy, to be followed a few months later by urinary tract investigation and careful anoplasty.[98]

When rectum terminates above the pubococcygeal line, there is almost invariably a urethral or vesical fistula. In these patients a diverting colostomy at birth is advised, this to be followed by a rectoplasty at age one to one and a half years. Hyperchloremic acidosis may result from persistent urinary drainage through the fistula, and in that event repair would have to be done earlier.[88] The three-incision approach for rectoplasty is recommended by Stephens[98]: First a small sacrococcygeal incision to accurately identify and mobilize the puborectalis sling, followed by an abdominal incision to mobilize the rectum and divide the urinary fistula, and then a perineal incision at the site of the anus. The rectum is brought through the puborectalis sling and anastomosis with the perineal skin is carried out. A modification has recently been advocated by Kiesewetter[52] in which the mucosa is stripped from the isolated rectal pouch below the peritoneal reflection without closing the urinary fistula and the rectosigmoid is then brought through the rectal tunnel and puborectalis sling, using the three-incision approach. This operation avoids extensive perineal dissection and possible nerve injury. A buttock-reflecting incision has recently been advocated by Sauvage and Bill[86] for high anal malformations to obviate the abdominal incision.

In females a classification of communicating anal malformations depends on the number of orifices which open into the vulva and perineum: (1) Rectocloacal fistula with one orifice in the vulva common to urethra and vagina and rectum should be treated with colostomy at birth, (2) rectovaginal fistula with two vulvar orifices (urethra and vagina) should initially be treated by colostomy, (3) ectopic anus, rectovestibular fistula or covered anus with three orifices (urethra, vagina and bowel) may be treated by local anoplasty, except for the ectopic anus which requires no surgical treatment. In the rare cases in which a fistula cannot be found, a perineal repair may be done for low pouches seen on roentgenograms as in the male, and a preliminary colostomy performed when the pouch is higher. Choice of subsequent repair following colostomy will depend upon the relationship of the rectal pouch to the puborectalis sling, as in the male.

Detailed investigation of the urinary and gastrointestinal tract is advised in all patients with anal malformations because of the 48 per cent incidence of associated anomalies.[56] More than one-fourth of patients with such malformations have anomalies of the sacrum, and most of them do not have normal innervation of the levator ani muscles, with the consequence that they may have both urinary and fecal incontinence, depending upon the extent of the neurologic deficit.[56]

If constipation persists following repair even though the anal orifice is adequate, a rectal biopsy should be performed, since there is a 3.4 per cent incidence of associated aganglionosis.[55] Bowel control may not be achieved for several years following surgical repair in many patients, and reassurance is advisable.

Generic and Trade Names of Drugs

Pancreatin—*Viokase*®
Acetylcysteine—*Mucomyst*®

REFERENCES

1. Allen, M. S., and Thomson, S. A.: Congenital diaphragmatic hernia in children under one year of age: A 24-year review, J. Pediat. Surg., 1:157-161, April 1966.

2. Allen, R. P., Akers, D. R., and Shopfner, C. E.: Midgut volvulus, Radiology, 74:784-789, May 1960.

3. Andersen, Dorothy H.: Pathology of cystic fibrosis, Ann. N.Y. Acad. Sci., 93:500-517, 15 August 1962.

4. Barnard, C. N.: The genesis of intestinal atresia, Surg. Forum, 7:393-394, 1956.

5. Beach, P. D., Brascho, D. J., Hein, W. R., Nicol, W. W., and Geppert, L. J.: Duplication of the primitive hindgut of the human being, Surgery, 49:779-792, June 1961.

6. Benson, C. D., Lloyd, J. R., and Smith, D. J.: Resection and primary anastomosis in the management of stenosis and atresia of the jejunum and ileum, Pediatrics, 26:265-272, August 1960.

7. Bentley, J. F. R.: Primary colonic substitution for atresia of the esophagus, Surgery, 58:731-736, October 1965.

8. Bill, A. H., and Chapman, N. D.: The enterocolitis of Hirschsprung's disease; its natural history and treatment, Amer. J. Surg., 103:70-74, January 1962.

9. Bill, A. H., and Grauman, D.: Rationale and technique for stabilization of the mesentery in cases of nonrotation of the midgut, J. Pediat. Surg., 1:127-136, April 1966.

10. Bill, A. H., Mebust, W. K., and Sauvage, L. R.: Evaluation of techniques of esophageal dilatation in relation to the danger of perforation, J. Thorac. Cardiov. Surg., 45:510-514, April 1963.

11. Bingham, J. A. W.: Herniation through congenital diaphragmatic defects, Brit. J. Surg., 47:1-15, July 1959.

12. Bishop, H. C., and Koop, C. E.: Management of meconium ileus: Resection, roux-en-Y anastomosis, and ileostomy irrigation with pancreatic enzymes, Ann. Surg., 145:410-414, March 1957.

13. Bishop, H. C., and Koop, C. E.: Surgical management of duplications of the alimentary tract, Amer. J. Surg., 107:434-442, March 1964.

14. Blanc, W. A., Santulli, T. V., and Andersen, D. H.: Pathogenesis of jejunoileal atresia, Amer. J. Dis. Child., 98:564-565, November 1959.

15. Classen, K. L., Wiederanders, R. E., and Symbas, P. N.: Meckel's diverticulum associated with carcinoid tumor, Surgery, 49:328-330, March 1961.

16. Clatworthy, H. W., Jr., Howard, W. H. R., and Lloyd, J.: The meconium plug syndrome, Surgery, 39: 131-142, January 1956.

17. Clatworthy, H. W., Jr., and Lloyd, J. R.: Intestinal obstruction of congenital origin: A study of diagnosis and management in 163 cases, Arch. Surg., 75:880-890, December 1957.

18. Clatworthy, H. W., Jr., Salerby, R., and Lovingood, C.: Extensive small bowel resection in young dogs: Its effect on growth and development, Surgery, 32:341-349, August 1952.

19. Danis, R. K.: Surgical hazards with hydrogen peroxide, presented at Surgical Section of the American Academy of Pediatrics meeting, October 1966.

20. Davis, D. L., and Poynter, C. W. M.: Congenital occlusions of the intestines; with report of a case of multiple atresia of the jejunum, Surg. Gynec. Obstet., 34: 35-41, January 1922.

21. Del Junco, T., and Franco, R.: Midgut volvulus with rectal hemorrhage in the newborn, Ann. Surg., 147: 112-115, January 1958.

22. Dennison, A. B., Shwachman, H., and Gross, R. E.: A review of 164 children with meconium ileus, Pediatrics, 37:833-850, May 1966.

23. Di Sant'Agnese, P. A., and Lepore, M. J.: Involvement of abdominal organs in cystic fibrosis of the pancreas, Gastroenterology, 40:64-74, January 1961.

24. Dobbins, W. O., and Bill, A. H.: Diagnosis of Hirschsprung's disease excluded by rectal suction biopsy, New Engl. J. Med., 272:990-993, 1965.

25. Ducharme, J.: Ileal atresia of the newborn, Amer. J. Surg., 101:688-691, May 1961.

26. Ehrenpreis, T., Livaditis, A., and Okmian, L.: Results of Duhamel's operation for Hirschsprung's disease, J. Pediat. Surg., 1:40-46, February 1966.

27. Ellis, D. G., and Clatworthy, H. W., Jr.: Meconium plug syndrome revisited, J. Pediat. Surg., 1:54-61, February 1966.

28. Eraklis, A. J., and Gross, R. E.: Esophageal atresia—management following an anastomotic leak, Surgery, 60:919-923, October 1966.

29. Fonkalsrud, E. W.: Initial esophageal dilatation in infants with benign esophageal stricture, Surgery, 59:883-885, May 1966.

30. Fonkalsrud, E. W., and Clatworthy, H. W., Jr.: Accidental perforation of the colon and rectum in newborn infants, New Engl. J. Med., 272:1097-1100, 27 May 1965.

31. Gerrish, E. W.: Operative management of congenital intestinal atresia, Ann. Surg., 142:469-477, September 1955.

32. Gillis, D. A., and Grantmyre, E. B.: Meconium plug syndrome and Hirschsprung's disease, Canad. Med. Assn. J., 92:225-227, 30 January 1965.

33. Graham, W. P., Halden, A., and Jaffe, B. F.: Surgical treatment of patients with cystic fibrosis, Surg. Gynec. Obstet., 122:373-381, February 1966.

34. Grewe, H. E., and Ney, R.: Spätergebnisse operierter zwerchfelldefekte im säuglingsalter, Zschr. Kinderchir., 2:431-440, October 1965.

35. Gross, R. E.: The Surgery of Infancy and Childhood, W. B. Saunders Co., Philadelphia and London, 1953.

36. Gremmel, H.: Das meckelshe divertikel seine gefahren und komplikationen, Med. Welt, 294-297, 10 February 1962.

37. Haight, C., and Townsley, H. A.: Congenital atresia of the esophagus with tracheoesophageal fistula: Extrapleural ligation of fistula and end-to-end anastomosis of esophageal segments, Surg. Gynec. Obstet., 76:672-688, June 1943.

38. Handelsman, J. C., Abrams, S., and Corry, R. J.: Improvement of therapy for congenital jejunoileal atresia, Surg. Gynec. Obstet., 117:691-702, December 1963.

39. Hays, D. M., Woolley, M. M., and Snyder, W. H.: Changing techniques in the management of esophageal atresia, Arch. Surg., 92:611-616, April 1966.

40. Hermann, R. E., Izant, R. J., and Bolande, R. P.: Aganglionosis of the intestine in siblings, Surg., 53:664-669, May 1963.

41. Hertzler, J. H.: Congenital esophageal atresia; problems and management, Amer. J. Surg., 109:780-787, June 1965.

42. Holcomb, G. W.: A new technique for repair of congenital diaphragmatic hernia with absence of the left hemidiaphragm, Surg., 51: 534-540, April 1962.

43. Holcomb, G. W., and Daniel, R. A.: Unusual tracheoesophageal fistulae with membranous atresia of the middle or distal esophagus, Surg., 59:1112-1119, June 1966.

44. Holder. T. M., and Ashcraft, K. W.: Esophageal atresia and tracheoesophageal fistula, Curr. Probl. Surg., August 1966.

45. Holder, T. M., Cloud, D. T., Lewis, J. E., and Pilling, G. P.: Esophageal atresia and tracheoesophageal fistula; a survey of its members by the Surgical Section of the American Academy of Pediatrics, Pediatrics, 34: 542-549, October 1964.

46. Holder, T. M., McDonald, V. G., and Woolley, M. M.: The premature or critically ill infant with esophageal atresia: Increased success with a staged approach, J. Thorac. Cardiov. Surg., 44:344-355, September 1962.

47. Hopper, C. L., Berk, P. D., and Howes, E. L.: Strength of esophageal anastomoses repaired with autogenous pericardial grafts, Surg. Gynec. Obstet., 117:83-86, July 1963.

48. Houston, H. E., and Lynn, H. B.: Duplications of the small intestine in children: Mayo Clinic experience and review of the literature, Mayo Clin. Proc., 41:246-256, April 1966.

49. Howard, R., and Myers, N. A.: Esophageal atresia: A technique for elongating the upper pouch, Surg., 58:725-727, October 1965.

50. Hurwitz, A.: Reappraisal of side-to-side and end-to-side intestinal anastomoses, Surg., 43:864-866, May 1958.

51. Johns, T. N. P., Wheeler, J. R., and Johns, F. S.: Meckel's diverticulum and Meckel's diverticulum disease: A study of 154 cases, Ann. Surg., 150:241-256, August 1959.

52. Kiesewetter, William: Imperforate anus: The technique of the sacroabdominoperineal pull-through approach, presented at the Surgical Section of the American Academy of Pediatrics, 20 to 23 October 1966.

53. Kiesewetter, W.: Personal communication.

54. Kiesewetter, W. B., and Smith, J. W.: Malrotation of the midgut in infancy and childhood, Arch. Surg., 77: 483-491, Otocber 1958.

55. Kiesewetter, W. B., Sukarochana, K., and Sieber, W. K.: The frequency of aganglionosis associated with imperforate anus, Surg., 58:877-880, November 1965.

56. Kiesewetter, W. B., Turner, C. R., and Sieber, W. K.: Imperforate anus: Review of a 16-year experience with 146 patients, Amer. J. Surg., 107:412-421, March 1964.

57. Koop, C. E., and Hamilton, J. P.: Atresia of the esophagus: Increased survival with staged procedures in the poor-risk infant, Ann. Surg., 162:389-400, September 1965.

58. Kottmeier, P. K., and Clatworthy, H. W., Jr.: Aganglionic and functional megacolon in children—a diagnostic dilemma, Pediatrics, 36:572-582, October 1965.

59. Ladd, W. E.: Surgical diseases of the alimentary tract in infants, New Engl. J. Med., 215:705-708, 15 October 1936.

60. Lind, J. F., Blanchard, R. J., and Ferguson, C. C.: Abnormality of esophageal motility associated with esophageal atresia and tracheoesophageal fistula, presented at the American Academy of Pediatrics meeting, 22 to 23 October 1966.

61. Lister, J.: An unusual variation of oesophageal atresia, Arch. Dis. Childhood, 38:176-179, April 1963.

62. Lister, J.: The blood supply of the oesophagus in relation to oesophageal atresia, Arch. Dis. Childhood, 39:131-137, April 1964.

63. Louw, J. H.: Intestinal malrotation and duodenal ileus, J. Roy. Coll. Surg., Edinb., 5:101-126, January 1960.

64. Louw, J. H.: Jejunoileal atresia and stenosis, J. Pediat. Surg., 1:8-23, February 1966.

65. Lynn, H.: Rectal myectomy for aganglionic megacolon, Mayo Clin. Proc., 41:289-295, May 1966.

66. MacDonald, J. A., and Trusler, G. A.: Meconium ileus, Canad. Med. Assn. J., 83:881-885, 22 October 1960.

67. Martin, L. W., and Altemeier, W. A.: Clinical experience with a new operation (modified Duhamel procedure) for Hirschsprung's disease, Ann. Surg., 156:678-681, October 1962.

68. Meeker, I. A., and Kincannon, W. N.: Acetylcysteine used to liquefy inspissated meconium causing intestinal obstruction in the newborn, Surg., 56:419-425, August 1964.

69. Meeker, I. A., and Kincannon, W. N.: The role of ventral hernia in the correction of diaphragmatic defects in the newborn, Arch. Dis. Childhood, 40:146-153, April 1965.

70. Mellish, R. W. P., and Koop, C. E.: Clinical manifestations of duplication of the bowel, Pediatrics, 27:397-407, March 1961.

71. Micolonghi, T., and Meissner, G. F.: Gastric-type carcinoma arising in duplication of the small intestine, Ann. Surg., 147:124-127, January 1958.

72. Moncrief, J. A., and Randolph, J. G.: Congenital tracheoesophageal fistula without atresia of the esophagus, J. Thorac. Cardiov. Surg., 51:434-442, March 1966.

73. Moutsouris, C.: The "solid-stage" and congenital intestinal atresia, J. Pediat. Surg., 1:446-450, October 1966.

74. O'Flynn, M. E. D., and Rowley, W. F.: Development of pancreatic function and cystic fibrosis, Pediat. Clin. N. Amer., 12:655-662, August 1965.

75. Olim, C. B., and Ciuti, A.: Meconium ileus; a new method of relieving obstruction; report of two cases with successful management, Ann. Surg., 140:736-740, November 1954.

76. Oppenheimer, E. H., and Esterly, J. R.: Observations in cystic fibrosis of the pancreas. II. Neonatal intestinal obstruction, Bull. Johns Hopkins Hospital, 111:1-13, July 1962.

77. Othersen, H. B., and Clatworthy, H. W., Jr.: Functional evaluation of childhood esophageal replacement, J. Thorac. Cardiov. Surg., (In press).

78. Othersen, H. B., and Gregorie, H. B.: Pneumatic rupture of the stomach in a newborn infant with esophageal atresia and tracheoesophageal fistula, Surg., 53:362-367, March 1963.

79. Parkins, R. A., Eidelman, S., Rubin, C. E., Dobbins, W. O., and Phelps, P. C.: The diagnosis of cystic fibrosis by rectal suction biopsy, Lancet, 2:851-856, 26 October 1963.

80. Pettersson, G.: Experiences in oesophageal reconstruction, Arch. Dis. Childhood, 37:184-189, April 1962.

81. Reichmann, J.: Seltene komplikationen durch Meckelsche divertikel, Zbl. Chir. Leipzig, 84:1725-1731, 17 October 1959.

82. Replogle, R. L.: Esophageal atresia: Plastic sump catheter for drainage of the proximal pouch, Surg., 54: 296-297, August 1963.

83. Rosenkrantz, J. G., and Cotton, E. K.: Replacement of left hemidiaphragm by a pedicled abdominal muscular flap, J. Thorac. Cardiov. Surg., 48:912-920, December 1964.

84. Rutherford, R. B., and Akers, D. R.: Meckel's diverticulum: A review of 148 pediatric patients with special reference to the pattern of bleeding and to mesodiverticular bands, Surg., 59:618-626, April 1966.

85. Santulli, T. V., and Blanc, W. A.: Congenital atresia of the intestine: Pathogenesis and treatment, Ann. Surg., 154:939-948, December 1961.

86. Sauvage, L. R., and Bill, A. H.: Imperforate anus repair through buttock-reflecting incision, Surg., 57:448-453, March 1965.

87. Shaffer, J. O.: Prosthesis for agenesis of the diaphragm, J.A.M.A., 188:1000-1002, 15 June 1964.

88. Shepard, R., and Kiesewetter, W. B.: Hyperchloremic acidosis as a complication of imperforate anus with recto-urinary fistula, J. Pediat. Surg., 1:62-69, February 1966.

89. Shim, W. K. T., and Swenson, O.: Treatment of congenital megacolon in 50 infants, Pediatrics, 38:185-192, August 1966.

90. Shwachmann, H.: Nail parings trim doubt in diagnosis, Med. World News, 30 April 1965, p. 35.

91. Sieber, W. K., and Kiesewetter, W. B.: Duhamel's operation for Hirschsprung's disease, Arch. Surg., 87:111-119, July 1963.

92. Smith, B.: Personal communication.

93. Smith, B., Okamoto, Leal, E., and Clatworthy, H. W., Jr.: Hyperbaric oxygen in the treatment of volvulus of the midgut: An experimental study. Presented at the Surgical Section of the American Academy of Pediatrics, 20 to 23 October 1966.

94. Snyder, W. H., and Greaney, E. M.: Congenital diaphragmatic hernia; 77 consecutive cases, Surg., 57: 576-588, April 1965.

95. Soave, F.: A new surgical technique for treatment of Hirschsprung's disease, Surg., 56:1007-1014, November 1964.

96. Sommers, L. A.: Meckel's diverticulum: An unusual position, Surg., 49:331-333, March 1961.

97. Stanley-Brown, E. G.: Congenital atresia of the small intestine, Amer. J. Surg., 104:448-457, September 1962.

98. Stephens, F. D.: Congenital Malformations of the Rectum, Anus, and Genitourinary Tracts, E. S. Livingstone, Ltd., Edinburgh, London, 1963.

99. Swenson, O.: Follow-up on 200 patients treated with Hirschsprung's disease during a 10-year period, Ann. Surg., 156:706-714, October 1957.

100. Swenson, O.: Pediatric Surgery. Second Edition, Appleton Century Crofts, Inc., New York, 1962.

101. Swenson, O., and Bill, A. H.: Resection of rectum and rectosigmoid with preservation of the sphincter for benign spastic lesions producing megacolon, Surg., 24: 212-220, August 1948.

102. Talbert, J. L., and Haller, J. A.: Temporary tube pharyngostomy in the staged repair of congenital tracheoesophageal fistula, Surg., 58:737-740, October 1965.

103. Talbert, J. L., Schnaufer, L., Cahill, J. L., and Ravitch, M. M.: Primary division of the Duhamel spur with an anastomotic stapling instrument, presented at the Surgical Section of the American Academy of Pediatrics, Chicago, Illinois, 20 to 23 October 1966.

104. Tawtel, C., and Ducharme, J. C.: A preliminary report on seven cases of congenital megacolon treated by the Soave operation, presented at the Surgical Section of the American Academy of Pediatrics, Chicago, Illinois, 20 to 23 October 1966.

105. Thomaidis, T. S., and Arey, J. B.: The intestinal lesions in cystic fibrosis of the pancreas, J. Pediat., 63: 444-453, September 1963.

106. Wangensteen, O. H., and Rice, C. O.: Imperforate anus: A method of determining the surgical approach, Ann. Surg., 92:77-81, July 1930.

107. Weinstein, E. C., Cain, J. C., and ReMine, W. H.: Meckel's diverticulum: 55 years of clinical and surgical experience, J.A.M.A., 182:251-253, 20 October 1962.

108. Weinstein, E. C., Dockerty, M. B., and Waugh, J. M.: Neoplasms of Meckel's diverticulum, Int. Abstr. Surg., 116:103-111, February 1963.

109. Welch, K. J.: Meconium ileus; From Surgical Diseases of the Pancreas, by J. M. Howard and G. L. Jordon, J. B. Lippincott Co., Philadelphia, 1960, p. 71.

110. White, Harvey: Meconium ileus: A new roentgen sign, Radiology, 66:567-571, April 1956.

111. White, R. R.: Abnormalities of intestinal fixation, Surg. Gynec. Obstet., 116:726-730, June 1963.

112. Winslow, P. R., Bryant, L. R., and Hasbrouck, J. D.: Cystoscope endoscopy in the H-type tracheoesophageal fistula, Arch. Surg., 93:520-522, September 1966.

113. Wrenn, E. L.: Tubular duplication of the small intestine, Surg., 52:494-498, September 1962.

CASE REPORTS

Recurrent Cystosarcoma Phylloides

JAMES R. HANSEN, M.D., *Burlingame*

THE TERM *cystosarcoma phylloides,* was first used by Mueller in 1838. Sarcoma comes from the Greek word *sar,* meaning flesh, and *oma,* meaning tumor or neoplasm. By definition, *cystosarcoma* would represent a sarcoma in which the formation of cysts has occurred.

Phylloides is also derived from the Greek *phyllon,* meaning leaf, and *oides,* meaning resemblance. These are the sources of the term *cystosarcoma phylloides,* meaning a fleshy tumor with cyst formation and having what appear, on microscopic examination, as leafy projections. This term is the most commonly used for tumors of this kind, although they are also called "giant cell fibroadenoma."

According to Lee and Pack,[4] these tumors were very rare a hundred years ago and are even rarer now because the precursor fibroadenomas are recognized and removed much earlier.

Genesis

There are many possible precursor lesions etiologically incriminated in the development of this tumor. According to Adair and Hermann[1] numerous investigators were able to demonstrate that cystosarcoma could develop from fibroadenoma of the breast.

It would appear Lester and Stout[5] felt that these tumors are sarcomas that develop in fibro-epithelial tumors.

Pack and Ariel[6] expressed belief that multiple births and lactation are the most frequent stimuli to the metamorphosis of fibroadenoma into cystosarcoma phylloides. However, there appeared to be no consistent agreement in the literature reviewed as to the exact origin.

Gross Description

These tumors are usually very large, bulky tumors of the breast when first seen. The lesions are usually mobile, circumscribed and discrete from the surrounding breast tissue. There is usually no fixation of the overlying skin, although Lee and Pack[4] noted skin ulceration in 21 of 65 cases reported. There is no nipple retraction and no bleeding from the nipple. Usually the veins overlying the lesions are very prominent. Rarely is there attachment to or invasion of the pectoral fascia. The primary difference between this tumor and the usual fibroadenoma is size. The smaller tumors differ in more proliferative epithelium and coarser, more cellular connective tissue. They may be called miniature cystosarcoma phylloides, the size ranging from 1.0 cm to 20.0 cm.

Cystosarcoma phylloides tumors are made up mainly of intracystic polypoid masses, and owing to their multiple leaf-like laminations have been likened to a cabbage.

Lee and Pack[4] reported the weight of 19 tumors averaged 7.6 pounds. Other investigators have reported breasts containing such tumors weighing as much as 40 pounds.

Microscopic Appearance

There is a very decided variation in the histological appearance of these tumors, both from one part to another of the same tumor and between tumors. Lester and Stout[5] said that in the 58 cases reported by them there were 20 malignant tumors, 10 borderline and 28 benign. They emphasized, as have other investigators, the difficulty in classi-

---

Submitted 17 October 1966.
Reprint requests to: 1515 Trousdale Drive, Burlingame 94010.

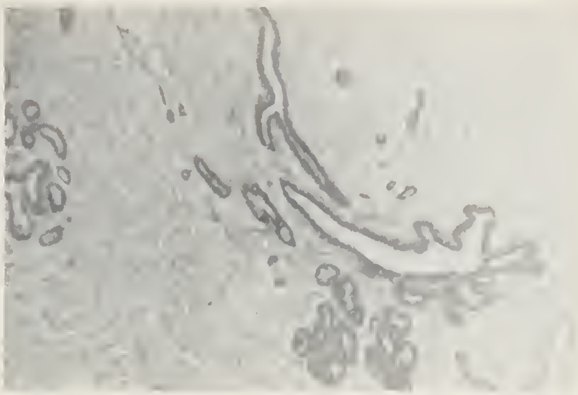


Figure 1.—Cellular stroma containing irregular ductal structures lined by a thick, deeply staining layer of epithelial cells (×53).

fication. They were unable to find any definite clinical guides that are explicitly indicative of malignant change. Size alone was not a basis for prediction.

The main criteria for identifying a tumor as benign is stromal overgrowth in a fibroadenoma pattern. Hyalinization and myxomatous change occur, and there are areas of irregular hyperchromatic nuclei, probably the result of degenerative change. Lee and Pack[4] emphasized the myomatous change in a series of cases they reported. Necrosis is not common. Calcareous deposits and pigment accumulation may occur in the stroma of the tumor. The ducts of the tumor are very dilated and tortuous.

The malignant tumors usually have a highly cellular stroma with considerable pleomorphism and mitotic activity and some invasive properties. In the great majority of cases the malignant change occurs in the connective tissue and not the epithelium. Occasionally bizarre patterns will develop, such as those of squamous carcinoma and osteogenic sarcoma. Tevis and Sunderland,[7] in a report of 77 patients, expressed the opinion that the lesion was more frequently malignant than had been previously suspected.

Clinical Features

According to Adair and Hermann[1] the average age of patients with this tumor is 45 years. Lee and Pack[4] reported the average known duration of the tumor is 6.7 years. The presence of a preexisting lesion is characteristic, as is rapid growth and attainment of large size. Ulceration of the overlying skin is uncommon and, when it does occur, is usually related to trauma. Percursory tumors are present in the vast majority of cases. Cystosarcoma phylloides tumors tend to develop more slowly than carcinoma of the breast and sometimes remain inactive for considerable periods.

Report of a Case

In November 1960, a 35-year-old patient was observed because of a palpable nodule in the axillary tail of the right breast. This particular portion of the breast was quite painful with each menstrual period; and just before she sought medical attention the patient had had increasing discomfort there.

Both breasts were very large and pendulous, and the very prominent, thick breast parenchyma made palpation of any discrete nodules difficult. A presumptive diagnosis of chronic cystic mastitis of the axillary tail of the right breast was made at that time, and the patient was kept under periodic observation.

Approximately nine months later the lesion in the axillary tail of the right breast had become more discrete and appeared to be 3 to 4 cm in diameter, although delineating margins was difficult.

A wedge of breast tissue 6 by 10 cm was excised for biopsy in August 1961, and the diagnosis at that time was "giant fibroadenoma with myxoid stroma (cystosarcoma phylloides) of the breast." Microscopically the lesion was fully circumscribed and had no capsule. The edges of the lesion extended with irregular processes into the surrounding tissues. Because of the benign appearance of the lesion, a quadrant excision of the breast was

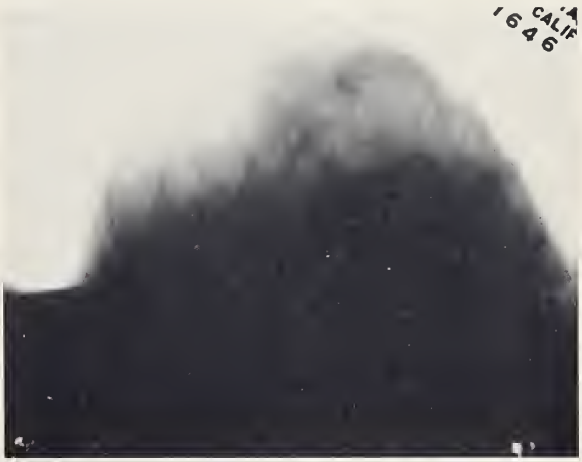


Figure 2.—A 9.0 cm giant fibroadenoma in the upper outer quadrant of the right breast is clearly visible in the mammogram.

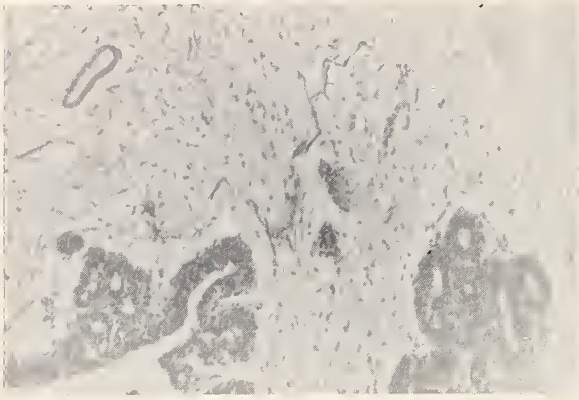


Figure 3.—The cells of the stroma are moderately pleomorphic, as are the epithelial cells lining the ductal structures (×133).

carried out as a definitive treatment and the patient made uneventful recovery.

She was observed periodically thereafter, and when she was examined in September of 1963 there appeared to be some fullness in the right breast, more prominent in the right upper and outer quadrant than elsewhere. Local recurrence of cystosarcoma phylloides was suspected. Mammograms revealed a 9 cm homogeneous, smoothly delineated mass in the upper outer quadrant of the right breast. One fairly large focus of calcification was present in this lesion, and most of the views taken at the time revealed some displacement of fatty tissue around the mass. There was no radiological evidence to suggest malignant disease. There was some compression of the adjacent normal surrounding breast tissue. The radiological appearance was quite characteristic of giant cell fibroadenoma (cystosarcoma phylloides).

On surgical exposure, a rounded, circumscribed mass about 8 cm in diameter was seen in the axillary tail of the right breast. Grossly it was very similar to the mass that had been removed two years earlier. On biopsy the lesion was found to be recurrence of cystosarcoma phylloides, and radical mastectomy was performed. There was no evidence of other tumor within the breast, and the axillary lymph nodes did not appear to be involved with tumor. The tumor, which was spherical, appeared to be completely encapsulated. Microscopically the tumor mass was histologically similar to the biopsy specimen that was excised in 1961. The present lesion showed numerous large and small glandular structures, some of which had a slit-like appearance. There was more hemorrhage and degeneration in the stroma of this lesion and a slight increase in the epithelial hyperplasia, with protrusion of small papillary processes into the gland lumen. The numerous axillary lymph nodes showed decided central fatty replacement of the lymphoid elements and in some nodes an apparent reticulo-endothelial hyperplasia. However, there was no evidence of malignant involvement. The pathological diagnosis was: "Giant fibroadenoma with stromal hemorrhage and degeneration—'Cystosarcoma phylloides.' " The patient made an uneventful recovery. When last seen, approximately five years after the first appearance of a lesion in the right breast, the patient had no recurrent disease.

Prognosis

Lee and Pack[4] reported six cases in a series of 91 cases. Of all the cases of benign cysts reported, none has caused death unless associated with independent carcinoma of the breast. Local recurrence can and does occur, but can be treated by more radical surgical procedures. Lester and Stout[5] reported five cases in which metastasis is known to have occurred, but in none of them was there metastasis to lymph nodes. The mortality rate for malignant cystosarcoma phylloides, including all lesions of unproved derivation, is about 20 to 25 per cent. Metastasis, when it occurs, usually is to the lungs and bone as hematogenous spread, and only in a very rare case is there local lymph node metastasis. Cooper and Ackerman[3] have reported one case of metastasis to lymph nodes. Ackerman also emphasizes the occurrence of the malignant form of these lesions.

Treatment

The preferred surgical procedure for dealing with such lesions ranges from simple local excision of the tumor mass to simple mastectomy to radical mastectomy. All have been used with varying degrees of success. If the lesion is benign and small and well localized to one quadrant of the breast, a wide quadrant excision of the area would appear from most reports to be adequate treatment. If the lesion is benign and large without evidence of lymph node metastasis, simple mastectomy would appear to be the treatment of choice. If it is benign and recurs after local excision, radical mastectomy is advisable, particularly if there is a prominent epithelial component. Radical mastectomy is also recommended for histologically malignant lesions with malignant epithelial elements.

If, as often is the case, histological examination leaves doubt as to whether the lesion is benign or malignant, radical mastectomy is the most prudent course. This is also the treatment of choice when the tumor has extended beyond the capsule.

Haffner[8] has expressed the opinion that these tumors are sarcomas that develop in fibroepithelial tumors, and that when they invade or spread, it is the mesenchymatous elements alone that are responsible. They believe the epithelial elements play only a passive role and never appear in metastatic lesions. The treatment they recommend for malignant cystosarcoma phylloides is extended simple mastectomy in which a wide margin of tissue, including surrounding skin and underlying pectoralis major muscle, is removed.

Adair and Hermann[1] expressed the opinion that, in light of the infrequency of lymph node involvement, radical mastectomy is not justified.

In the literature reviewed, there was little report of the use of radiation therapy in the treatment of this lesion. Adair and Hermann[1] said that there is not enough data on irradiation of a breast sarcoma by which to evaluate its efficacy.

In 1852, Dr. John Birkett[2] in a letter to the editor of *Lancet* said, "The unusual fact of a return of a cystosarcoma of the breast is undoubtedly most rare"; also, "The development of carcinoma subsequent to cystosarcoma is, happily, very rare"; and "Recurrence of cystosarcoma of the breast appears so important in a practical, as well as pathological point of view, that I beg to call your attention to the subject." These words, written in 1852, sound strangely similar to those statements appearing in our present-day literature on this very same rare entity.

Summary

A 35-year-old woman with a palpable nodule in the axillary tail of the right breast had pain in that area on menstruation. A presumptive diagnosis of chronic cystic mastitis was made but nine months later the nodule became more discrete and biopsy showed cystosarcoma phylloides, appearing to be benign. Quadrant excision of the breast was carried out. Two years later, fullness was noted in the right breast and when biopsy of a lesion observed on surgical exposure showed it to be recurrence of cystosarcoma phylloides, radical right mastectomy was done. When last observed, three years later, there was no evidence of recurrence.

REFERENCES

1. Adair, Frank E., and Hermann, Julian B.: Sarcoma of the breast, Surgery, 19:55, 1946.
2. Birkett, J.: Case of cystic sarcoma of the breast, Lancet, 1:161, 1852.
3. Cooper, Jr., W., and Ackerman, J. V.: Cystosarcoma phylloides: With a consideration of its more malignant variety, Surg., Gynec., and Obst., 77:279, 1943.
4. Lee, B. J., and Pack, G. T.: Giant intra-canalicular myoma of the breast, Ann. Surg., 93:250-268, 1931.
5. Lester, J., and Stout, A. P.: Cystosarcoma phylloides, Cancer, 71:335-353, March 1964.
6. Pack, G. T., and Ariel, I. M.: Tumors of the Soft Tissues—A Clinical Treatise, Paul B. Hoover, Inc. (Medical Book Dept. of Harper & Brothers), New York, 1958, pp. 286-300.
7. Treves, N. E., and Sunderland, D. A.: Cystosarcoma phylloides of the breast, Cancer, 4:1286-1332, 1951.
8. Zentr. Gynaekol (Haffner), 72:967-69, 1950.

Membranous Glomerulonephritis: Rapidly Fatal Course

W. H. TU, M.D., AND MARTIN A. SHEARN, M.D.,
Oakland, AND JOHN C. LEE, M.D.,
San Francisco

MEMBRANOUS GLOMERULONEPHRITIS has been distinguished histologically from lipoid nephrosis, proliferative glomerulonephritis[2,4] and poststreptococcal glomerulonephritis[5] by its characteristically diffuse thickening of the basement membrane of glomerular capillary tufts, with little proliferation of endothelial cells. Although precise information on the survival of patients with membranous glomerulonephritis after the onset of nephrotic syndrome is lacking, most investigators agree that the condition is relatively stable and progresses slowly, over a number of years, to renal insufficiency.[3,11] The purpose of this report is to record the rapidity with which membranous glomerulonephritis may progress and to point out that in its early stage the disease may be indistinguishable from pure lipoid nephrosis.[4]

Report of a Case

A 69-year-old white woman had been well until August 1963, when a trace of protein was found

---

From the Department of Medicine, The Permanente Medical Group, Kaiser Foundation Hospital, Oakland, and the Departments of Medicine and Pathology, University of California School of Medicine, San Francisco.

Submitted 27 October 1966.

Reprint requests to: 280 W. MacArthur Boulevard, Oakland 94611 (Dr. Tu).

in her urine during routine examination. Results of the last previous urine analysis, in 1961, were within normal limits. The only abnormality now noted on physical examination was a basal diastolic murmur of aortic insufficiency. There was no history of rheumatic fever, syphilis or hypertension. During the ensuing four months ankle edema developed. In February 1964 a 24-hour urine specimen contained 9.1 gm of protein which consisted largely of albumin, as shown by paper electrophoresis. The urine sediment contained 8 to 10 erythrocytes and 3 to 5 leukocytes per high power field, with occasional granular casts. A test for Bence Jones protein was negative. No abnormalities were noted on complete hemogram. Fasting blood sugar was 90 mg per 100 ml. The serum cholesterol level was 332 mg, blood urea nitrogen 13 mg and serum creatinine 0.9 mg per 100 ml. The 24-hour endogenous creatinine clearance rate was 60 ml per minute per 1.73 square meters of body surface. Serum protein by paper electrophoresis revealed albumin 1.5, α-1 globulin 0.5, α-2 globulin 0.86, β-globulin 0.57, and γ-globulin 1.0 gm per 100 ml. Two LE (lupus erythematosus) preparations were negative. The antistreptolysin-O titer was 12 Todd units. An electrocardiogram and roentgenogram of the chest were within normal limits.

A specimen of kidney tissue obtained in February 1964 by percutaneous needle biopsy contained six glomeruli. The renal architecture was well preserved. The glomeruli appeared normal except for questionable, irregular thickening of the basement membrane of glomerular tufts (Figure 1).

The patient's clinical course is outlined in Chart 1. Prednisone (60 mg daily) was administered for two weeks, then was discontinued by the patient because of severe weakness. The urinary protein excretion was essentially unchanged during that time. Then for two months 150 mg of 6-mercaptopurine (6 MP) was given daily without effect on the clinical course. In April 1964 the patient was admitted to the hospital because of weakness. There was pitting edema of all extremities. The serum albumin was 1.5 gm per 100 ml. Blood volume, determined with radioactive human albumin, was 2,589 ml (44 ml per kg of body weight). Transfusion of 1,000 ml of blood was followed by diuresis, with loss of five pounds in body weight.

The nephrotic syndrome persisted despite daily intramuscular injection of 40 units of ACTH gel for one month. In January 1965 the blood pressure was 250/90 mm of mercury and the pulse rate was 106 per minute. In July 1965, and repeatedly thereafter, the patient was admitted to hospital with signs and symptoms of uremia. She died in October 1965. The duration of illness was 24 months.

Necropsy

At necropsy, the kidneys weighed 120 and 130 gm. The capsules stripped with mild difficulty, leaving a smooth surface with rare V-shaped grooves. The cortex of each was bright yellow and uniform, averaging 0.4 cm in thickness, and the corticomedullary junction was sharp. On microscopic examination, the cortex showed extensive stromal fibrosis. Tubules occasionally contained bright eosinophilic granular casts, and a few showed an intraluminal neutrophilic infiltrate. The most prominent change was the marked diffuse thickening of the glomerular capillary basement membrane (Figure 2). The axial and stalk portions were thickened. Medium sized and larger

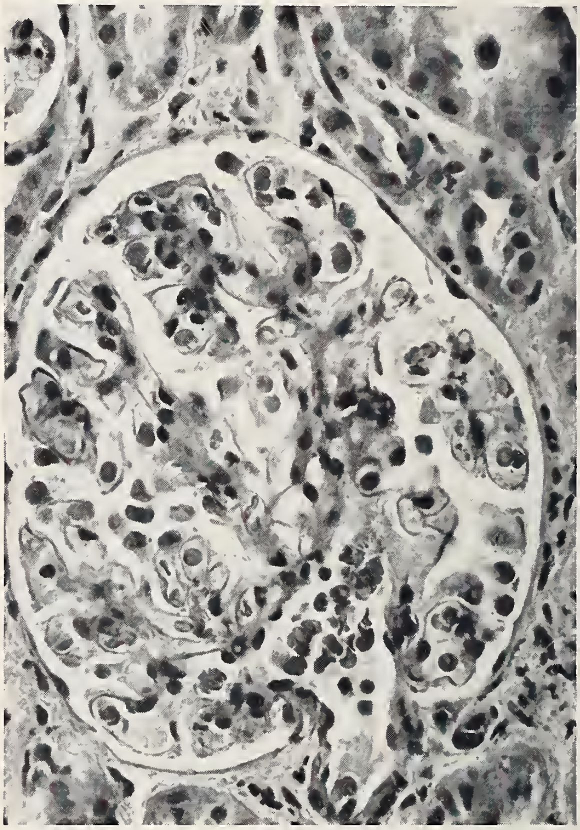


Figure 1.—A representative glomerulus from the renal biopsy specimen obtained in February 1964. The glomerular cellularity is normal and no leukocytic infiltration is present. Glomerular capillary basement membranes are not significantly thickened (hematoxylin and eosin, ×400).

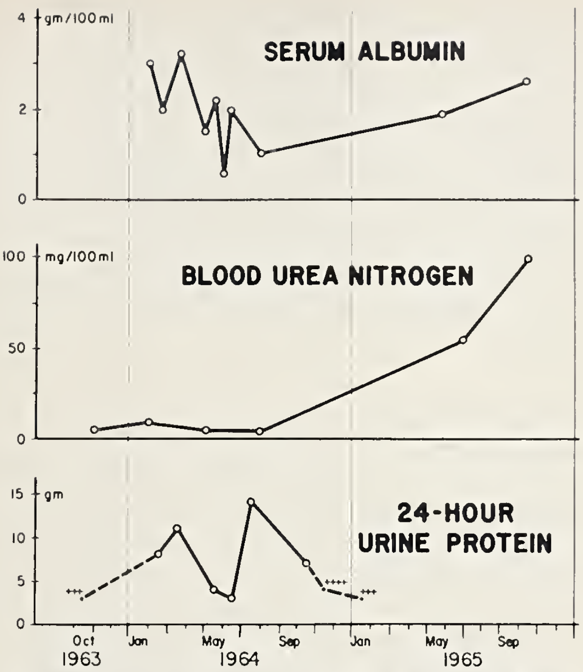


Chart 1.—Clinical course (in the lowest graph 24-hour urinary proteins are indicated by circles; dots refer to random samples).

arteries showed hypertrophy of the media, and some hyalinization. Arterioles were only slightly thickened.

Discussion

The insidious onset of nephrotic syndrome was the initial manifestation of renal disease in the patient reported here. With no evidence of other primary renal disease, or of an associated systemic disorder, this case belongs in the category of so-called "idiopathic nephrotic syndrome," in which the renal changes are primarily glomerular and are limited to the capillary walls of glomerular tufts. Proliferation of mesangium or cellular components is absent or minimal, distinguishing this syndrome from proliferative or poststreptococcal glomerulonephritis.[5]

Idiopathic nephrotic syndrome is further divided into two types[4]: (1) Lipoid nephrosis (foot process type, showing minimal or no change by light microscopy but fusion of epithelial foot processes by electron microscopy), and (2) membranous glomerulonephritis, showing diffuse homogeneous thickening of the basement membrane of glomerular capillary tufts by light microscopy and subepithelial deposits of dense material by electron microscopy. Some degree of fusion of foot processes is present.

A diffuse deposition of immune globulin and complement on the glomerular basement membrane is seen in membranous glomerulonephritis, but not in lipoid nephrosis.[7] This further suggests that these two conditions may be different entities.

In the present case, while the renal biopsy findings 22 months before death were indistinguishable from those of lipoid nephrosis, the autopsy findings were those of advanced membranous glomerulonephritis. Although transformation of lipoid nephrosis into membranous glomerulonephritis could not be ruled out because of the unavailability of electron microscopy, there has been no evidence in the literature that such a transformation occurs.[4]

The rate of progression to renal failure in membranous glomerulonephritis is usually slow;[3,6,10,11] in fact, renal biopsy findings may remain unchanged for many years.[10] Patients with membranous glomerulonephritis who proceed to renal insufficiency are noted to have a tendency to begin with more than the usual degree of hematuria,

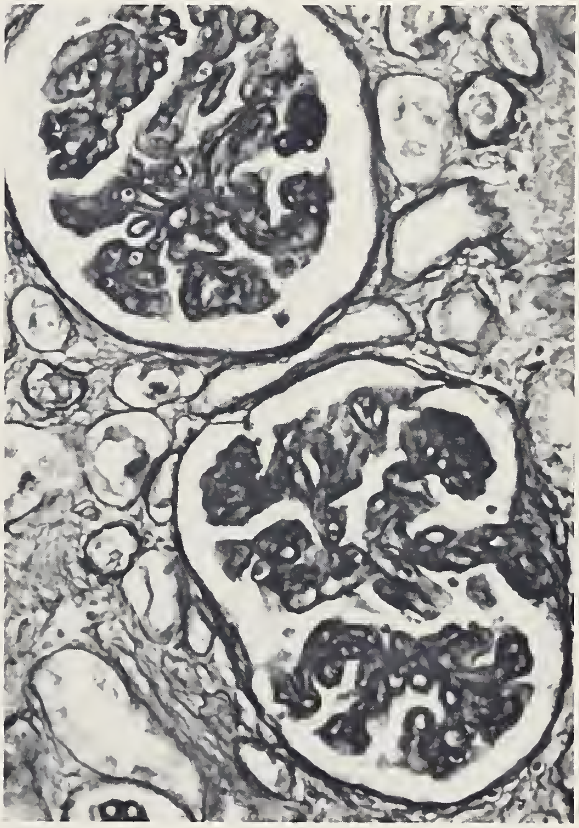


Figure 2.—Renal tissue at autopsy, 20 months after renal biopsy (Figure 1). There is diffuse, prominent thickening of the glomerular capillary basement membranes with no increase in glomerular cellularity. There is now pronounced interstitial fibrosis (para-aminosalycilic stain, ×250).

nitrogen retention or hypertension.[1] Such tendencies were not present initially in the case here reported; the cause of the rapid progression of the renal lesion is not clear. The possibility of superimposed renal vein thrombosis, which would result in rapid deterioration of renal function,[2] was not supported by the autopsy findings.

The observations in this patient are in agreement with the general experience of others in that the course of membranous glomerulonephritis seems to be little affected by adrenal steroids[2,4,8,9] or by 6 MP,[12] even when treatment is begun early in the course of the disease. The rapid progression of renal disease in the case herein reported suggests that the nephrotic syndrome initially associated with minimal glomerular changes and normal renal function may progress rapidly to renal insufficiency, and to the advanced renal lesion of membranous glomerulonephritis.

Summary

A case of adult idiopathic nephrotic syndrome, with renal lesions initially indistinguishable by light microscopy from lipoid nephrosis, progressed to advanced membranous glomerulonephritis within 20 months. Accompanying these histological changes in the kidneys were clinical progression to renal insufficiency and arterial hypertension with a fatal outcome.

The prognosis of nephrotic syndrome associated with membranous glomerulonephritis may be grave even when the initial renal changes are minimal.

REFERENCES

1. Arneil, G. C.: The syndrome of proteinuria; the nephrotic syndrome, Am. Heart J., 65:723, June 1963.

2. Blainey, J. D., Brewer, D. B., Hardwicke, J., and Soothill, J. F.: The nephrotic syndrome; diagnosis by renal biopsy and biochemical and immunological analyses related to the response to steroid therapy, Quart. J. Med., 30:235, April 1960.

3. Churg, J.: Personal communication.

4. Churg, J., Greshman, E., Goldstein, M. H., Yunis, S. L., and Porush, J. G.: Idiopathic nephrotic syndrome in adults; a study and classification based on renal biopsies, New Engl. J. Med., 272:165, 28 January 1965.

5. Jennings, R. B., and Earle, D. P.: Post-streptococcal glomerulonephritis; histopathologic and clinical studies of the acute, subsiding acute and early chronic latent phases, J. Clin. Invest., 40:1525, August 1961.

6. Lange, K., Treser, G., Sagel, I., Ty, A., and Wasserman, E.: Routine immunohistology in renal diseases, Ann. Int. Med., 64:25, January 1966.

7. McCluskey, R. T., Vassalli, P., Gallo, G., and Baldwin, D. S.: An immunofluorescent study of pathogenic mechanisms in glomerular diseases, New Engl. J. Med., 274:695, 31 March 1966.

8. Michael, A. F., Drummond, K. N., Good, R. A., and Vernier, R. L.: Immunoglobulins; clarification of their significance in renal disease and demonstration of response to immunosuppressive therapy (abstract), J. Clin. Invest., 43:1291, 1964.

9. Nesson, H. R., Sproul, L. E., Jr., Relman, A. S., and Schwartz, W. B.: Adrenal steroids in the treatment of idiopathic nephrotic syndrome in adults, Ann. Int. Med., 58:268, February 1963.

10. Pearl, M. A., Burch, R. R., Carvajal, E., McCracken, B. H., Woody, H. B., and Sternberg, W. H.: Nephrotic syndrome; a clinical and pathological study, Arch. Int. Med., 112:716, November 1963.

11. Pirani, C. L.: Personal communication.

12. Shearn, M. A.: Mercaptopurine in the treatment of steroid-resistant nephrotic syndrome, New Engl. J. Med., 273:943, 28 October 1965.

Generalized Mastocytosis

Fatal Case with Autopsy Findings

MEYER BERKE, M.D., *Lakewood*, AND
PHYLLIS C. WALKER, M.D., *Long Beach*

THE EMERGENCE of urticaria pigmentosa from the concept of a dermatosis limited to the skin to that of a widespread disorder (mastocytosis) has gradually evolved over the past three decades. This evolution began with the demonstration, by Touraine, Solente and Renault[17] in 1933, of mast cell accumulations in organs other than the skin. Urticaria pigmentosa was first described by Nettleship[12] in 1869 and was given this designation by Sangster[15] in 1878. Unna[18] in 1887 noted the presence of large numbers of mast cells in the cutaneous lesions although it was Paul Ehrlich[5] in 1877 who originally recognized the mast cell as a distinct cell type and gave it the name "mastzellen."

Ellis[6] in 1949 described the first autopsy findings in a one-year-old infant who died with systemic mastocytosis. Sagher, Cohen and Schorr[13] in 1952 demonstrated roentgenographic evidence of widespread osseous lesions in a 53-year-old man with the "adult" type of urticaria pigmentosa. In the past 14 years reports of additional cases of systemic mastocytosis have substantiated internal involvement and indicated that the liver, spleen, lymph nodes, peripheral blood, bone and bone marrow are often affected.

From the Department of Medicine, Division of Dermatology, UCLA School of Medicine and the Department of Pathology, Long Beach Memorial Hospital.

Submitted 14 November 1966.

Reprint requests to: 621 Butte Street, San Pedro 90732 (Dr. Berke).

Since the original report by Ellis, in 1949, of a case of urticaria pigmentosa with postmortem examination, more than two dozen additional cases with autopsy data have been reported.

Our interest in this disorder was recently spurred by observation of a patient with mastocytosis involving the skin, bone marrow and skeletal system. He also had arteriosclerotic heart disease, and later severe recalcitrant anemia of unknown cause developed.

Report of a Case

A 74-year-old retired white man was first seen in December 1962 because of a generalized mildly pruritic erythematous hyperpigmented eruption of approximately one year's duration. Clinically the diagnosis of urticaria pigmentosa seemed apparent and this was confirmed on biopsy. Toluidine blue stains showed typical mast cells with characteristic granules in moderate number, primarily in the upper cutis.

In November 1963 the patient entered Long Beach Memorial Hospital because of sudden onset of weakness and dyspnea. Previously the patient had been treated for mild hypertension and diabetes by weight reduction. He had been admitted to Seaside Hospital in Long Beach in 1955 for diverticulitis of the sigmoid colon. Results of complete blood count and urinalysis at that time were within normal limits.

On physical examination, the patient was dyspneic and pale with blood pressure of 100/80 mm of mercury and pulse rate of 120 per minute. There was a diffuse eruption of the trunk and extremities consisting of many discrete, macular, papular to nodular erythematous slightly hyperpigmented lesions varying from 0.5 cm to 1.5 cm in diameter. (Figure 1).

Moist rales were present at the lung bases, and the heart rhythm was irregular and rapid. The abdomen was relaxed and the liver and spleen were not palpable. No abnormality was noted on rectal examination. The hemoglobin was 8.6 gm per 100 ml, the hematocrit 28. Erythrocytes numbered 2.9 million per cu mm of blood, and leukocytes 15,300 per cu mm with 82 per cent polymorphonuclear leukocytes, 11 per cent lymphocytes and 7 per cent monocytes. There were 210,000 platelets per cu mm and the reticulocyte count was 1.2 per cent.

Specific gravity of the urine was 1.023, and it contained a trace of sugar and 2+ protein. There were a few white blood cells and many fine granular casts in the urinary sediment. Blood urea nitrogen, acid phosphatase, bilirubin and calcium were within normal limits. By electrophoresis the albumin was 3.3 gm and the globulin 2.8 gm per 100 ml, with normal distribution of the globulin fractions.

Auricular fibrillation was demonstrated by electrocardiogram. X-ray films of the chest showed extensive involvement of the ribs by an osteosclerotic process thought to represent metastasis from cancer of the prostate. Similar changes were noted in the pelvis and vertebrae, but the skull was not involved. (Figure 2). On bone marrow examination the marrow was very hypercellular with a myeloid:erythroid ratio of 11.1. Erythropoiesis was normoblastic and the maturation pattern of the white precursors appeared normal and accelerated. Iron stores were increased. No tumor cells or mast cells were seen.

The patient responded to treatment with packed red cells and digitalis. After discharge from the hospital he was given iron by mouth for approximately six weeks. In January 1964 he was again in hospital because of progressive weight loss and anemia. The hemoglobin at that time was 6.2 per 100 ml, the hematocrit 20, erythrocytes 2.0 million per cu mm and reticulocytes 1.2 per cent. Leukocytes numbered 10,400 per cu mm—81 per cent polymorphonuclear cells, 6 per cent lymphocytes and 13 per cent monocytes. On careful examination of a specimen of peripheral blood, moderate numbers of hypochromic cells were seen. The bone marrow was hypercellular marrow but as before, there was no evidence of mast cells or tumor cells. The marrow iron was increased and

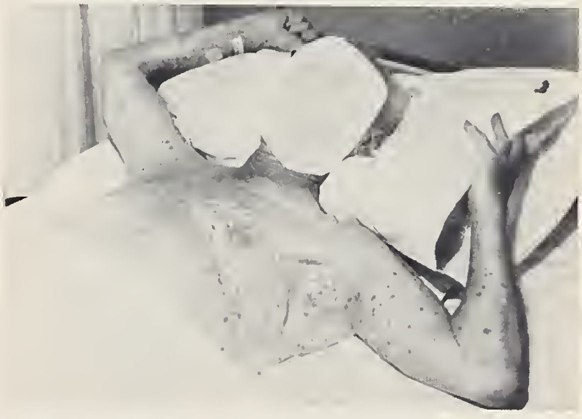


Figure 1.—Characteristic hyperpigmented papular to nodular lesions of urticaria pigmentosa with tendency to confluence on the chest and abdomen.

ringed sideroblasts were observed. There appeared to be poor hemoglobinization of the red cells and moderate numbers of dysplastic red cells were present. The serum iron was 166 mcg and the iron binding capacity was 181 mcg per 100 ml.

The patient's condition deteriorated and he required repeated transfusions. He died in July 1964 of acute pulmonary edema. During the illness he received 22 units of packed red blood cells.

Autopsy

At autopsy 18 hours after death, acute pulmonary edema was confirmed as the terminal event. The heart weighed 290 grams and throughout the myocardium of the left ventricle there were patchy areas of fibrosis. Iron was present within the myocardial fibers, but no mast cells were seen. There was severe coronary atherosclerosis. The liver, which weighed 1,650 gm, was smooth and reddish brown. On microscopic examination a moderate increase in fibrous tissue in the periportal areas was noted. Iron stains revealed large amounts of iron within the hepatic parenchymal cells and in the reticuloendothelial cells. Only scattered mast cells were demonstrated by toluidine blue or Giemsa stains.

In the spleen, which weighed 350 gm, large areas of fibrous tissue replaced much of the splenic parenchyma. Iron was abundant and mast cells were found in modest numbers.

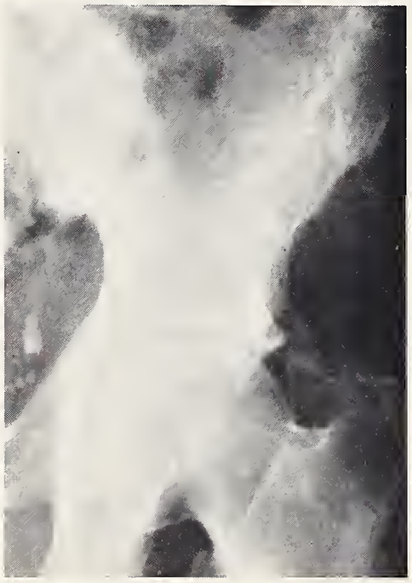


Figure 2.—X-ray film of a portion of the pelvis. In the wing of the ilium there is loss of the normal trabecular pattern with the areas of increased and decreased density producing a mottled effect.

Sections of bone from the ribs, vertebrae and pelvis revealed irregularly thickened trabeculae. Localized areas of fibrosis, in close approximation to the bony trabeculae, contained variable numbers of mast cells. The adjacent bone marrow was decidedly hypercellular and mast cells were not seen in the myeloid areas. (Figure 3).

The lymph nodes in the mesentery and in the retroperitoneal space in the region of the pancreas were enlarged, to as much as 2.5 cm. These nodes were white and firm, and within them were areas of fibrosis. Iron was abundant and only a few mast cells were seen.

In the pancreas there was a slight increase in fibrous tissue. Many of the islet cells appeared hyalinized. Iron was present within the parenchymal cells of the organ.

Sections of the pigmented lesions of skin revealed accumulations of mast cells in the upper cutis.

Discussion

Systemic mast cell disease is characterized by the presence of unusual numbers of mast cells in various organs of the body. Those most commonly involved include the skin, bone marrow, spleen, liver and lymph nodes. Because of this distribution and the belief that mast cells are derived from mesenchyme, this entity has been classified as a reticulo-endothelial disorder.

The mast cell has been considered as a unicellular endocrine gland because of its several secretory functions.[10,11] The granules in the mast cells are with certainty the sites of formation of heparin, an acid mucopolysaccharide, and of histamine. There is also strong evidence that hyaluronic acid, a nonsulfated mucopolysaccharide, is also secreted by the mast cells.

The clinical varieties of urticaria pigmentosa are well described in the literature. These include the "juvenile" and "adult" types which differ somewhat in clinical appearance, histologic formation and prognosis. The childhood variety more often tends to be nodular and the lesions are at times bullous and occasionally solitary. About 50 per cent of all cases appear before the age of six months. In the adult category the individual lesions are inclined to be smaller and more macular. There is a pronounced tendency for eventual spontaneous remission in the juvenile type; this is uncommon in the adult classification. Histologically mast cells in the cutis frequently are quite numerous in childhood and often sparse in adult cases. Pruritus and Darier's sign (irritation with rubbing) tend to be less pronounced in both types with increased duration of the disease.

The morphologic variations in addition to the macular, papular, nodular and bullous forms include the rare telangiectatic classification—telangiectasia macularis eruptiva perstans (Weber and Hellenschmied)[20] and a diffuse type.[3] Lesions of the mucous membranes have been described. The color of individual lesions may vary from flesh colored, to reddish yellow to dark brown.

The symptoms of urticaria pigmentosa include flushing, tachycardia, shock, pruritus, dermographia, headaches and gastrointestinal disturbances, including peptic ulcer, diarrhea and malaise.

Systemic mastocytosis is apparently more often associated with the adult type of urticaria pigmentosa than with the childhood variety. Also, instances of systemic mastocytosis without cutaneous findings have been reported.[7,9]

Hematologic abnormalities have been described as common in generalized mastocytosis. However, they are not always present, nor are they consistent. Many of the patients with bone lesions roentgenographically demonstrable do not have anemia. Friedman and coworkers[8] reported severe anemia in a patient having what has been termed tissue mast cell leukemia, with large numbers of mast cells in the peripheral blood and bone marrow. Conversely, Bowdler and Tullett[2] described a case of urticaria pigmentosa associated with polycythemia vera, and they speculated that both conditions represented hyperplastic conditions of mesenchymal cells.

The patient in the present case had refractory anemia which undoubtedly contributed to heart failure and death. The anemia was normochromic, normocytic without reticulocytosis, and was associated with a decidedly hypercellular bone marrow. There was a mild absolute monocystosis and lymphopenia. The presence of ringed sideroblasts, mypochromic as well as normochromic cells in the peripheral blood smear, and a high serum iron content with a high degree of saturation of the iron-binding capacity, suggested anemia of sideroblastic type.

Another finding in this patient was large amounts of iron not only in the reticuloendothelial cells but also in the parenchymal cells of the pancreas, liver and heart. Bothwell and Finch[1] pointed out that parenchymal deposits of iron, even the tissue changes of hemochromatosis, are found in patients with anemia and ineffective erythropoesis. It would appear that the severe iron overloading in the present case was the result of the refractory anemia, the administration of oral iron and blood transfusions.

It is of interest that the roentgenologic findings in the case here reported were first interpreted by the radiologist as highly suggestive of metastatic malignant disease, possibly prostatic in origin. However, no primary tumor was found while the patient was living, or at autopsy, and it became apparent that the radiologic changes in the ribs, pelvis and spine were undoubtedly the result of mast cell infiltration. Hemochromatosis had been considered.

In the reported cases in which systemic mastocytosis was noted at autopsy, the eventual causes of death include in the majority of instances a variety of blood dyscrasias including such diagnoses as "myeloblastic,"[16] "monocytic"[14] and "mast cell"[4,19] leukemias. In other cases, even though there was no apparent leukemic process, the patient had progressive cachexia and at autopsy there was evident mast cell invasion of the vital organs, most commonly the bone, bone marrow, liver, spleen and lymph nodes. Hemorrhage was an outstanding feature in at least two cases.[4,19]

It is significant that Touraine, Solente and Renault[17] in 1933 considered urticaria pigmentosa as-

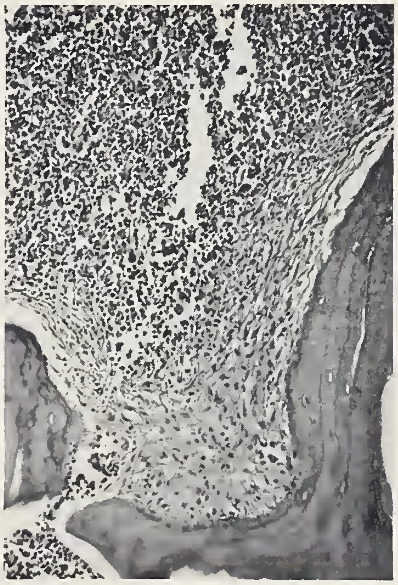


Figure 3.—Section of the lumbar vertebra. Adjacent to the thickened bony trabecula there is a granulomatous area. The adjacent bone marrow is hypercellular (×40).

sociated with internal involvement as a pseudoleukemia because of the splenomegaly and lymphoid hyperplasia. In retrospect it is apparent from the autopsy material that systemic mast cell disease may terminally manifest itself as a true leukemic process. Why this is true in some cases and not in others is not understood.

It would appear from reviewing the literature that systemic mast cell disease occurs in a small minority of patients with urticaria pigmentosa and should be more suspect in adults with this cutaneous disorder.

The most common findings which will confirm the systemic nature of this disease in the adult are the characteristic radiologic changes, with mottled osteoporosis and osteosclerosis involving particularly the skull, rib cage, vertebral column and pelvis. These roentgen findings can be easily confused with metastatic malignant change, as well as with multiple myeloma, Paget's disease and, at times, leukemia. The localized roentgenologic changes thus far described are not especially diagnostic. In addition, bone marrow aspiration, biopsy of the liver, spleen and lymph nodes may show mast cell infiltration confirming the diagnosis.

It is evident that many patients with systemic mastocytosis may live with their disease for long periods and eventually die of other causes. In a minority who are less fortunate, more serious abnormalities are likely to develop—anemia, cachexia and at times, terminally, a fulminating leukemia.

Summary

In a case of fatal mastocytosis in an elderly man, the outstanding feature was severe recalcitrant anemia which seemed without explanation until the association of the cutaneous lesions of urticaria pigmentosa with mast cell involvement of the bone and bone marrow was noted. The anemia was probably related to the mast cell infiltration of the bone marrow. There was no evidence of leukemia.

REFERENCES

1. Bothwell, T. H., and Finch, C. A., Iron Metabolism, Little, Brown & Co., 1962, pp. 284-286, 403.
2. Bowdier, A. J., and Tullett, G.: Urticaria pigmentosa and polycythaemia vera, A case report, Brit. Med. J., 1:396-397, 6 February 1960.
3. Degos, R., Lortat-Jacob, E., Hewitt, J., and Ossipovski, B.: Reticulose à mastocytes. Forme cutanée diffuse, Bull. Soc. Franc. Dermo et Syph., 59:247, 1952.
4. Efrati, P., Klajman, A., and Spitz, H.: Mast cell leukemia?—Malignant mastocytosis with leukemia-like manifestations, Blood, 12:869, 1957.
5. Ehrlich, P.: Beiträge Zur Kenntniss der Anilinfarbungen und ihrer Verwendung in der mikroskopischen Technik, Arch. Mikr. Anat., 13:263, 1877.
6. Ellis, J. M.: Urticaria pigmentosa: Report of a case with autopsy, Arch. Path., 48:426, 1949.
7. Ende, N., and Cherniss, E. I.: Splenic mastocytosis, Blood, 13:631, 1958.
8. Friedman, B. I., Will, J. J., Freiman, D. G., and Braunstein, H.: Tissue mast cell leukemia, Blood, 13:70, 1958.
9. Gonnella, J. S., and Lipsey, A. I.: Mastocytosis without skin involvement, New Engl. J. Med., 271:533, 1964.
10. Jorpes, J. E.: Heparin in the Treatment of Thrombosis, Ed. 2, Oxford Medical Publishers, New York and London, 1946, pp. 59-66.
11. Jorpes, J. E.: Origin and physiology of heparin, specific therapy in thrombosis, Ann. Int. Med., 27:361, 1947.
12. Nettleship, E.: Rare forms of urticaria, Brit. Med. J., 2:323, 1869.
13. Sagher, F., Cohen, C., and Schorr, S.: Concomitant bone changes in urticaria pigmentosa, J. Invest. Dermat., 18:425, 1952.
14. Sagher, F., Liban, E., Ungar, H., and Schorr, S.: Urticaria pigmentosa with bone involvement. Mast cell aggregates in bones and myelosclerosis found at autopsy in a case dying of monocytic leukemia, J. Invest. Dermat., 27:355, 1956.
15. Sangster, A.: An anomalous mottled rash, accompanied by pruritus. Factitious urticaria and pigmentation, "Urticaria Pigmentosa," Trans. Clin. Soc. Lond., 11:161, 1878.
16. Tenborg, J. A. G.: Urticaria pigmentosa bij chronische myeloide leucaemie or reticulose? Nederl, tijdschr. geneesk., 98:60, 1954.
17. Touraine, A., Solente, G., and Renault, P.: Urticaire pigmentaire avec reaction splenique et mylemique, Bull. Soc. Franc. Derm. Syph., 40:1691, 1933.
18. Unna, P. G.: Beiträge zuranatomic und pathogenese der urticaria simplex und pigmentosa, Dermat. Studien, 3:1, 1887.
19. Waters, W. J., and Lacson, P. S.: Mast cell leukemia presenting as urticaria pigmentosa: Report of a case, Pediatrics, 19:1033, June 1957.
20. Weber, F. P., and Hellenschmied, R.: Telangiectasia macularis eruptiva perstans, Brit. J. Dermat., 42: 374, 1930.

MEDICAL STAFF CONFERENCE

Aortic and Mitral Valve Replacement

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California Medical Center, San Francisco. Taken from transcriptions, they are prepared by Drs. Martin J. Cline and Hibbard E. Williams, Assistant Professors of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine.

DR. HUTCHINSON*: The patient is a 28-year-old housewife and the mother of two boys, ages 10 and 11. When she was 12 years old, she had inflammatory rheumatism followed by a rash and chorea. A diagnosis of acute rheumatic fever was made and she was put to bed for a year. There is no information as to whether she had cardiac abnormalities at that time.

For the next five years, she had recurrent arthralgia, although she was able to be active during this period. At age 17 and 18 she had the first two of three pregnancies. They were complicated only with minimal ankle edema and mild hypertension requiring no therapy. At age 22 she had an exacerbation of rheumatic fever following streptococcal pharyngitis and was told of cardiac murmurs for the first time. She was bedridden for a number of months, but there were no signs of heart failure.

Two years later she became pregnant for the third time. Her physicians felt that she would not survive a delivery and a therapeutic abortion was performed. The next year she had influenza and atrial fibrillation developed which resulted in severe palpitations, orthopnea and significant dyspnea on exertion. It was possible to convert the rhythm to sinus rhythm with digitalis and quinidine. To the present, sinus rhythm has been maintained while she is taking these two drugs.

For the last three years she has had recurrent arthralgia despite penicillin given for prophylaxis. She has noticed the gradual appearance of easy fatigability, palpitations on exertion and occasional ankle edema. In the last year she has experienced several episodes of classic paroxysmal nocturnal dyspnea and has severely restricted her activities.

The pertinent physical findings on admission to hospital were as follows: The blood pressure was slightly widened—110/55 mm of mercury; the pulse was regular at a normal rate; there was a prominent A-wave in the neck. Pulsus bisferiens with a systolic thrill was noted in the carotid arteries. A Quincke pulse and Duroziez's sign were present. The lungs were clear. The heart was enlarged to the anterior axillary line. Pronounced overactivity of both left and right ventricles was noted. The first heart sound was normal; the second heart sound was normally split. An opening snap was not heard but there was a third heart sound at the apex. Murmurs of both aortic and mitral valve disease were present. At the base and extending into the neck there was a grade 3/6 ejection murmur of moderate length. There was a grade 3 diastolic murmur best heard at the left sternal edge. At the mitral area and at the apex there was a grade 3 holosystolic murmur transmitted to the axilla, and a grade 3 fairly long diastolic murmur with presystolic crescendo.

An electrocardiogram was characterized by sinus rhythm, a PR interval of 0.20 second, p-mitrale and moderate ventricular hypertrophy (Figure 1). The patient was receiving quinidine and digitalis at this time. On the phonocardiogram the aortic ejection murmur was diamond-shaped and not particularly long. Both aortic and pulmonic components of S_2 were visible. A diastolic

---

*John C. Hutchinson, M.D., Assistant Clinical Professor of Medicine.

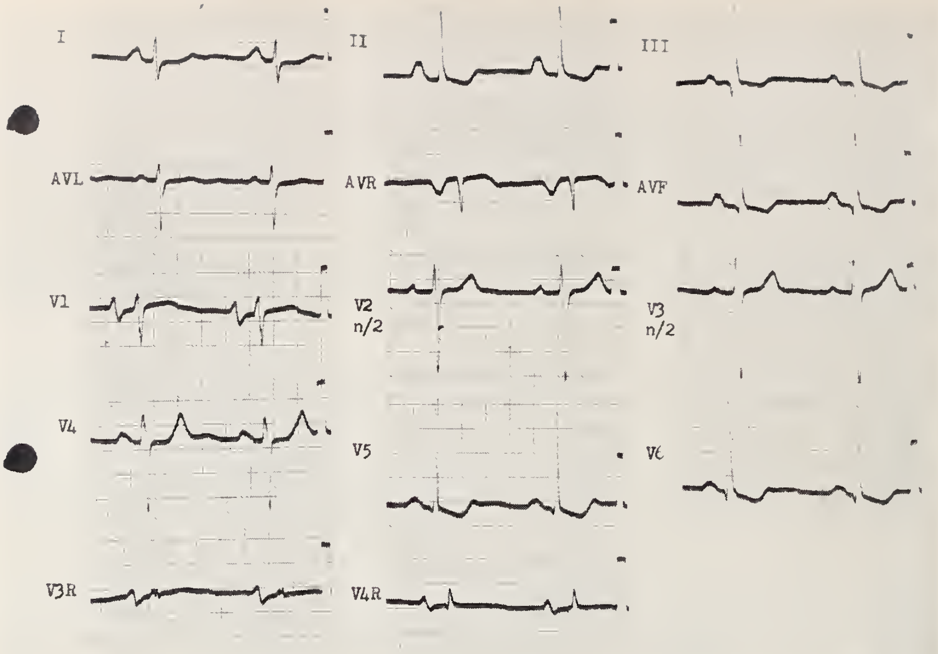


Figure 1.—Electrocardiogram, 4 October 1965, characterized by sinus rhythm, PR interval of .20 seconds, p-mitrale, and moderate ventricular hypertrophy.

Interpretation: Sinus rhythm. Rate 60/min. P-R 2.20 sec. QRS 0109 sec. Axis 90°. Tall, broad, notched P in II. ST sagging with low T waves in II, III, AVF, V5-V6. R in V6 + S in V2 = 46 mm.

Conclusion: Abnormal record due to moderate left ventricular hypertrophy and atrial hypertrophy; 1° A-V block. Since tracing of 6-3-65 no significant change.

murmur of very high frequency was not well recorded along the left sternal border. The carotid upstroke time was 0.08 second, which is in the normal range. There was a rather pronounced fourth heart sound, and a presystolic crescendo of the diastolic murmur.

Dr. Carlsson will discuss the roentgenograms.

DR. CARLSSON*[1]: The heart was enlarged. The most prominent finding was a decided enlargement of the left atrium as indicated by posterior displacement of the esophagus, slight compression and cranial displacement of the left main bronchus and visibility of the left atrial contour through the cardiac shadow in the frontal projection. In addition, the left and the right ventricles were moderately enlarged (Figures 2 and 3). Tomographic studies revealed calcification of the mitral valve (Figure 4). The pulmonary vessels were enlarged centrally and in the upper portions of the lungs, while in the lower portions the vessels had a smaller than normal caliber. In the peripheral and lower portion of the lungs, horizontal lymphatic lines were seen. The pulmonary vascular findings indicate pulmonary arterial and venous hypertension.[13]

The calcification of the mitral valve and the degree of atrial enlargement, as well as the pulmonary and vascular changes, strongly indicate mitral valve lesion.

The mitral valvular lesion could be of stenotic type with or without an element of insufficiency.

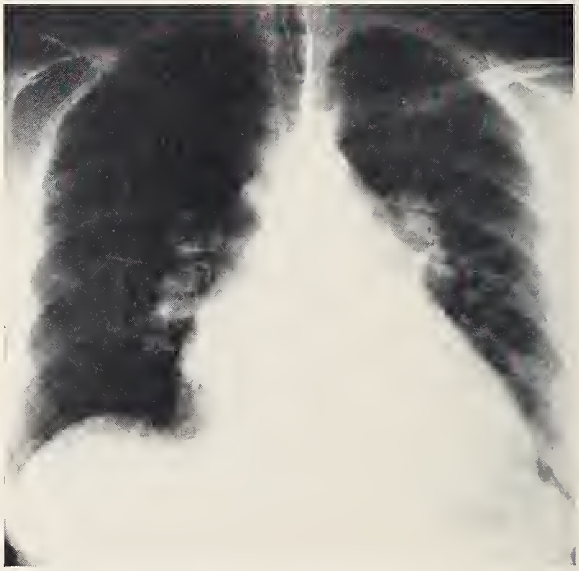


Figure 2.—X-ray film of chest showing moderate enlargement of left and right ventricles.

---

*[1] Erik Carlsson, M.D., Associate Professor of Radiology.

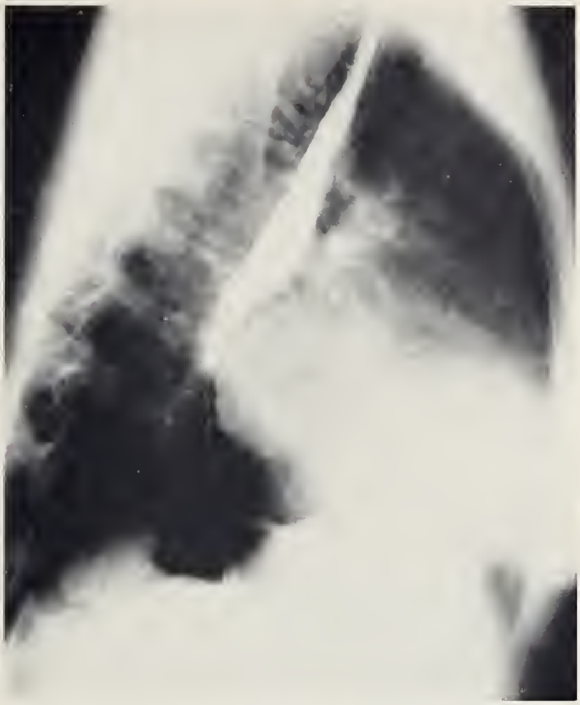


Figure 3.—Lateral x-ray film of chest showing enlargement of ventricles.

The moderate enlargement of the left ventricle indicates some degree of mitral valve regurgitation, if aortic valve insufficiency and myocardial lesion can be excluded.

Dr. Hutchinson: Thank you, Dr. Carlsson. The cardiac catheterization data are summarized in Table 1. The catheter was inserted through a vein to the right heart and from the femoral artery retrograde to the aorta and the left ventricle. These data are interpreted as indicating fixed mitral and aortic valve disease with pulmonary hypertension. The predominant lesion was mitral stenosis, with an end diastolic gradient of 25 mm of mercury. The systolic aortic gradient was 37 mm of mercury, indicating some degree of aortic stenosis; but in the presence of moderate aortic insufficiency this gradient is increased due to the increased stroke volume. There is a moderately severe increase of pulmonary vascular resistance to 7 mm of mercury per liter per minute (N=1-2). The combined effect of these lesions was to reduce

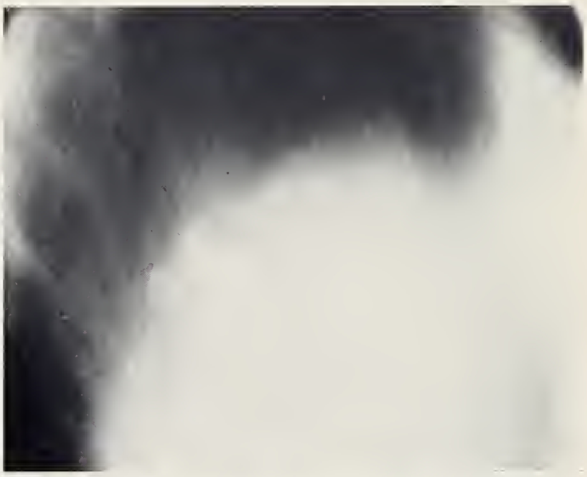


Figure 4.—Tomographic study reveals calcification of mitral valve.

TABLE 1.—*Data on Cardiac Catheterization*

Technique:

1. Right heart to pulmonary capillary wedge
2. Left heart retrograde from femoral artery to left ventricle.
3. Angiocardiograms from left ventricle and aorta

Results:

		Normals
Cardiac output	3.0 liters per minute	>3.5 liters per minute
Cardiac index	1.6 liters per minute/M^2	2-4 liters per minute/m^2
Pressures	aorta—123/70 mm Hg	120/80 mm Hg
	left ventricle—160/9 mm Hg	120/5 mm Hg
	wedge—a=25 mm Hg	<12 mm Hg
	x=22 mm Hg	<5 mm Hg
	v=25 mm Hg	<12 mm Hg
	y=30 mm Hg	<5 mm Hg
	mean=27 mm Hg	<11 mm Hg
	pulmonary artery =80/30 mm Hg	25/10 mm Hg
	pulmonary artery mean=48 mm Hg	15 mm Hg
	right ventricle =81/8 mm Hg	25/3 mm Hg
	right atrium (mean) =7 mm Hg	3 mm Hg
Pulmonary vascular resistance= 7 mm Hg liters per minute		<2 mm Hg liters per minute

---

L=liter; m^2=square meter of body surface; Hg=mercury.

the cardiac index to 1.6 liter per minute per square meter of body surface (N=2.0 or greater).

At the time of the catheterization, angiograms were done from the left ventricle and from the aorta. Dr. Carlsson will present the information.

Dr. Carlsson: Contrast medium was injected with the catheter tip in two different positions—in the ascending aorta 4 cm above the aortic valve, and in the left ventricular cavity. The aortic injection showed moderate dilatation of the ascending aorta, normal coronary arteries, thickened aortic valvular cusps, which were somewhat restricted in their movements and a slight to moderate regurgitation of contrast medium from the aorta to the left ventricular cavity. The left ventricular injection demonstrated a large left ventricular cavity, a ventricular wall of normal thickness, good myocardial contractions with a large ventricular output, a thickened mitral valve with restricted movements as in mitral valve stenosis, and regurgitation of a large amount of contrast medium from the left ventricle to the decidedly enlarged left atrium. The left ventricular volume was measured: End diastolic volume=105 ml, end systolic volume=300 ml, stroke volume=75 ml. Effective forward stroke volume as measured by Fick was 42 ml, regurgitant flow per stroke, 75 minus 42, was 33 ml. Valvular efficiency was 56 per cent. The main part of the total regurgitant flow occurred through the mitral ostium.

Figure 5 shows the angiocardiographic films which were used for volume calculations of the left ventricle. (For those interested in methods for

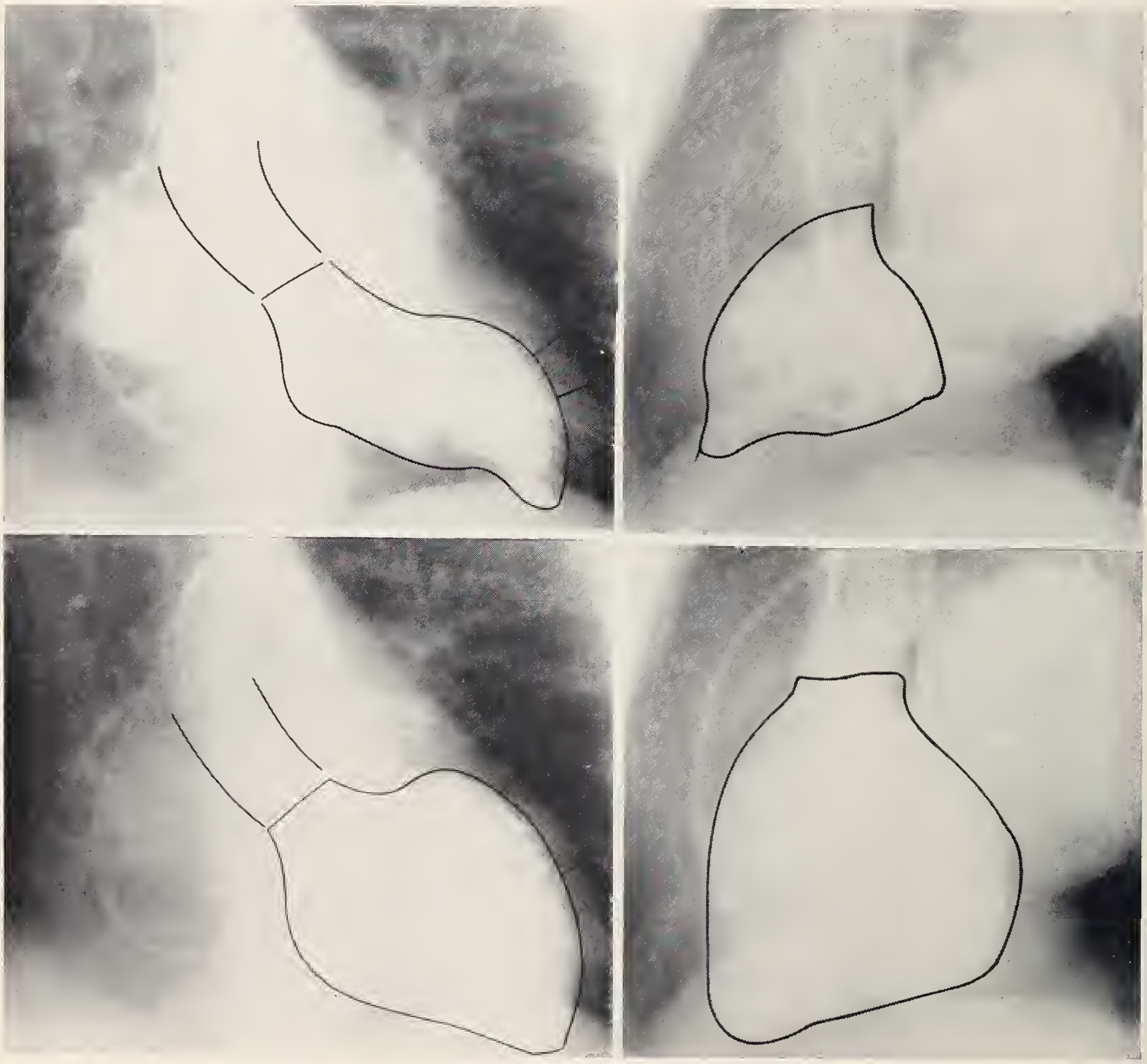


Figure 5.—Angiographic films used to calculate volume of left ventricle.

angiocardiographic volume measurement of cardiac ventricles, the following references are given:[2,7,8].)

Dr. Hutchinson: It was concluded that the patient had both aortic and mitral insufficiency and a moderate degree of mitral stenosis as well. She was in class III condition despite excellent medical care and could not be expected to be improved by medical therapy. Consequently she was taken to the operating room on the fifth of November and the aortic and mitral valves were replaced. The by-pass was in effect for 242 minutes. Both coronary arteries were perfused for 194 minutes at 200 ml a minute and a pressure of 110 mm. This is considered excellent coronary perfusion.

The mitral valve was found to be fused and the commissures were calcified. The edges of the valve were so retracted and scarred that there was not enough substance to cover the mitral orifice in systole. The calcium was so dense that reconstruction of the valve was precluded. The aortic valve was insufficient; the commissures were fused about halfway along their length and the apex of each cusp was retracted and fibrotic: Here, too, there was not enough valve substance to close the valve orifice in diastole. Consequently both valves were removed and replaced with prosthetic devices.

The operative period was essentially uncomplicated.

Immediately after the termination of the operative ventricular fibrillation with one shock of direct current, the aortic pressure was 150/80 mm at the "cost" of a left atrial pressure of 10 mm of mercury. We feel that this ratio of the mean arterial pressure to the left atrial pressure is an excellent index of ventricular function.

Anticoagulation therapy was started on the third postoperative day and a prothrombin time of 25 seconds (15 per cent activity) was established by the time of discharge. The patient went home on the fifteenth postoperative day with the heart in sinus rhythm, taking digoxin, quinidine, warfarin, vitamin C, ferrous gluconate and penicillin in prophylactic doses.

Plain cardiac films were made after the aortic and mitral valve replacement, showing the prosthetic valves in place as well as a decrease in cardiac size as compared with the preoperative films (Figure 6).

When last observed, three and a half months later, she was able to perform all her household chores and could climb two or three flights of stairs without any disability at all. She said that the noise and vibration of the valve closures bothered her although she was becoming accustomed to them to some extent.

Dr. Smith*[2]: This patient was referred to the hospital by Dr. Alan C. Calkin from Sacramento.

I think we will all agree that this accomplishment is a splendid example of what can now be achieved through the advances in cardiac surgery. It represents, as brought out by Dr. Hutchinson's very thorough presentation, the effect of the cooperative effort of a group of specialists—radiologists, cardiologists and surgeons—working to-

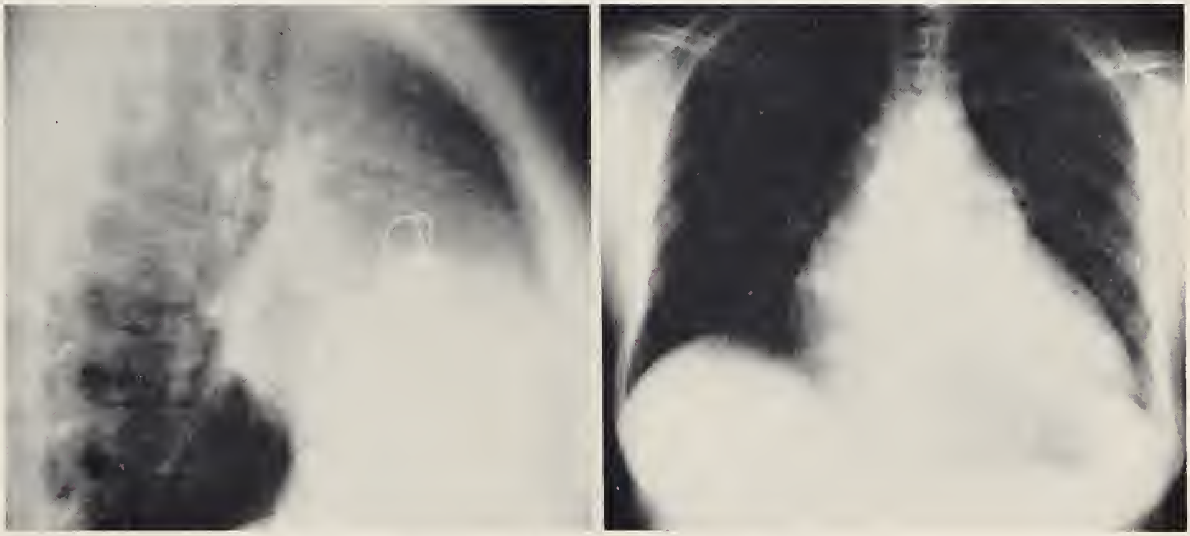


Figure 6.—Plain cardiac film showing prosthetic valve in place.

---

*[2] Lloyd H. Smith, Jr., M.D., Professor and Chairman, Department of Medicine.

gether to achieve a common end. In our plan for the program this morning, we intend to call upon representatives of these three specialties. First of all, Dr. Erik Carlsson will comment more generally upon the use of x-ray and angiography in this problem.

DR. CARLSSON: In mitral stenosis it is possible to determine the approximate pressure in the pulmonary arterial tree from the anatomic appearance of the pulmonary arteries. If the pressure is slightly increased up to around 30 mm of mercury, there is a dilatation of all the vessels both at the base and at the upper portion of the lungs.

At a pressure of around 30 to 40 or perhaps 50 mm of mercury, it is usually possible to see a difference between the width of the vessels in the upper portions of the lungs and the lower portions. This results from the hydrostatic pressure on the vessels at the bases. At the next phase of mitral stenosis, when the pressure is still higher, above 60 and up to 90, we see constricted or narrowed vessels, first in the lower portion of the lungs, then in the upper portions.

In addition, there are secondary changes in the lungs, the best known of which are so-called Kerley's lines. They appear as horizontal lines in the periphery of the lungs in cases where the venous pressure is about 20 mm of mercury. In order to make a more specific diagnosis, we have to use more specific methods, usually angiocardiography. With this technique we can demonstrate tricuspid stenosis, mitral insufficiency, filling defects such as thrombus and the primary and secondary manifestations of aortic valve disease. We can also demonstrate the condition of the coronary arteries, abnormalities of which can influence the decision for surgical operation. If the coronary arteries are severely compromised by aortic valve disease, the stronger the indication that something ought to be done to the valve.

DR SMITH: At this point, Dr. Maurice Sokolow is going to comment more generally about the problem of patient selection for valve replacement.

DR. SOKOLOW*[3]: The report on this patient is an example of a typical modern investigation to define the anatomical lesion and its severity. As Dr. Smith said earlier, the treatment also represents a real triumph for modern cardiac surgery, particularly since both valves were insufficient. Anyone who managed patients of this kind only two or three years ago realizes now that the reconstructive procedures available then were totally inadequate when the valves were fixed, retracted, fused and calcified.

Our purpose today is to bring the subject of valvular replacement up to date. Advances are so rapid that papers published one or two years ago are already out of date. Chart 1 illustrates the fact that reconstructive procedures for the mitral valve which were in vogue in the first part of the 1960's are for practical purposes no longer in use.[9] Reconstructive procedures have become progressively less common and practically all operations now involve replacement of a valve, the single exception being repair of pure mitral stenosis with a pliant valve. We have come to appreciate the concept of the "fixed" valve and to realize that separation of valve lesions into pure stenosis and pure insufficiency is unrealistic in most cases, since these valves are, in fact, both stenotic and insufficient. Consequently, valvulotomy has usually failed and replacement is preferred in most instances. This change in operative approach applies also to aortic valve disease, and, for practical purposes, no reconstructive procedures are being done in most of the larger centers for acquired aortic valve disease.

Why have valve replacements become so prevalent? Why are the reconstructive procedures not being done? Primarily because of the high rate of re-stenosis and because despite a high surgical mortality the clinical and hemodynamic response to reconstructive operations were poor. Chart 2, taken from Logan in Edinburgh (1963), demonstrates the progressive rise in the percentage of

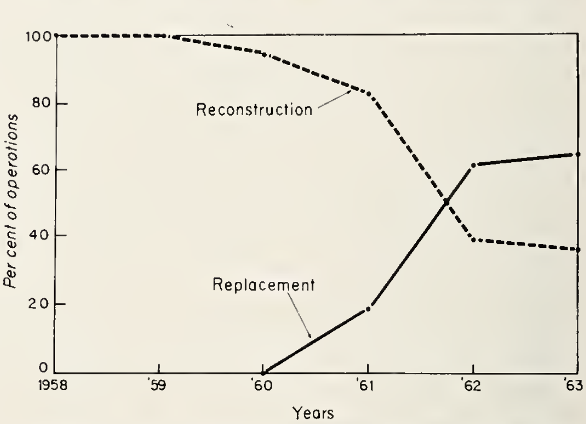


Chart 1.—Open operation for acquired mitral valve disease. The percentage of operations done by either reconstructive or replacement techniques through December 1963 is shown. From Ellis et al.[9] Reproduced by permission of the author and of the New England Journal of Medicine.

[*3] Maurice Sokolow, M.D., Professor of Medicine.

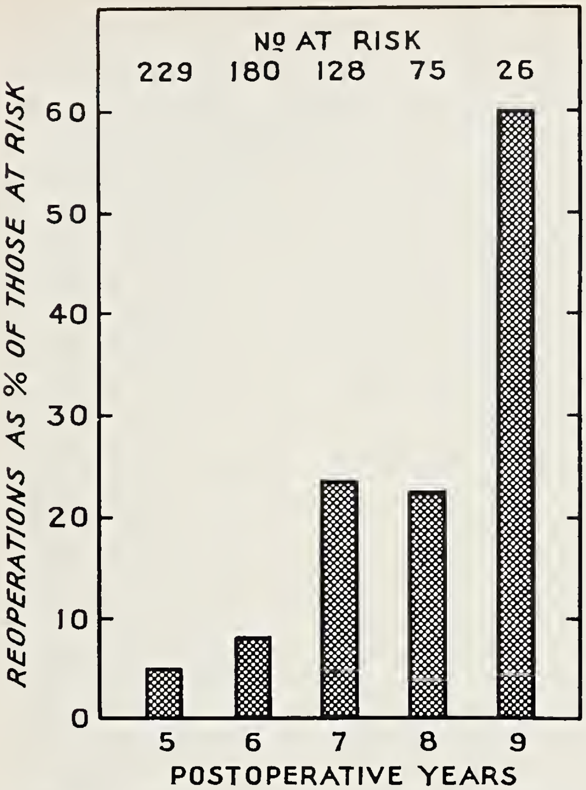


Chart 2.—Incidence of re-stenosis. From: Logan, Andrew, et al., Lancet, p. 444, 3 March 1962. Reproduced by permission of the author and the journal.

re-stenosis in patients who had valvulotomy for mitral stenosis. One can see as the years pass that a higher proportion of all patients who had mitral valvulotomy are re-operated upon, especially within five years. Furthermore, the survival of patients with both mitral and aortic reconstructive procedures proved disappointing. Bruce's group in Seattle recently published a report on a series of 31 patients with aortic valve reconstruction; only one patient was alive five years later.[11] One of the reasons for these poor results is that when the aortic valve ring is grossly calcified, it takes a magician not a surgeon to make a satisfactorily working valve.

From the literature of 1964 and 1965, I have tabulated the current status of the hospital mortality from valve replacements (Table 2). We have included the late mortality even though the deaths occurred in some instances a year or two after operation, since it has become apparent, particularly in aortic valve disease, that late sudden deaths from arrhythmia may occur when the heart remains large even after a satisfactory surgical procedure.

The operative mortality rate has progressively decreased and is now reaching a range where operation can be recommended to patients with severe disease, such as the patient presented this morning. The rapid decrease in surgical mortality reflecting the efforts of a team that has learned to handle the surgical and postoperative problems, has been the major factor in the increased number of valve replacements. In Dr. Benson Roe's experience here, practically all of the deaths from valve replacement have occurred in patients with severe aortic stenosis. There have been no deaths in the last 27 cases of mitral valve replacement and only three deaths in a roughly equivalent number of replacements of both the aortic and the mitral valve. Analysis of the deaths showed that half of the patients who died were over 60 years of age, 75 per cent had hearts heavier than 600 grams and 80 per cent had coronary heart disease. I think we are now in a position to say that isolated mitral valve replacement or combined mitral and aortic valve replacement can be accomplished with surgical mortality of approximately 5 per cent in patients whose dominant lesion is in the mitral valve.

Not only has mortality decreased, but there is good evidence of clinical and hemodynamic improvement after valve replacement.[4,5,10,12,14] In most cases of combined mitral and aortic valve replacement, there is a fall in pressure in the left atrium in the pulmonary artery and in the right ventricle, and a normal rise in the cardiac output with exercise. A very gratifying decrease in pulmonary vascular resistance occurs following mitral valve replacement.

As you know, many of the clinical manifestations of mitral valve disease stem from right ventricular failure secondary to pulmonary hypertension. It was feared that this hemodynamic problem would be irreversible, and it was quite heartening to find that in fact it was not. It is of interest that

TABLE 2.—*Surgical Mortality of Aortic-Mitral Valve Replacement*

Reported By	*Date*	*Number of Cases*	*Per Cent Mortality*		
			In Hospital	*After Discharge*	*Total*
Starr	1/64	129	20	9	29
Cooley	9/1/64	364	13	11	24
Lillihei	6/64	41	22	17	39
McGoon, Ellis, Kirklin	4/65	138	4	14	18
Morrow, et al.	4/64	22	—	—	17
Roe	1965	84	14	8	22

the average pulmonary vascular resistance in Braunwald's series[4] was much higher in patients with mitral regurgitation than in patients with mitral stenosis. The patient presented here with predominantly mitral regurgitation had a pulmonary vascular resistance which was quite high.

Not all patients with aortic stenosis require valve replacement. In congenital aortic stenosis, calcification is uncommon, the valves are bicuspid in 75 per cent of cases and reconstructive procedures are still being done. The results are not entirely satisfactory, however, and it may well be that as time goes on these patients, too, will have valve replacement.[16]

Selection of Patients

The major problem to be solved is reducing the mortality of operations on aortic valves, particularly in cases of valvular stenosis. This brings us to the matter of selection of patients. We obviously increase the surgical risk if we give the surgeon patients who are old or who have very large hearts. On the other hand, in the past, many of us were not willing to recommend operation for patients with less severe disease until the mortality of valve replacement had become more reasonable. I think that we are now justified in sending to the surgeon patients who have progressive symptomatic disease, with clear objective findings, but who are not yet functionally in class IV. Testing the response to graded exercise as an objective method of evaluating the functional capacity of the heart is an important preoperative assessment currently being evaluated here by Dr. Malcolm McIlroy.

One of the problems in selecting patients at an earlier stage concerns the prognosis of aortic stenosis. We used to think this was a fairly straightforward problem: In the presence of left ventricular failure, dyspnea, syncope or angina, the prognosis for life in the absence of operation was about two years.[1] However, in a relatively small but well studied group of patients, the average life expectancy in patients with these signs and symptoms was two to four years; and a smaller group was still living after a considerably longer time.[15] Syncope has been shown also to be present in some patients with mild to moderate aortic stenosis, and absent in some patients with severe disease.[14] Thus, if one takes the group as a whole, the majority who have these symptoms die quite early; but some do not, and it is very difficult to differentiate these groups.

I would not like to leave you with the impression that all our problems are solved; the mortality is still substantial and the long-term history of the "survival" of the Starr-Edwards valve is unknown. For these reasons we do not operate on patients with slight disease. Complications include leaks around the valve, slight residual stenosis, hemorrhage and infection. In addition, patients have to take anticoagulants unremittingly because thromboembolic phenomena are a continuing problem.

In recent years, we have begun to appreciate that aortic stenosis is a more complicated lesion than involvement solely of the valve. In 1957 Russell Brock (now Lord Brock) in England drew attention to subaortic stenosis, which may be a discrete or diffuse obstruction proximal to the valve resulting from hypertrophy of the septum.[6] Then in 1961, Barrett-Boyes in New Zealand and others emphasized the importance of supra-aortic obstruction. Patients with different anatomical lesions may present with relatively similar physical findings and so it is necessary to identify the anatomic cause of the left ventricular outflow lesions by angiocardiography and left heart cardiac catheterization. There are distinguishing features which we do not have time to go into, but at least superficially the patients when first observed have symptoms of obstruction of left ventricular outflow, and for this reason aortic and left ventricular angiocardiography is essential in making the anatomical diagnosis. The main problem is to differentiate valvular disease from subvalvular disease. Figure 7 shows an example of the most recent type of outflow obstruction to be described—supravalvular obstruction.[3] There is discrete narrowing above the valves. Patients with this abnormality usually have some hypoplasia of the aorta as well, which makes the operation difficult. Another feature of these patients is a characteristic facial expression with a wide forehead and a very small mandible.[3] They not only look mentally retarded, they are. One other interesting finding is that practically all have bilateral peripheral pulmonary artery stenosis.

We have not said much about mitral stenosis, but perhaps we can summarize by saying that at present our surgeons perform closed mitral valvulotomy when we are sure that there is pure mitral stenosis with a pliant valve. By that I mean that the diastolic murmur is long, the first sound is snapping with an early opening snap; a systolic murmur, if present, is short and of low intensity,

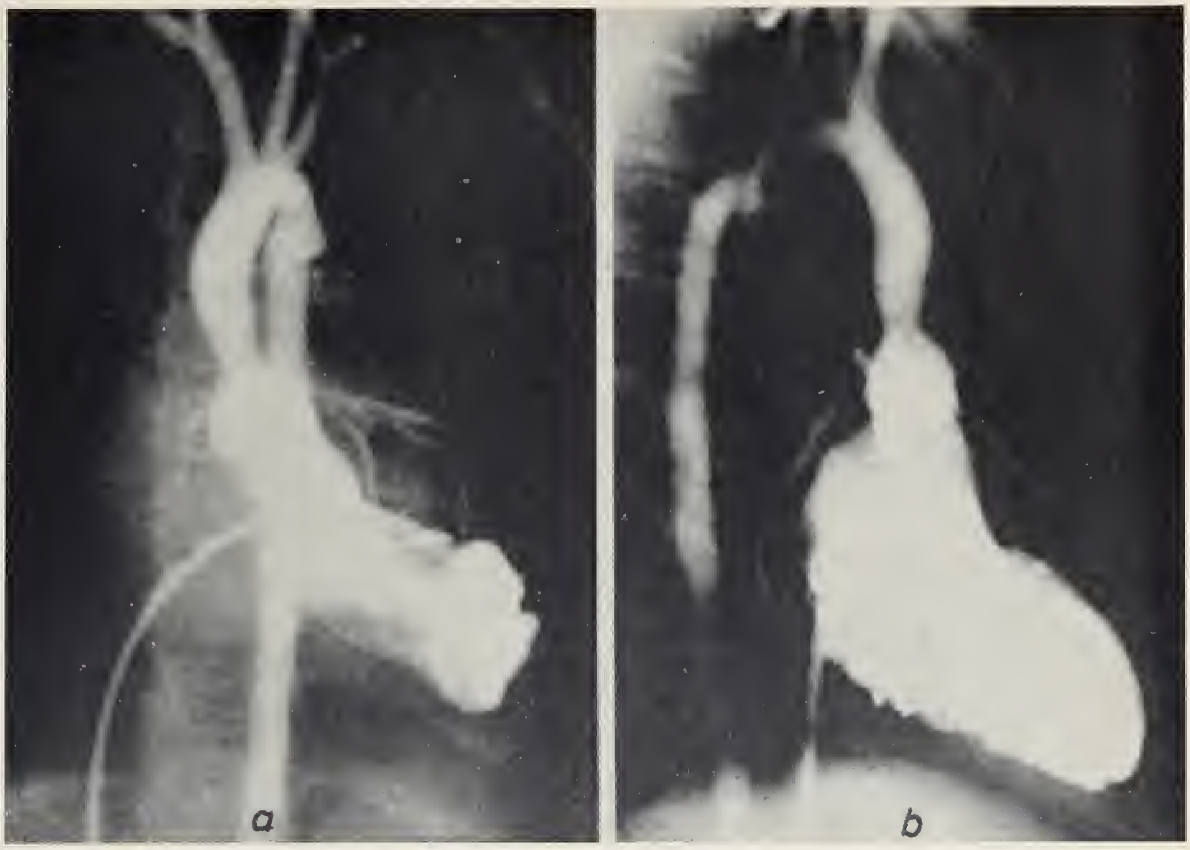


Figure 7.—Left ventricular angiocardiogram following transeptal left heart catheterization. Postero-anterior view shows supravalvular aortic stenosis. Marked bulging of aorta at the site of the ligamentum arteriosus can be seen in all patients with this syndrome. Lateral view shows stenosis above the sinus of Valsalva. From Beuren et al.[3] Reprinted by permission of the author and the journal.

and there is no third heart sound and no left ventricular hypertrophy in the absence of hypertension or an aortic lesion. We exclude mitral insufficiency with a left ventricular angiography, and we exclude aortic insufficiency with an aortic angiogram.

In concluding we must state that although we have made great strides in the past five years, we must continue our research efforts toward better prosthesis (possibly homografts), better coronary perfusion, better understanding and treatment of the physiologic derangements which occur during and following cardiac operations. Diagnostic methods must become even more precise than now so that the surgeon, knowing all the anatomical defects, can plan his corrective procedures more accurately. As the surgical mortality and morbidity falls, and if the long-term history of valvular or homograft replacement is favorable, corrective surgical procedures will be undertaken earlier in the course of valvular disease, preventing cardiac hypertrophy and failure.

DR. SMITH: Thank you, Doctor Sokolow. We are now very pleased to have with us the man whose skill has in large measure been responsible for the excellent results of cardiac operations in this hospital over the last year. I believe Dr. Benson Roe will comment about the problems encountered in operations on the heart, with some emphasis on the postoperative problems as well.

The Surgical Procedure

DR. ROE*[4]: It is indeed gratifying to be emerging from the crushing disappointments and frustrations of the developmental phase to our present level of performance.

If one were to watch the work in our operating room on an ordinary day, I think he would be deceived by what appears to be an orderly, rapid, simple execution of procedures. If the observer were a surgeon, he would say to himself that the technical challenges seldom seem to be overwhelm-

---

*Benson B. Roe, M.D., Chief of Cardiac Surgery.

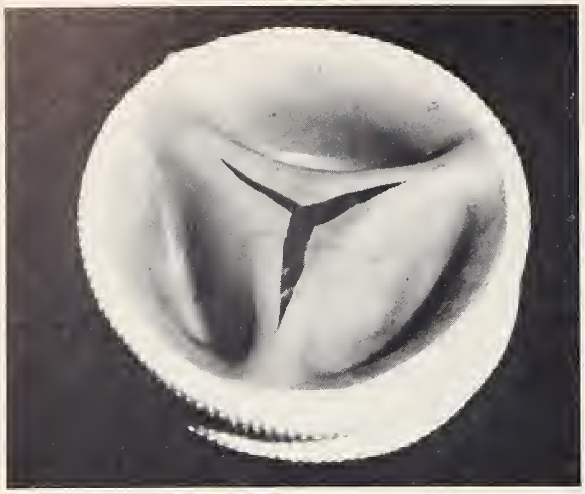


Figure 8.—Valve with leaflet mechanism.

ing. I certainly had this impression when visiting the various clinics around the country. Like other surgeons, I was confident of my capacity to master the techniques and to direct the performance of the rest of the team without too much difficulty. However, it is impossible to emphasize adequately that this is indeed a real team effort. This team concept is foreign to the traditional surgical approach of years gone by when the surgeon did everything. What he did with his hands determined the patient's course; what happened before and after was relatively unimportant. This has changed. The team must be composed of people who are interested, trained, dedicated, and who have enough practice to remain proficient. Unless the program maintains a case load that keeps the team on its toes, good results cannot be expected, regardless of how skilled or able the surgeon himself may be.

The history of surgical therapy of acquired heart disease is a long and interesting one. The first successful attempt to open an aortic valve was done in 1912. The development of open heart surgery and the successful treatment of congenital heart disease antedated the effective treatment of acquired heart disease. The early open heart operations for acquired heart disease had some colorful and interesting chapters, but the word "failure" must be written in big red letters across all of these efforts until quite recently.

We have been interested in the development of valve prosthesis for many years. Figure 8 shows a valve with a leaflet mechanism which we introduced a number of years ago. In fact, I think that, in this country, the prosthetic replacement that is

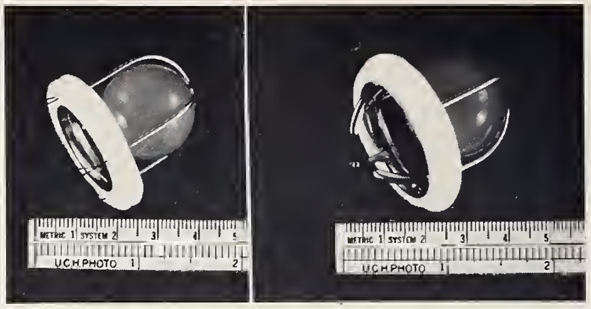


Figure 9.—*Left,* Starr-Edwards ball and cage valves. *Right,* Cutter-Smeloff ball valve.

in longest continuous operation is an earlier model of this valve which was placed in a man in August 1960. He is still alive and well. However, many subsequent ones failed for numerous reasons, which led to the withdrawal of this valve. It may be reintroduced, however, for it has many features which are superior to those of ball valves. It does not obstruct, and it is not noisy.

Another valve is the well-known Starr-Edwards ball and cage device (Figure 9). It is made from a single casting of polished stainless steel, and it has a teflon cloth cuff into which the sutures are placed, this providing a cushioning effect against the tugging that occurs on abrupt deceleration of the flow of blood when the valve closes. One of the technical problems is that of maintaining fixation. The pounding along the line of insertion would result in disruption were it not for the cushioning of this impact provided by the cuff.

Figure 9 shows a new valve which we have been using recently. This was developed by Smeloff and is made by the Cutter Laboratories. As one can see, the ball is not larger than the orifice but fills it in the closed position; the ball is seated against small counterprongs instead of being seated against the ring. This adds a potential source of clot formation but there is a distinct improvement in the size of the central orifice. The cage is shorter, which is a valuable feature for small hearts.

We believe that the currently available mitral prosthetic devices are functionally less perfect than those for the aorta and that every effort should be made to preserve the mitral valve even if only temporarily. The prosthetic tricuspid valve performs least well and the diseased valve is usually amenable to annuloplasty. Tricuspid annuloplasty was performed in 12 patients last year and replacement of the tricuspid valve along with the other two valves was necessary in only two instances. We have had five other patients in whom we replaced the mitral and the tricuspid but we left the aortic valve alone.

We have not yet solved all our problems, but we can now offer safe, effective treatment for symptomatic acquired valve disease. The risk associated with it, although not negligible, is not much greater than the risk associated with simple abdominal operations in the same group of patients.

REFERENCES

1. Abelmann, W. H., and Ellis, L. B.: Severe aortic stenosis in adults: evaluation by clinical and physiologic criteria, and results of surgical treatment, Ann. Intern. Med., 51:49, 1959.

2. Arvidsson, H.: Angiocardiographic determination of left ventricular volume, Acta. Radiol., 56:321, 1961.

3. Beuren, A. J., Schulze, C., Eberle, P., Harmjanz, D., and Apitz, J.: The syndrome of supravalvular aortic stenosis, peripheral pulmonary stenosis, mental retardation and similar facial appearance, Amer. J. Cardiol., 14: 471, 1964.

4. Braunwald, E., Braunwald, N. S., Ross, J., Jr., and Morrow, A. G.: Effects of mitral valve replacement on the pulmonary vascular dynamics of patients with pulmonary hypertension, New Engl. J. Med., 273:509, 1965.

5. Bristow, J. D., Farrehi, C., McCord, C. W., Starr, A., and Griswold, H. E.: Clinical and hemodynamic observations after combined aortic and mitral replacement, Circulation Suppl. I, April 1965, p. I-67.

6. Brock, R.: Surgical treatment of aortic stenosis, Brit. Med. J., 1:1019, 1957.

7. Carlsson, E.: Angiocardiographic measurements of the cardiac ventricles in dogs, (Accepted for publication in Acta Radiol.).

8. Dodge, H. T., Sandler, H., Ballew, D. W., and Lord, J. D.: The use of biplane angiocardiography for the measurement of left ventricular volume in man, Amer. Heart J., 60:762, 1960.

9. Ellis, F. H., Jr., Callahan, J. A., McGoon, D. C., and Kirklin, J. W.: Results of open operation for acquired mitral valve disease, New Engl. J. Med., 272:869, 1965.

10. Hohn, A. R., Van Praagh, S., Moore, A. A. D., Vlad, P., and Lambert, E. C.: Aortic stenosis, Circulation Supl. 3, December 1965, p. III-4.

11. Johnson, W. P., Cobb, L. A., Bruce, R. A., and Merendino, K. A.: Inexorable aortic stenosis, Arch. Intern. Med., 113:705, 1964.

12. McGoon, D. C., Ellis, F. H., Jr., and Kirklin, J. W.: Late results of operation for acquired aortic valvular disease, Circulation Suppl. I, April 1965, p. I-108.

13. Milne, E. N. C.: Physiological interpretation of the plain radiograph in mitral stenosis including a review of the criteria for radiological assessment of pulmonary arterial and venous pressures, Brit. J. Radiol., 36:902, 1963. (Also, Year Book of Radiology, 1965.)

14. Morrow, A. G., Clark, W. D., Harrison, D. C., and Braunwald, E.: Prosthetic replacement of the mitral valve. Operative methods and the results of preoperative and postoperative hemodynamic assessments, Circulation, 29:2, April 1964.

15. Takeda, J., Warren, R., and Holzman, D.: Prognosis of aortic stenosis. Special reference to indications for operative treatment, Arch. Surg., 87:931, 1963.

16. Thompson, N. B., and Fisher, F. C.: Long-term postoperative followup in surgery for congenital aortic valvular stenosis, Circulation, 32:732, 1965.

California
Medicine

Editor
DWIGHT L. WILBUR, M.D.
•
Assistant to the Editor
ROBERT F. EDWARDS
For information on preparation of manuscript, see advertising page 2

Policy Committee—Editorial Board
JOHN G. MORRISON, M.D., San Leandro
MALCOLM C. TODD, M.D., Long Beach
WILLIAM F. QUINN, M.D., Los Angeles
JOSEPH F. BOYLE, M.D., Los Angeles
ALBERT G. MILLER, M.D., San Mateo
HELEN B. WEYRAUCH, M.D.
San Francisco
DWIGHT L. WILBUR, M.D., San Francisco

EDITORIAL

Quality of Health Care

THE QUALITY OF HEALTH care rendered to patients has long been the subject of formal and informal discussion among practitioners of medicine. More recently this question as it applies to patients whose care is paid for by county, state or federal agencies has come under consideration in legislative bodies and other interested groups.

Historically the medical profession more than others has been concerned with the quality of training of its practitioners and the quality of care they have supplied. Indeed the Flexner Report, which was the source work for great improvements in medical education, beginning early in this century, grew out of the American Medical Association's efforts to raise the quality of care. The continuing devotion of the medical profession to medical care of high quality is expressed in many ways and carried forward by serviceable devices—among them the AMA Council on Medical Education for the approval of medical schools and internships, the Joint Committee on Accreditation of Hospitals which reviews the facilities and the organization for care given in hospitals, the residency review committees which approve residency programs under which young physicians receive their training, and locally the various committees set up by hospital staffs themselves for tissue review and utilization review, among others, as a way to maintain a constant survey of the quality of care and the proficiency of physicians.

The determination of quality in medical care is of course largely subjective, but evaluations that can be expressed in objective terms that mean as nearly as possible the same thing to all who use them are needed—more than ever, now that "third parties" are much in the medical care picture.

Attempts to arrive at such evaluations are going forward in many places. At the First National Congress on the Socio-Economics of Health Care, sponsored by the AMA, a physician suggested ways to arrive at better conclusions by more intensive and concerted application of assessment techniques already known and used[4]; a mechanical engineer put forward a plan for developing a mathematical index that would draw together the factors relating to the quality of medical care[2]; a representative of the United States Air Force described a method for determining the medical personnel needs in relation to various kinds of medical services in Air Force facilities.[3]

The California Medical Association's own Committee on the Role of Medicine in Society has been hard at work on the question, and we believe it timely to publish a thought-provoking report prepared by the Committee [see page 486]. It has been received by the Council and the House of Delegates. No official action has been taken on it and it is presented here, as it was received by the Council and the House, for information. It is the Committee's consensus that while "quality health care" means many things to many people it is still quite possible to define the term in a standard sense and determine whether quality care is being provided and to what extent.

At present, many physicians throughout California are voluntarily serving on hospital medical staff, foundation or Blue Shield utilization review committees not only to determine the nature and amount of medical care but to maintain high quality. Reviews of this kind serve to educate the attending physician in terms of future patient treatment and in no way are they a cost control or disciplinary function for private insurance or government-financed medical care programs. Admittedly the system is not a perfect one. Much more needs to be done in developing checks and balances in quality control and utilization review. However, it is a start.

Certainly more specific guidelines need to be developed and the term "quality health care" needs to be defined so that it means the same thing to the providers of service, to patients, to insurance carriers and to governmental agencies. As to the need for guidelines and definitions, a case in point is a bill now before the California Legislature which authorizes the establishment of several prepaid medical pilot programs. The law would require that "such programs shall demonstrate different methods of providing health care services and shall emphasize methods of utilization review and preventive care, and shall provide incentive for using the most economical level of such care."[1]

There are pressing reasons for finding serviceable criteria for determining the quality and utilization of medical care, and we welcome the help and cooperation of insurers, of foundations, of Blue Shield, of governmental agencies—indeed of anyone who can contribute to a solution of this problem.

But we must not lose sight of the fact that the end purpose of these efforts is to make sure that the quality of the care of patients is kept high. Hence, the responsibility for appraising the adequacy of indices of quality and for determining how they are to be used in the patient's best interest is the responsibility, first and ultimate, of the medical profession.

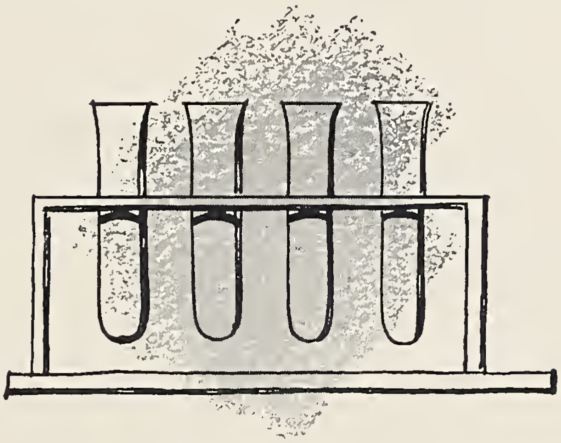
REFERENCES

1. Assembly Bill 1140, California Legislature, 1967.

2. Huber, G. P.: Assessing the quality of patient care, *in* Papers from The First National Congress on the Socio-Economics of Health Care, AMA Council on Medical Service, 22 to 23 January 1967, pp. 67-74.

3. Patterson, R. A.: Manpower requirements and productive measurement, *in* Papers from The First National Congress on the Socio-Economics of Health Care, AMA Council on Medical Service, 22 to 23 January 1967, pp. 137-140.

4. Sidel, V. W.: Techniques for assessment: Validity and limitations, *in* Papers from The First National Congress on the Socio-Economics of Health Care, AMA Council on Medical Service, 22 to 23 January 1967, pp. 51-58.



California Medical Association

NOTICES AND REPORTS

Responsibility for Quality In Health Care

A Report by the Committee on the Role of Medicine in Society, California Medical Association

Concern with the quality of medical or health care is nothing new. The American Medical Association was organized over 100 years ago for the purpose of improving the quality of the health care of the American people. Medical researchers and educators have always been concerned with pressing forward to gain new knowledge and to incorporate new information and new techniques into everyday patient care. By the turn of the century enough progress had been made so that Lawrence J. Henderson, a biochemist turned philosopher, was able to state that for the first time in history a random patient who saw a random doctor stood a better than even chance to benefit from the encounter. Since then, the body of scientific knowledge has approximately doubled with each passing decade. Research in the health sciences has been well financed and the advances have been substantial. Medical educators, organized medicine, and the pharmaceutical industry have worked together to transfer the new knowledge into actual practice. This overall effort has had an impact not only upon the level of health care, but upon society itself, which has developed rising expectations of a high quality of health care which shall be equally available to every person as a matter of his right. These expectations found expression in the health legislation of the 89th

---

This report and two others by the Committee on the Role of Medicine in Society have been received by the Council and the House of Delegates of the California Medical Association. No official action has been taken on them by either body and they are being published here, as they were received by the Council and the House of Delegates, for information.

The other two reports, "A New Relationship with Government and Others" and "Organizing for Health Care," will appear in succeeding issues of California Medicine.

Committee on Role of Medicine in Society: Gerald Besson, Burt L. Davis, Sanford Feldman, Elmer F. Gooel, Malcolm C. Todd, and Malcolm S. M. Watts, chairman; and ex officio Samuel R. Sherman and Ralph C. Teall.

JOHN G. MORRISON, M.D. . . . . President
MALCOLM C. TODD, M.D. . . . . President-Elect
WILLIAM F. QUINN, M.D. . . . . Speaker
JOSEPH F. BOYLE, M.D. . . . . Vice-Speaker
ALBERT G. MILLER, M.D. . . . . Chairman of the Council
HAROLD KAY, M.D. . . . . Vice-Chairman of the Council
HELEN B. WEYRAUCH, M.D. . . . . Secretary
DWIGHT L. WILBUR, M.D. . . . . Editor
HOWARD HASSARD . . . . Executive Director

General Office, 693 Sutter St., San Francisco 94102 • 415 776-9400
JACK CURLEY . . . . Southern California Office
1515 N. Vermont Ave., Los Angeles 90027 • 213 663-8071
RICHARD W. LEMOS . . . . Sacramento Office
1127 11th St., Sacramento 95814 • 916 444-7496

Congress, with the result that increasing attention is being focused not only upon the quality of health care, but also on its cost.

As both the cost and effectiveness of health care have spiraled upward, a growing interest in what represents good or poor medical care has developed outside of as well as within the medical profession. More specifically, those who pay the costs, be they the patient, an employer, a labor union health plan, or a government program, are seeking criteria by which they can determine whether or not they are getting their money's worth. They seek and need "standards" but do not wish to impair or destroy "high quality" in the process of standardization. Part of the difficulty is that there is no easy definition of "high quality," or of "health care" for that matter. Nor can one easily describe what is meant by, and what are the advantages and disadvantages of, "custom care," (who should be entitled to this?) "high quality care," (higher quality than what?) and "cut rate" or "supermarket" care (whatever these may be). Yet there is a clear need to know with some precision what "high quality health care" is and the circumstances which make it effective and efficient.

The Central Role of the Physician in High Quality Care

The Committee believes that the central role of the physician in the delivery and evaluation of high quality health care service to patients should receive the first emphasis in any discussion of quality. Ward Darley has stated:

"In the last analysis, the ultimate situation in medical care is the one in which two human beings are working together upon the basis of a close interpersonal relationship. This relationship is the atmosphere in which scientific and technical procedures are applied. And as scientific and technical capacities increase in their capabilities to have specific and profound effects upon basic human biology, the more important the question of this interpersonal relationship will become, not only to physicians but to all other professional personnel that will be taking increasing responsibility for patient care."*

The practicing physician has a clear responsibility that medical care be individualized and that it be of as high a quality as possible under the circumstances. The physician has a responsibility to maintain his own competence and to assist in monitoring the quality of care which individual patients receive. Physicians cannot stand aloof from the responsibility to assess the quality of this care.

Assessments of Quality Now in Progress

The record indicates that there are many ongoing assessments of quality now in progress and further shows that the effect of these methods of assessment has been to discourage substandard care and to encourage improvement even at the most sophisticated levels, with the result that overall quality continually improves year by year.

Educational and professional standards for medical schools are established by the AMA Council on Medical Education and Hospitals and by the Association of American Medical Colleges. State licensure board requirements are based upon standards set by the American Medical Association, the American College of Surgeons, the American College of Physicians, and the American Academy of General Practice. The Joint Commission on Hospital Accreditation and the Central Inspection Board of the American Psychiatric Association inspect and accredit general hospitals and mental hospitals respectively, and their requirements include controls which influence the quality of care. The Advisory Board for Medical Specialties recognizes 19 different examining boards which set the standards and requirements for some 33 separate and distinct specialties of medical practice.

In the hospital setting at least 16 methods of internal appraisal exist through which the physician's work is judged and surveillance exercised through Tissue, Utilization Review, Medical Record, Internal Audit Committees, professional activity studies, tumor registries, and the like. In California, the "Guiding Principles for Physician-Hospital Relationships" of the California Medical Association and California Hospital Association which require that these and related activities be carried on are enforced through inspection by survey teams. These techniques are now being extended to nursing homes and extended care facilities.

Quality assessment of care rendered in physicians' offices is the subject of much study and concern. The ongoing function of the professional review and grievance committees of the component medical societies have been augmented in several counties by feasibility studies to evaluate the quality of medical care in individual medical practice which were carried out under the auspices of the California Medical Education and Research Foundation.

---

*Conference on "Medical Care of the Future," University of California Medical Center, San Francisco, 18 to 19 June 1966.

In summary, a multiplicity of endeavors resulting either from professional self-initiative or in concert with legislative efforts which involve official health agencies, characterizes the self-imposed responsibilities and obligations assumed by physicians and their professional organizations in enhancing, improving, and evaluating the quality of medical care provided to the public. These efforts represent the dynamic and vigorous actions of a viable profession which is at once responsive to its own needs and to the desires of all people for the application of the most modern skills and medical advances to the preservation and prolongation of life.

A Role for Organized Medicine

It is noteworthy and significant that government so far has recognized existing professional standards in lieu of precise fixed government regulation, wherever such professional standards exist. But any weakness or incompetence in this area of professional responsibility will not be overlooked for very long by an expectant public or by a government that will be footing much of the bill. Voluntary techniques encourage progress by permitting a greater degree of experimentation with new methods while eliminating the substandard as rapidly as possible. Fixed regulation by government tends to embed mediocrity into bureaucratic concrete and should be discouraged in medical care.

The Committee believes that to protect quality, organized medicine must now build upon the precedents to be found in existing standards for professional ethics and conduct, and in accreditation and certification of educational programs and various kinds of health care facilities. These programs must be expanded and whatever new mechanisms are necessary must promptly be developed to ensure that the profession continues to fulfill its traditional responsibility for the standards and definitions of quality in *every* aspect of health care.

Some "Meanings" of Quality

The terms "quality" and "health care" mean many things to many people. Webster defines quality as "the degree of fineness." The World Health Organization defines health as a "state of complete physical, mental, and social well-being, and not merely the absence of disease." Both of these definitions have some claim to be authoritative. However, to most people, as to Alice in Wonderland, they mean "what I want them to mean." Thus, there are many meanings which reflect the interest, background and motivation of those who use these terms. A few examples follow.

To many patients, high quality health care means prompt availability of a well trained, up-to-date physician who will take a personal interest and who will assume responsibility for their care, together with availability of up-to-date facilities and adequate financing. The physician is more concerned with the appropriateness of the care which the patient receives and the factors governing motivation and sense of responsibility on the part of both physician and patient. The consumer or the purchaser of health care tends to give emphasis to more units of care for less money, comprehensiveness of coverage, continuity of care, and provision of health care with little or no cost barrier to the individual. Some social scientists and planners of public policy equate "high standards" with the right to, or availability of, care or even to the "social consciousness" of the physician, a concept with many interpretations. Others emphasize health maintenance, prevention of disease, diagnosis and treatment of illness, injury or emotional disturbance, or rehabilitation of the sick, injured, mentally ill, or even the socioeconomically deprived.

It should be noted that for many dedicated individuals the search for criteria and standards against which the quality of health care can be measured or assessed carries the implication that somehow there can be created a more or less perfect health care instrument with standardized components which will in some way solve the problem of providing every person high quality health care and with an equal opportunity for health. This of course is illusory for a number of reasons. But even if an intrinsically perfect instrument or system were created, it would fail of its purpose simply because there are no such thing as standardized or perfect patients, nor is there any such thing as high quality health care that does not take into account the individuality of the patient and the individuality of his health needs. Thus, paradoxically, and perhaps tragically for those dedicated idealists, the more "perfect" the instrument or system of health care, the less likely it will be to satisfy the individual needs of patients and thus the less perfect will be the quality of the health care they receive.

A Description of High Quality Health Care

While it is quite impossible to define precisely high quality health care in terms of static standards or measurement against fixed criteria, the Committee believes it is possible to describe what is meant by high quality health care with sufficient precision so that it is possible to tell whether or not it exists and is being provided to those who need it.

It is suggested that high quality medical care is up-to-date from the standpoint of scientific knowledge and technology, that it is available without significant geographic, social, economic, or political barrier to those who need it, that it embodies a high degree of motivation on the part of the patient, the physician, the health care team, on the part of those who are responsible for equipment and facilities, community and regional services, the health care programs and plans themselves, as well as those who are responsible for the financing. High quality health care is effective in terms of meeting the needs of the individual patient, it is efficient in terms of not being wasteful, in terms of value received for the money expended, and it is satisfying to the patient and satisfactory to the physician, to all others involved in the health care industry, to those who provide the financing, and to the public itself.

Parameters and Elements of High Quality Health Care

The Committee suggests that the above description recognizes the futility of efforts to measure quality in health care with precise accuracy since anything which can be measured objectively must by its very nature be something other than the quality which in the final analysis can only be judged subjectively. The description does suggest that it may be useful to consider quality in terms of certain parameters and in terms of the more important ingredients or elements of health care.

The Committee would like to suggest and seek to delineate six "parameters" of quality which might be applied to each of the elements or ingredients of health care:

1. *Competence:* Competence refers to the scientific and technologic adequacy and to the judgment, integrity, skill, and capability to utilize currently available knowledge in health care.
2. *Availability:* Availability refers to the extent to which a resource in health care is accessible or obtainable at the time and place and in the amount needed.
3. *Motivation:* Motivation refers to the drive or incentive to receive, render, or otherwise provide health care services of high quality.
4. *Effectiveness:* Effectiveness refers to the extent to which the aim or purpose is accomplished, be it prevention, diagnosis, treatment, rehabilitation, health maintenance, or any other aspect of health care.
5. *Efficiency:* Efficiency refers to performance in the sense of producing the desired result with a minimum of expenditure or waste of talent, time, money, or materials.
6. *Satisfaction:* Satisfaction refers to fulfillment of reasonable expectations of those who receive, render, or otherwise provide for health care.

The Committee suggests that the following are essential ingredients or elements of health care and that they be defined as below:

1. *Patient:* A patient is an individual who receives health care, but who may or may not use it to good advantage. A potential patient is an individual who needs health care, who may or may not know that it is available, and who may or may not choose to seek it. The potential patient becomes a patient at the point at which he seeks help.
2. *Physician:* A physician is a doctor of medicine who is engaged in patient care, research, or health education.
3. *Health Team:* This team has yet to be defined, but refers to the various allied professions and other health care personnel and the way they relate to the patient, to the physician (or physicians) and to one another.
4. *Equipment and Facilities:* Equipment and facilities includes not only health care equipment and laboratories, but also hospitals, doctors' office facilities, dispensaries, extended care facilities, convalescent homes, nursing homes, and the like.
5. *Health Care Services:* Health care services include not only physicians' services as listed in the CMA Relative Value Studies, but the patient care services of allied health professionals, preventive services, screening surveys, health maintenance and rehabilitation services including community and regional health care services.

6. *Plans and Programs:* This includes both governmental and private or voluntary health care plans or programs, or where applicable, combinations of these.

7. *Financing:* Financing includes both public and private financing, from voluntary and personal sources.

"CMA Quality Assessment Index for Health Care"

It is suggested that an effort be made to develop a "CMA Quality Assessment Index for Health Care" which would provide a description of the quality of health care and its various components and some sort of gradation of value just as in the "CMA Relative Value Studies" professional services are identified and a value placed upon them. It is suggested that such a quality index could be based upon the description of high quality health care to be found above through the application of the "parameters" to the "essential elements" of health care.

It is believed that when this concept is examined it will be found to encompass most of the existing approaches to the assessment or "measurement" of quality and to recognize the various viewpoints of those who are properly concerned with this problem. In application it could prove a valuable tool to identify areas of strengths and weakness in quality, and if kept up-to-date, it should provide a powerful ongoing incentive to up-grade quality.

Conclusion

1. The future of health care depends upon whether the concept of high quality can be maintained as a reality as the costs rise and the need to control costs increases, or whether "quality" will deteriorate to a meaningless slogan.

2. The practicing physician has a central role and responsibility in the core situation where the individual patient does or does not receive a high quality of medical care. The medical profession as a whole, and organized medicine in particular, therefore have a central role and responsibility for high quality health care.

3. It is clear that the responsibility for achieving the goal of high quality of health care for every person is widely shared and includes the patient, other members of the health care team, the health care industry, the voluntary and government health care plans, public and private sources of financing, together with educators, scholars in many academic disciplines, and many others, as well as the physician.

4. The Committee believes that there is now an unusual opportunity to develop a "CMA Quality Assessment Index for Health Care" as a tool to document quality and to encourage its betterment on a continuing and ongoing basis. It believes that such an index could be constructed which would assess the components of health care, utilize the "parameters of quality" described in this report, and, by means of statistically significant sampling maintain an ongoing record of the state and effectiveness of the control on "high quality health care" in California. Such a mechanism would be at once authoritative yet flexible and would encourage improvement in "high quality" rather than regulation and control at some fixed standard. The parameters, essential elements, and components could be adjusted periodically to reflect progress in science and society.

A Statement of CMA Goals

A Supplementary Report to the House of Delegates of the California Medical Association, 15 April 1967

At its 29 October 1966 meeting, the CMA Council voted "to prepare an annual broad statement of CMA objectives for presentation to the House of Delegates." The following statement of goals is an effort to comply with the Council's wishes regarding the annual establishment of goals for the Association.

WE HAVE RECOGNIZED that Medicine is an integral, interrelated and interdependent segment of the total population which it serves.

A state medical association exists as such because of the unique geographic, social, economic and political nature of the state itself. California is unique in its geography, its population size and varied composition, its industry, its economy, its educational facilities and its state government—especially its state government's involvement in, and regulation of the health professions and health care services.

The constitutional purposes of our medical association are the improvement of the art and the science of medicine and the betterment of the public health. It follows then, that the objectives of the California Medical Association in the years 1967-68 are: (1) **To improve the practice of**

medicine, and (2) **to aid in bettering the health of the public, accomplishing these tasks within the unique and changing social, economic and political environment of the State of California.**

Our major continuing goal, therefore, is to fulfill these objectives to the best of our abilities, utilizing as intelligently as possible all of the many diverse resources available to us.

In order to achieve this goal we must ask ourselves and others some searching questions to which we need complete and informative answers in order to formulate realistic programs and to guide our activities:

- *How can we effectively improve the art and science of medicine in California in the next few years?* Where are we now doing a "really good job?" Where are we "spinning our wheels" and wasting our resources? In what areas are we failing to do all that we can to help fulfill the needs and desires of our physicians to practice the art and science of medicine efficiently, productively and with the satisfactions which should be inherent in the practice of medicine?

- *What are the problems of public and community health toward the solution of which medicine could make more significant contributions?* In which of the large social problems affecting the health of the people are we providing effective medical guidance? In which of these problem areas are we failing for lack of direct involvement or for other causes? Are we identifying real community health problems and offering realistic proposals for their solution?

Evaluation of CMA Activities

Finding answers to just these few questions would require a thorough examination, not just of the profession itself, but of our relationships with other professions, paramedical personnel, medical schools, labor, industry, insurance carriers and with government at all levels. It would require realistic evaluation of the effectiveness of our relationships with county medical associations, the AMA, specialty societies, the Academy of General Practice and closed panel organizations. It would require critical evaluation of our communications techniques, our continuing education programs, group insurance, and retirement programs, to mention just a few.

Several of our committees and commissions and our Bureau of Research and Planning are scratching the surface of these problems, but in a manner often lacking coordination and clear recognition of purpose.

One of our immediate goals for 1967-68 might well be a complete critical evaluation of all CMA programs and liaison activities to determine whether or not they are realistically beneficial to either medicine or the public. The Council could conduct this evaluation using existing committees, with heavy reliance upon the Research Department.

Expansion of Governmental Relations Function

As such an evaluation progresses, it may become apparent that our manpower and other resources should be more heavily committed to some of our programs and withdrawn from others. For example, it is becoming increasingly apparent that there needs to be developed a strong, closely-coordinated government relations functions within the framework of CMA. This is made necessary by the vast involvement of government in the health care field and the complex, far-reaching implications of existing governmental programs and those planned for the future. Using larger numbers of well-informed physicians and an expanded staff, we must reach directly into the heart of those governmental advisory committees and administrative departments concerned with health matters. This we must do on a continuing basis, armed with current facts developed by meaningful research and supported by effective communications and by functional liaison with allied groups working in the same field.

This operation would not and should not replace nor duplicate the vital functions of the Public Health League, AMPAC, or CALPAC in their political and legislative activities. It would serve to support those functions and be coordinated with them, yet reach beyond their present spheres of influence into the vast maze of the administrative branch of government.

Modernization of Fact-Finding Methods To Utilize Resources Effectively

As one contemplates such a necessary redeployment of our available resources, it would seem wise for us to know just what these resources are. This would appear almost axiomatic, yet our state organization has never developed a systematic, effective way of maintaining, for example, an up-to-date listing of those physicians in various county societies who are particularly interested in, and working on, problems of school health, voluntary health agencies, medical schools, or other specific areas. We have no ready index file of those influential leaders in industry or labor or education who could be called upon to assist medicine in a particular problem area. In this day and age, business and government achieve their objectives with the use of computer systems, rapid-fire changes of communications emphasis and of personnel deployment. Our medical organizations are lagging far behind.

An immediate goal for this Association should be the development of an inventory of our resources both within and outside of our immediate membership, utilizing modern, rapid methods of gathering this information and keeping it up-to-date and ready for immediate use. Your officers and executive staff should be charged with this responsibility. They will need to be supported by communications, research, and other segments of the Association.

Representation of All Segments of the Profession

It is perhaps more important now than ever before that CMA represent and speak for virtually all California physicians. We have yet to define clearly the mechanisms by which CMA can become a desired and valued place for participation by employed and salaried physicians in medical schools, closed panel groups, government services and training programs. **A "task force on membership" should be charged with study and recommendations on this subject.**

Enlargement of Physician Participation

Much of the work of an effective state medical association must be done by its physician members. Increasing demands on the profession have required untold hours, days and weeks of time from a handful of men and women at the helm of our organization, and to only a slightly lesser extent from various Councilors and members of committees, bureaus, task forces and commissions. There is still not nearly enough expenditure of time by well-informed physicians on all of the work the profession is or should be doing. The process of being well-informed on any given program or subject is itself tremendously demanding of time.

The pressures of practice, hospital committees, specialty societies, continuing education, community activities, family life and the need to make a living are such that only a few doctors have studied and worked in depth on the serious problems which confront the profession. **An immediate goal should be to increase significantly the number of physicians who will spend the necessary time to do this work, on a continuing basis on behalf of the Association and the profession.**

A major barrier to this goal is appropriate and reasonable compensation. We have long since passed the time when we could expect a physician to give up large segments of time away from his practice without some form of reasonable compensation. **This is a problem to be faced squarely. A realistic formula needs to be found promptly.**

Expansion of Member Services

The CMA should provide the individual physician all possible assistance in carrying out his responsibilities in his practice and in his community. We currently are doing a great deal for the individual doctor indirectly through his county society, and directly through CMA: Communications, physician placement, group insurance, the Keogh retirement plan, continuing education, and representation with government and many private sector associations. We could be doing much more. Today's physician faces many problems which did not exist 10 years ago. New problems have arisen from complex new laws and regulations; new drugs; new concepts in business, communications, and health facility planning; new insurance programs; new responsibilities for hospital medical staffs, extended care facilities and the organization of hospital-based practice groups.

Do we know what information and assistance our doctors need and want in tackling these new problems? **One of our goals should be to find out what physicians need in these areas and to do our best to fulfill these needs.**

Continued Assurance of Highest Quality Health Care

In pursuing these objectives and carrying out the many programs of CMA, we must not lose sight of the fact that the primary function of the profession and of related health personnel is to provide to all the people in the State of California, the highest possible quality of health care, delivered in such a manner as to afford satisfaction to the public and the health professions, while conserving the time and resources available to each.

Our Association must continue its studies to identify conditions and situations which detract from maximum performance in the rendering of health care services—and to make suggestions for improvement. In cooperation with Blue Shield, insurance carriers, foundations and county societies we must continue our activities to assure ourselves and the public that the profession is indeed providing high quality of care, at a reasonable cost and with a minimum of abuse. It would appear that we are doing a commendable job. **Our goal should be that of continued and improved performance, which will justify our own pride in our distinguished profession as well as high public esteem for doctors of medicine.**

SUMMARY OF 1967-1968 CMA GOALS

1. To work toward fulfillment of our primary objective of improving the practice of medicine and the health of the public within the unique and changing environment of the State of California, utilizing as intelligently and as effectively as possible all of our available resources.

2. To evaluate all CMA programs and activities to determine their effectiveness in achieving significant benefit to the public and the profession.

3. To expand and strengthen CMA's governmental relations activities.

4. To modernize our methods of fact-finding so that we may better utilize the resources available to CMA and component societies.

5. To launch a concerted effort to bring all segments of the medical profession into CMA membership.

6. To increase active physician participation in the affairs of organized medicine, providing adequate financial compensation when it appears justified.

7. To expand necessary and desirable services for the individual physician to help him practice more efficiently, to reduce frustrating annoyances and to increase his satisfactions from practicing his profession.

8. To continue studies and promote cooperative programs to assure a high level of performance in providing quality medical care to all of the people of California.

Controls on Outlays For Medi-Cal Care

COMPROMISE AMENDMENTS offered by the Committee on Conference on A.B. 583 were adopted by both houses of the legislature 4 May 1967 and sent to Governor Reagan for his signature.

The conference amendments removed those persons who are not Group I or Group II (categorically aided or certified medically indigent) recipients from the $18.25 per month formula for computing the maximum amount available for care of California Medical Assistance Program recipients.

The compromise amendments also give the Health and Welfare administrator the authority not only to limit the scope of services to be provided, but also to "limit the rates of payment for such services."

The compromise puts two controls on county expenditures for noncategorical aid recipients. One control requires counties, after 30 June 1967 to have prior authorization from the state for any increase in cost because of new standards for medical aid and care, scope and level of services, or obligations for medical care.

The other limits the state's obligations to pay counties for authorized increased costs to $44 million in the 1967-68 fiscal year. The amount of this maximum obligation would be fixed each subsequent year by the legislature in the budget.

In Memoriam

Churchill, James Forrest, San Diego. Died 21 April 1967, aged 85. Graduate of Rush Medical College, Chicago, Illinois, 1906. Licensed in California in 1916. Doctor Churchill was a retired member of the San Diego County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✤

Clark, George Sherman, Los Angeles. Died 20 April 1967, in Los Angeles, aged 89, of carcinoma of prostate. Graduate of McGill University Faculty of Medicine, Montreal, Quebec, Canada, 1906. Licensed in California in 1923. Doctor Clark was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

✤

Clark, Roscoe Loyal, Pittville (Lassen County). Died in McArthur, 9 April 1967, aged 73. Graduate of the University of Pennsylvania School of Medicine, Philadelphia, 1919. Licensed in California in 1937. Doctor Clark was a retired member of the Sacramento County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✤

Cox, Thomas Joseph, San Rafael. Died 17 April 1967, in Greenbrae (Marin County), aged 63. Graduate of the University of California School of Medicine, Berkeley-San Francisco, 1931. Licensed in California in 1931. Doctor Cox was a member of the Marin Medical Society.

✤

Enterline, Joseph Donald, Reedley. Died 5 April 1967, in Fresno, aged 51. Graduate of Harvard Medical School, Boston, Massachusetts, 1943. Licensed in California in 1952. Doctor Enterline was a member of the Fresno County Medical Society.

✤

Farquharson, R. E. Hughes, Redwood City. Died 15 April 1967, in Los Angeles, aged 46. Graduate of the University of Alberta Faculty of Medicine, Edmonton, Alberta, Canada, 1944. Licensed in California in 1949. Doctor Farquharson was a member of the San Mateo County Medical Society.

✤

Giovannetti, Rinaldo P., San Francisco. Died 6 April 1967, in Oakland, aged 85. Graduate of Cooper Medical College, San Francisco, 1911. Licensed in California in 1912. Doctor Giovannetti was a member of the San Francisco Medical Society.

✤

Grant, William Thomas, Sierra Madre. Died 13 April 1967, aged 60. Graduate of the University of Toronto Faculty of Medicine, Ontario, Canada, 1931. Licensed in California in 1937. Doctor Grant was a member of the Los Angeles County Medical Association.

✤

Gray, Frederick Lyle, Long Beach. Died 3 May 1967 in Long Beach, of ventricular fibrillation, aged 50. Graduate of University of Cincinnati College of Medicine, 1952. Licensed in California in 1953. Dr. Gray was a member of the Los Angeles County Medical Association.

✤

Guenther, Robert Edward, Midway City. Died 11 March 1967, in Anaheim, aged 57, of a malignancy. Graduate of Kirksville College of Osteopathy and Surgery, Kirksville, Missouri, 1959. Licensed in California in 1960. M.D. degree from California College of Medicine, 1962. Doctor Guenther was a member of the Orange County Medical Association.

✤

Hamilton, Michael J., Redding. Died 26 March 1967 from injuries in a fall while climbing Mt. Whitney, aged 42. Graduate of Creighton University School of Medicine, Omaha, 1952. Licensed in California in 1960. Doctor Hamilton was a member of Shasta-Trinity County Medical Society.

✤

Irvine, A. Ray, Beverly Hills. Died 14 April 1967, in Beverly Hills, aged 87. Graduate of Medico-Chiurgical College of Philadelphia, Pennsylvania, 1906. Licensed in California in 1923. Doctor Irvine was a member of the Los Angeles County Medical Association, a life member of the California Medical Association, and a member of the American Medical Association.

✤

Johnstone, Rutherford Thompson, Los Angeles. Died 27 April 1967, in Sepulveda, aged 73, of arteriosclerotic thrombosis. Graduate of University of Pittsburgh School of Medicine, 1925. Licensed in California in 1936. Doctor Johnstone was a member of the Los Angeles County Medical Association.

✤

Langton, John Nesbitt Kent, Berkeley. Died 13 April 1967, in Carmel, aged 53, of myocardial infarction. Graduate of the University of California School of Medicine, Berkeley-San Francisco, 1950. Licensed in California in 1950. Doctor Langton was a member of the Alameda-Contra Costa Medical Association.

✤

Mayes, Duward Lacy, Santa Ana. Died 29 March 1967, aged 63. Graduate of the College of Osteopathic Physicians and Surgeons, Los Angeles, 1941. Licensed in California in 1941. M.D. degree from California College of Medicine, 1962. Doctor Mayes was a retired member of the Forty-first Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✤

Niemand, Frederick, George, Fullerton. Died 22 April 1967, in Anaheim, aged 62, of renal failure. Graduate of the College of Medical Evangelists, Loma Linda-Los Angeles, 1930. Licensed in California in 1930. Doctor Niemand was a member of the Orange County Medical Association.

✤

Nierenberg, Harry H., Beverly Hills. Died 14 April 1967, in Los Angeles, aged 55, of coronary artery disease. Graduate of the University of Illinois College of Medicine, Chicago, 1934. Licensed in California in 1945. Doctor Nierenberg was a member of the Los Angeles County Medical Association.

✤

Owen, Lincoln L., Vista. Died 26 April 1967, in Vista, aged 54. Graduate of the College of Medical

Evangelists, Loma Linda-Los Angeles, 1945. Licensed in California in 1945. Doctor Owen was a member of the San Diego County Medical Society.

◆

Pearl, Felix L., San Francisco. Died 31 December 1966, in San Francisco, aged 69, of myocardial infarction. Graduate of University of California School of Medicine, Berkeley and San Francisco, 1923. Licensed in California in 1923. Doctor Pearl was a member of the San Francisco Medical Society.

◆

Ryan, Andrew Morris, San Francisco. Died 13 April 1967, in San Francisco, aged 61, of heart disease. Graduate of Tulane University School of Medicine, New Orleans, Louisiana, 1931. Licensed in California in 1931. Doctor Ryan was a member of the San Francisco Medical Society.

◆

Schneider, John L., Imperial Beach. Died 5 May 1967, in an automobile accident in Mexicali, aged 43. Graduate of California College of Medicine, Los Angeles, 1953. Licensed in California in 1954. M.D. degree from California College of Medicine, 1962. Doctor Jones was a member of the San Diego County Medical Society.

◆

Schwartz, Joseph Leon, Sr., Los Angeles. Died 22 April 1967, in Los Angeles, aged 73, of cardiovascular disease. Graduate of the College of Physicians and Surgeons, Medical Department, University of Southern California, Los Angeles, 1916. Licensed in California in 1916. Doctor Schwartz was a member of the Los Angeles County Medical Association.

Seal, Alfred L., Beverly Hills. Died 13 April 1967, in Los Angeles, aged 61, of acute myocardial infarction. Graduate of Tulane University School of Medicine, New Orleans, Louisiana, 1929. Licensed in California in 1944. Doctor Seal was a member of the Los Angeles County Medical Association.

◆

Smith, Charles E., Berkeley. Died 19 April 1967, in San Francisco, aged 62, of myocardial infarction, coronary sclerosis. Graduate of Stanford University School of Medicine, Palo Alto-San Francisco, 1931. Licensed in California in 1931. Doctor Smith was a member of the Alameda-Contra Costa Medical Association.

◆

Wagenseller, Samuel Miller, Ahwahnee. Died 26 March 1967, in Ahwahnee, aged 64. Graduate of Johns Hopkins University School of Medicine, Baltimore, 1931. Licensed in California in 1951. Doctor Wagenseller was a member of the Merced-Mariposa Medical Society.

◆

Weeks, Walter G., Oakland. Died 11 April 1967, aged 33. Graduate of Columbia University College of Physicians and Surgeons, New York City, 1958. Licensed in California in 1963. Doctor Weeks was a member of the Alameda-Contra Costa Medical Association.

◆

Wyers, Robert E., Whittier. Died 25 April 1967, in Whittier, aged 65, of bronchogenic carcinoma. Graduate of the University of Arkansas School of Medicine, Little Rock, 1927. Licensed in California in 1929. Doctor Wyers was a member of the Los Angeles County Medical Association.

PUBLIC HEALTH REPORT

Lester Breslow, M.D., M.P.H.
Director, State Department of Public Health

A SHARP RISE in the incidence of malaria took place nationwide during 1966 with a total of 678 cases reported, more than four times that of 1965 and 12 times that of the record low in 1959. More than 700 cases were reported in the first four months of 1967.

This trend is reflected in the figures for California. There were 105 cases recorded in this State with onset in 1966, more than twice as many as in 1965 and 10 times as many as in 1960. In 1967 to date there have been more than 50 cases.

Of the 105 cases in 1966, 84 occurred in military personnel. The rest were in civilians, of which most were merchant seamen, Peace Corps volunteers and students returning from Africa, Asia and South America. Eighty of the 84 military personnel contracted the disease in Vietnam.

Among the 80 cases from Vietnam, the distribution of species of plasmodia is of significance: 53 *Plasmodium vivax,* 38 *P. falciparum* (*malignant tertian*), five mixed, one each *P. malariae* and *P. ovale,* and six unknown.

Indigenous malaria was eradicated from the United States following the initiation of the National Malaria Eradication Program, in 1947. Before that time the incidence of this disease had steadily declined from the latter part of the 19th century. Periodic resurgences occurred, mostly in the southeastern states, until the development and widespread use of effective insecticides, particularly DDT.

Records of the latter half of the 19th century describe malaria as a major public health problem in California, especially in the Central Valley. The first mosquito control work was begun in this state in 1903 at San Rafael. The State Legislature passed a Mosquito Abatement District Act in 1915. During the next 10 years the reported incidence of malaria declined from a case rate of 17.9 to 1.7 per 100,000 population.

While the natural transmission of malaria has been eliminated from the United States, the possibility of reintroduction will remain as long as the disease is present in other parts of the world. Civilian travel and military operations are exposing a substantial number of persons to malarious areas throughout the world. Modern transportation is so swift that often the first symptoms of the disease do not occur until after the person has returned to the United States.

Experience gained during and after World War II and the Korean conflict suggests that it is unlikely that the natural transmission of malaria will become reestablished in this country if the medical and allied professions remain alert to this possibility. Prevention depends upon effective surveillance (recognition and reporting of cases), prompt treatment of patients, and intensive abatement efforts against the mosquito vectors.

In the United States there have been only two documented episodes in the past 10 years of malaria acquired by mosquito transmission from a person who contracted the disease abroad. In both instances the organism was *P. vivax* and the first symptoms occurred in May 1966. The patients were siblings living in close proximity to military personnel returning from malarious areas in Asia and neither had a history of travel abroad or of blood transfusions. Transmission was probably by mosquito transmission from a person who had become infected in another country.

Delay in onset of primary attacks after transmission is a common occurrence with vivax malaria. Typical of *P. vivax* malaria is a longer period before symptoms occur, a longer interval between relapses and greater adaptation to temperature range within the United States, than with other forms. The interval between initial infection and primary attack may be as long as two years. Malaria due to *P. vivax* rarely persists longer than four years and is not often fatal. Suppression of primary attacks and the treatment of either primary attacks or relapses are usually successful with chloroquine, mepacrine and primaquine.

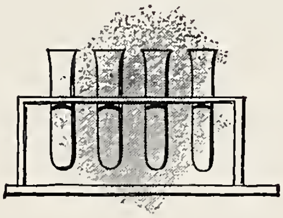
During 1966 and 1967 an increasing number of cases of falciparum malaria (malignant tertian) have occurred among military personnel serving in Southeast Asia. As noted above, many persons with this severe type of the disease have come back from abroad. When caused by *P. falciparum,* the disease rarely persists longer than a year; however, the cerebral form of malaria may occur. The interval from transmission to initial symptoms is usually less than one month but it can be longer. Deaths have occurred in the United States among military personnel and civilians in the past year due to falciparum malaria which was recognized after return from Southeast Asia. The symptoms are often bizarre and include unexplained fever, delirium, irrational behavior, hypotension and shock. Early recognition and prompt therapy are essential.

Strains of *P. falciparum* which are resistant to chloroquine were discovered in 1961. At present a variable but significant number of cases of falciparum malaria are caused by strains resistant to most antimalarial drugs so that combinations of drugs are needed for treatment in many persons. For any form of malaria the outline of therapy contained in the American Public Health Association Report *Control of Communicable Diseases in Man,* Tenth Edition, 1965, contains a regimen most likely to be effective. For the suppression or prevention of malaria a combination of chloroquine diphosphate or sulfate (300 mg base) and primaquine (45 mg) each once weekly on the same day and continued for eight weeks after leaving an endemic area will be successful for most persons. This will not suppress symptoms or prevent recruldescences in some instances of infection with *P. vivax* or resistant *P. falciparum* strains. Diaminodiphenylsulfone has recently shown promise for the prevention of both types of malaria when administered daily in conjunction with the weekly doses of chloroquine and primaquine.

With the continued rotation of military personnel from the Far East, more cases of malaria are expected to occur in California this year. Current information from mosquito abatement districts and other sources indicate a high mosquito density early in the year. In California the two species of anopheline mosquitoes capable of transmitting malaria are *A. freeborni* and *A. punctipennis.* Adult anophelines are found from June to October, primarily in the Central Valley and Sierra foothills from about Red Bluff on the north to Fresno on the south. A high sustained density of mosquito vectors, certain climatic conditions and the presence of cases of malaria could lead to transmission in California.

Malaria can also be acquired through blood transfusion Two such cases, each due to *P. falciparum,* occurred during the past year. In both instances the donor was a returnee from Vietnam in whom the diagnosis of malaria became established after he had given blood for transfusion. One of these episodes occurred in Northern California.

Physicians should be aware of the increasing numbers and variable manifestations of malaria and keep the possibility of this in mind so that therapy can be begun early.



WOMAN'S AUXILIARY

to the California Medical Association

Membership

MEMBERSHIP IS THE LIFE of any organization and the effectiveness of any organization is measured by the number and type of members it comprises.

The Woman's Auxiliary to the California Medical Association has been the largest state Auxiliary in the country since 1961. We now have some 9,200 members and with about 23,000 physicians in California our potential seems a great deal more. Of course, our goal is 100 per cent.

With the greatly increased income that this increase would bring, much more could be accomplished. Not only would there be more money to aid medical education, health careers and similar projects, but we should be able to turn to other things which would increase our influence and effectiveness. Time and effort now used to recruit members could be used more valuably on Auxiliary projects.

How can 100 per cent membership be accomplished? There must be a way but obviously we need the complete cooperation of physicians and their medical associations. Every physician should encourage his wife to be a member of the Auxiliary—she is wanted and needed.

The Woman's Auxiliary to the California Medical Association was organized at the request of the Association to complement its organization. All projects of the Auxiliary have the approval of CMA. Our prime objectives are to assist the Association in its program for the best medical care for all.

Membership in the Auxiliary provides an opportunity for physicians' wives to work toward a common goal with others who have the same interest. This association with others promotes the best interest of this group by cultivating friendly relations, encouraging mutual understanding among physicians' families.

As a member of the Auxiliary, a physician's wife will be kept well informed on all pertinent questions and will be able to speak intelligently on matters of medicine. A well informed physician's wife is an asset to medicine as well as to each physician individually.

The Auxiliary helps in many fields such as public relations, legislation, Physicians' Benevolence and the American Medical Association Education and Research Foundation. For the last two years, the California Auxiliary has received a citation for contributing more than other Auxiliary to AMAERF. Taking care of physicians and their wives in time of need is part of Auxiliary's help in Physicians' Benevolence.

Many of our county Auxiliaries give scholarships for allied health professions in the form of loans, grants or scholarships. We believe in free enterprise.

We find ourselves competing with hospital Auxiliaries and other voluntary agencies for the time of our capable physicians' wives. Many of the wives are leaders in those fields, so we must make them realize that we need them also.

If you are a physician who is a member of the California Medical Association, I hope you will urge your wife to join the Auxiliary. We need her support and her help.

MRS. EDMUND MAHON
Membership Chairman
39 Mirabel
Mill Valley 94941

LETTERS *to the Editor*

OUR MEMBERS WILL undoubtedly appreciate your factual summary of the resolutions passed by the House of Delegates of the California Medical Association since 1962 as revealed in the editorial entitled, "Where We Stand on the Question of Therapeutic Abortion," in the April 1967 issue of CALIFORNIA MEDICINE. As you know, the resolutions recommend increased relaxation of the law pertaining to legalized abortions. You obviously are quite enthusiastic about modifying the present law and accordingly make some very biased and exaggerated comments on this very controversial issue.

In your editorial, you abandon objectivity, or refuse to recognize reality when you state, "Yet, paradoxically, today's physicians still find themselves bound to outdated abortion legislation which perpetuates needless suffering and fosters poor medical practices." This fantastically ridiculous claim warrants critical analysis and comment, far beyond the intent of this letter. If the obstetricians and gynecologists on your editorial board, however, agree with this exaggerated statement, I hope that you would permit a guest editorial or a series of articles presenting both sides of this question, in order to place this problem in proper perspective.

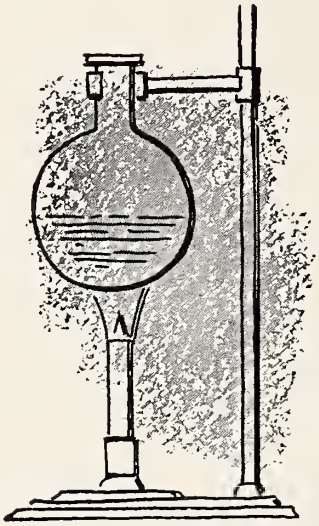
Furthermore, I notice you frequently refer to the "products of conception." You are perhaps, consciously or unconsciously, trying to avoid thinking of the fetus as an embryonic living human being. There is very scientific reason for the latter concept. Nowhere in your article do you suggest that this "product of conception" is the beginning of a new life, different from any other human being living or dead. You unashamedly justify the murder of this human being on the basis of a "substantial risk to the physical and mental health" of the mother, vague as are all these terms.

Dr. Herbert Ratner states in an article scheduled for publication in the *Illinois Medical Journal,* May 1967 issue, "After taking up the cudgel against the battered child syndrome, are we now going to pick up the curette and replace the extra-uterine with the intra-uterine battered child syndrome?" The inconsistency of our attitude or the hypocritical nature of it, must be apparent to many laymen. We probably have not made a profound enough study into the serious implications of the resolutions passed by the House of Delegates since 1962. Thus, we are in a very vulnerable position from the public relations standpoint, not to mention the disservice that we are guilty of, in our innocent acceptance of the pro-abortionists' propaganda. The official position of the California Medical Association on this problem has shocked many members of our organization to the realization that there is a serious communication gap between us and the business of our Delegates and Officers. Thus, many of us have already signed a petition protesting the official California Medical Association viewpoint. It is hoped that an appropriate remedial policy will be established in the future to inform our membership of the various resolutions being considered by the California Medical Association in advance of the annual meetings of the House of Delegates so that our membership may more effectively voice their opinions.

It would appear appropriate, again, to quote Dr. Herbert Ratner, a Public Health Officer, who rightfully states ". . . let us not forget that the liberty to abort makes the physician more like a god than is good for him." Again, in quoting Dr. Joseph DeLee, an eminent obstetrician whom we all admire, he writes: "At the present time, when rivers of blood and tears of innocent men, women, and children are flowing in most parts of the world, it seems silly to be contending over the right to live of an unknowable atom of human flesh in the uterus of a woman. No it is not silly. On the contrary, it is of transcendent importance that there be in this chaotic world one high spot, however small, which is against the deluge of immorality that is sweeping over us. That we the medical profession hold to the principle of the sacredness of human life and of the right of the individual even though unborn is proof that humanity is not yet lost . . ."

In the Beilensen Abortion Law, we are dealing with very basic concepts of life and the extermination of life, rather than life and the preservation of life. The latter has been the guiding philosophy of the medical profession since the time of Hippocrates. The present peculiar trend to reverse our traditional philosophy is not, I pray, the consensus of our membership. You must open the pages of CALIFORNIA MEDICINE to an appropriate literary debate and a discussion of these very very basic issues. In the future, also, the House of Delegates must reconsider their position on these resolutions that they passed since 1962.

EDWARD J. WIATER, M.D.



INFORMATION

Poverty in the United States As Defined by Federal Standards

A Report of the Bureau of Research and Planning, California Medical Association

■ *The subject of this Socio-Economic Report is of tremendous importance to the medical profession because physicians should be aware that future programs for the expansion of health care services will be based and, in fact, are being based upon information which this Report contains. The relationship between poverty and accessibility of health care services is therefore quite direct. So, too, will be the impact upon the profession and the organization of medical practice.*

The 1966 amendments to the Poverty Act are concerned with neighborhood health centers and a vast array of other programs which will touch every physician and every community which can be identified by the standards indicated in this Report as low income, poor, or near poor. For this reason the California Medical Association Committee on Welfare Medical Programs, among several others concerned with aspects of this problem, is trying to alert every county medical society of developments as well as of the responsibilities they should assume in working with the Office of Economic Opportunity and other community organizations in providing guidance and leadership in structuring programs compatible with the interests of the public and the health care professions.

This Report on poverty presents a current and prospective view of the problems and issues to be faced. Unless physicians see the relationship and join in a community effort to aid in resolving an issue which underlies public policy, we shall be looking back five or ten years from now to point out that we failed to take advantage of opportunities to assist in the development of a rational system of medical care for low-income groups.

Individual physicians, component medical societies on a grass-roots level and CMA as a state organization should all be concerned with and aware of the facts.

Samuel R. Sherman, M.D., *Chairman*
Bureau of Research and Planning

Background

AMERICA'S "FORGOTTEN POOR" have, in recent years, received their fullest and most vivid exposure. Examination of the extent, nature, selectivity and relative permanence of poverty, despite surroundings of increasing prosperity, have aroused both government and private concern, resulting in a multiplicity of programs for action. Among them are those undertaken by the Office of Economic Opportunity, along with other agencies at various levels of national and local government. Since health care is one of the major concerns of a number of these programs, it will be of interest to the Medical profession to review the data which provide the rationale for such massive undertakings. This Report is based on the most recent account by the Social Security Administration of the extent of poverty in the United States,* which notes that, although impressive gains have been made, "there was yet much (poverty) to view with alarm."

In 1965 the Social Security Administration developed two criteria of poverty to bring some coherence and common definitions to the extensive data collected annually since 1959 by the Federal government. (See appendix for glossary of terms.) Using these criteria, numbers and characteristics of the "low-income" population over the five-year period 1959-64, a period distinguished for other groups by its rising prosperity and generally high employment, were analyzed. The problem was to determine if during this period the group had diminished or grown, or changed in consistency or habitat, in anticipation of later comparison with those data collected after the effects of the recently initiated government programs have been felt.

Changes in Low-Income Group, 1959-1964

Of the 60 million households in March 1965 (counting as a separate unit every family group and every person living alone or with nonrelatives only), 12 million or 20 per cent were defined as poor. An additional 4½ million units had incomes low enough to qualify as near-poor. Thus, amidst increasing abundance and prosperity, at least 34 million Americans lived in a state of deprivation. Included in this group were from 21 to 31 per cent of the Nation's children and from 31 to 43 per cent of the aged.

The total number of persons with low-income status fell approximately five million and 5 per cent during the period 1959-64, this drop occurring almost exclusively in the lowest income category. While the poor decreased from 38.9 million or 22.1 per cent in 1959 to 34.1 million or 18.0 per cent in 1964, the near-poor changed only slightly from 15.8 million or 9.0 per cent in 1959 to 15.7 million or 8.3 per cent in 1964. This would indicate at least some upward shift economically within the low income groups.

Poverty is created through a selective process, with some groups always more prone than others. Therefore, any real improvement in the nation's economic well-being should ideally provide greater equalization in the differential risks of poverty among different groups. The groups that are still the most likely to be poor include the non-white, the aged, rural families, large families, and those with a woman as the head.

In most of the groups the story remains the same—still more improvement needed! In fact, the circumstances of some have actually deteriorated over the period, particularly where more than one "poor-prone" characteristic appears. For example, in 1964, 76 per cent of all non-white families with five or more children were poor, compared with 71 per cent in 1959. The two major hazards of the non-white child are a broken family or the low income of the father if he is present. One-third of the half-million non-white families with five or more children in 1964 had a woman as their head and one-third had an unemployed male head. Half of the remaining families—one-third of all—had a male head who was fully employed.

Large families, particularly if vulnerable on other accounts, are more susceptible to poverty. In 1964 more than four out of ten families with a non-white head or a female head had less than the minimum standard, as did three out of ten farm families. These characteristics, combined with other high-risk variables, compounded the chances for an inferior economic status. This situation is reflected in the 69 per cent of all families with three or four children, headed by a woman, that are classed as poor and the 84 per cent with five or more children, that are found in the lowest category.

Among non-white families in 1964, while under one-fourth of those with no children under 18 were poor, over three out of four of those with five children were so classified. This compares with approximately one in ten and three in ten, respectively, among white families.

---

*Recounting the poor—A five-year review: Social Security Bulletin, 29:20-37, April 1966.

Reprint requests to: 693 Sutter Street, San Francisco 94102.

The unfortunate fact is that among all households headed by a woman, 44 per cent were in poverty in 1964, a relatively scant improvement from 50 per cent in poverty in 1959. The rate of poverty among households headed by a man fell from 18 per cent to 14 per cent in this period. As of 1964, therefore, a household headed by a woman had three times as great a chance of being poor as the household headed by a man.

The nation's rural population in 1964 had almost three in ten labeled poor, as compared with under two in ten chances for the city dweller, despite the fact that the criterion for a rural family is set 30 per cent below that of its urban counterpart. However, the incidence of poverty among the farm families dropped 12 points, from 41 per cent to 29 per cent, during this period, while it dropped only four points, from 23 per cent to 19 per cent, during the same period among city dwellers.

The age group 65 and older has the highest incidence of poverty in the population. Higher still is the subgroup of aged who live alone. During the five-year period, while the total number of poor decreased by five million, the total number of aged poor increased by 300,000, one reason being an increase in the proportion living alone. Because the last five years has brought substantial increase in the number of aged, however, there was a decrease in percentages of the aged who are poor. The rate of poverty for aged unrelated individuals decreased significantly from 68 per cent in 1959 to 59 per cent in 1964. When broken down by race, however, the reality of this decrease is more evident. While the poverty rate among the white aged fell from 67 per cent to 57 per cent between 1959 and 1964, among the non-white aged it actually increased from 78 per cent to 79 per cent, a fact clearly illustrating that the programs and prosperity that have assisted the white aged population, at least through 1964, did not reach the non-white aged.

The Monetary Gap to Fill

If the government were to subsidize all 34 million persons defined as poor in order to raise them above the poverty threshold, an expenditure of $11.7 billion, as of 1964, or 2.4 per cent of the Nation's personal income would have been required. Little improvement is likely, however, as long as expansion of the general economy moves at about twice the rate of the income of the poor, as has been the case in recent years. During the five-year period, while national personal income increased by 29 per cent, the number in poverty decreased by 12 per cent and their unmet income need by 15 per cent. As a group, the poor in 1964 had to manage on incomes totaling 59 per cent of estimated need, compared with 56 per cent in 1959.

The more favorable position of the aged poor in 1964 reflects to some extent the fact that three out of four persons aged 65 and older were receiving social security benefits, compared with three out of five in 1959.

The Poverty Problem in 1964

Since 1959 the poor have diminished by 1,200,000 persons. The most susceptible age groups are still the young (under 18) and aged (over 65), who comprise 60 per cent of those designated poor or near poor, while accounting for only 49 per cent of the total population. For many in this group, particularly children from fatherless homes, employment cannot be a solution. Fourteen per cent were 65 or over and over 14 per cent were under age six. As many as 31 per cent of the aged were living below the poverty line. Among all children 18 and under, 20 per cent are growing up in an environment of poverty and nearly half of these with five or more siblings. Forty-one per cent of these children had fathers or mothers working full time, but were still unable to make enough, enough being $65 per person per month. Including the near poor (which raises the outlay to 90 cents per day for food) would swell the poor aged group by 12 per cent and the nation's poor children by 11 per cent.

This still excludes the "hidden poor," those 65 and older living with more fortunate relatives rather than alone below the poverty line, and subfamilies who, if living alone, would qualify as poor. Including these would raise the number in poverty to 37 million and the total who are low income to 53 million. Of subfamilies who would have been poor on their own, however, half of those headed by a man and three-fifths of those headed by a woman kept off the poverty register by living with a non-poor relative.

Age and Poverty

Age and poverty are highly correlated, as has already been suggested. This is intensified somewhat by the tendency for this characteristic to be accompanied by others wherein poverty is an above average occurrence. For example, there is a high correlation between sex and poverty, women often having less education and lower earning power than men. Also, the aged have a preponderance of women, particularly women living alone, a variable likewise related to poverty. Few of the aged are employed, and social security, which covers only three-fourths of the aged, is but a fraction of their former wage. The fact that the number of aged living alone has increased may also be interpreted as an improvement position, in that they are enabled to maintain a household at all. It is significant that three times the percentage of aged poor live alone than of the aged non-poor. While living alone might have been the cause of their final impoverishment, however, it can not be assumed that they had a choice. It should be pointed out that, with the lower poverty index criterion for the aged, the aged in general have fewer expenses and may by then own their own homes, etc. Also, savings are not included in the income evaluation. In 1959, 97 per cent of the men 65 and over and 75 per cent of the women had some money of their own saved. By 1964 the proportion with some other income was 98 per cent for the men and 82 per cent for the women. The significance of this fact should not be exaggerated, however, since in 1962, data taken from an earlier Social Security Administration survey* the median assets less home equity were still only $3,000 for married couples and less than $1,000 for the unmarried aged. It was in fact, as low as $310 for unmarried men without OASDI and $160 for unmarried women without OASDI.

The improvement in the position of the aged runs parallel and is in large part accountable to the extension of social security protection, including both the increase in benefits and in number of eligible beneficiaries, particularly women and non-white, and in higher earning records of those receiving it. The average payment to an aged beneficiary in this period increased from $74 per month to $79 per month. It was estimated that about 35 per cent of all aged social security beneficiaries in 1965 were living in poverty, as defined by the poverty criterion. An additional 38 per cent would have been poor except for their benefit checks. Only about one-fourth of all beneficiaries could have lived above the poverty line in the absence of social security benefits.

In 1964, all families with an aged head derived 25 per cent of all their income from social security payments, and about half from earnings. Aged families classed as poor, on the other hand, received 60 per cent of their income as a group from social security benefits and only 16 per cent from earnings, the rest coming from such sources as veteran benefits, dividends and rents, public assistance, contributions from persons outside of home, etc.

Youth and Poverty

Nearly 15 million of the 34 million poor in 1964 were under 18 living in families, 40 per cent of which were non-white and slightly more of which had five or more children. Almost 15 per cent of these children came from homes where the head was fully employed the year around, half of them as laborers, service workers or farmers. Only 8 per cent of them had professional, technical or clerical jobs, thus suggesting that education for better paying jobs might be one way to combat this problem. Among these families the tendency is to marry and reproduce early, exposing others to the same hopeless environment from which they will experience the same difficulty emerging, because of the same handicaps accrued by living in it. One in three of the poor age group 16 to 21 were neither high school graduates or in school, while one in seven of the non-poor in this age group were neither. There is thus a pattern to drop out of school, though education is often the only way to overcome one's low status. The cycle is thus perpetuated. Those in the age group 18 to 19 who leave school before graduating are more likely to marry, possibly as an escape from family surroundings. Twenty percent of the dropouts in this group are are married, as compared with 11 per cent of the high school graduates. Income differences between those without and with a high school diploma increase with age. The sad fact is that while the dropout earns comparatively less with age, his family responsibilities and obligations become comparatively greater.

Conclusion

The problem of poverty is particularly acute today because of earlier neglect. Since there is still much to be discovered about the life patterns of the poor, long-term effects of deprivation can only be speculated upon, but the prospects appear to be dim. In the area of health care, it has been well established (see *Socio-Economic Report,* Vol. VI, No. 8, September 1966, U.S. Consumer Expenditures for Health Care, 1950-1964) that the utilization of medical care decreases with a decrease in income and education and an increase in family size. In addition, low-income groups are less likely to be insured than those who are better off and reports show that the insured receive about 50 per cent more medical care than the uninsured.

---

*Assets of the aged in 1962: Findings of the 1963 survey of the aged: Social Security Bulletin, 27:4, November 1964.

Thus, society is faced with finding solutions to alleviate or to correct those conditions resulting from or inducing poverty. Public policy is becoming more clearly defined as public concern is reflected in these manifold programs for the poor.

Appendix

These measures of poverty and low-income, defined in 1965 by the Social Security Administration, are based on the amounts needed by families of different size and composition to purchase nutritionally adequate diets at minimum cost when no more than a third of the family income is used for food. The food estimates are derived from the Department of Agriculture economy food plan for emergency use or when funds are very low.

Low-income: Those persons classified as either "poor" or "near poor" are included in this category. It is substantially below (by about 50 per cent) the "modest but adequate" level described by the city worker's family budget, developed and priced by the Bureau of Labor Statistics.

Poor: The poverty line, which is still above the line of mere subsistence, considers family size, composition and farm/non-farm residence. From data compiled by the Council of Economic Advisors, a four-person non-farm family in 1964 was defined as poor if its money income for the year was less than $3,130. (Median income for the country then was $7,490.) For farm families the line is 30 per cent lower. This works out to about 70 cents a day per person for food, with twice as much again for other expenses.

Near poor: Still within the low-income category, this group, located just above the poor, earns approximately a third more than they do. In 1964 the upper limit was set at a yearly income of $4,075 (and lower limit, the poverty line of $3,130), for a four-person non-farm family. Farm families had a line 30 per cent lower. Per person per day this amounts to a food budget of 90 cents and a total daily budget of $2.70.

Ethical Responsibilities in Prescribing Drugs and Devices

THE FOLLOWING STATEMENT on the ethical responsibilities of physicians in prescribing drugs and devices was adopted by the Judicial Council, American Medical Association, 12 March 1967:

It is unethical for a physician to be influenced in the prescribing of drugs or devices by his direct or indirect financial interest in a pharmaceutical firm or other supplier. It is immaterial whether the firm manufactures or repackages the products involved.

It is unethical for a physician to own stock or have a direct or indirect financial interest in a firm that uses its relationship with physician-stockholders as a means of inducing or influencing them to prescribe the firm's products. Practicing physicians should divest themselves of any financial interest in firms that use this form of sales promotion. Reputable firms rely upon quality and efficacy to sell their products under competitive circumstances, and not upon appeal to physicians with financial involvements which might influence them in their prescribing.

Prescribing for patients involves more than the designation of drugs or devices which are most likely to prove efficacious in the treatment of a patient. The physician has an ethical responsibility to assure that high quality products will be dispensed to his patient. Obviously, the benefits of the physician's skill are diminished if the patient receives drugs or devices of inferior quality.

Inasmuch as the physician should also be mindful of the cost to his patients of drugs or devices he prescribes, he may discuss with patients both quality and cost.

NEWS & NOTES

NATIONAL • STATE • COUNTY

NEW DEAN AT UNIVERSITY OF CALIFORNIA—SAN DIEGO

Clifford Grobstein, Ph.D., one of the nation's leading biologists, has been named Vice Chancellor for Medicine and Biological Sciences and Dean of the new School of Medicine on the San Diego campus of the University of California, which is to accept its first class of medical students in the fall of 1968. Dr. Grobstein succeeds Dr. Robert Tschirgi, Vice Chancellor-Academic Planning at UCSD, who has served as Acting Dean of the School of Medicine for the past year.

The new dean, who is a member of the National Academy of Sciences and a leader in the field of biology of development, joined the faculty at UCSD as Professor of Biology and Chairman of the Department on July 1, 1965. Before that he had served for two years as Chairman of the Department of Biological Sciences at Stanford University.

His appointment as Dean of the new School of Medicine is closely associated with the developing curriculum and its interrelationship with other graduate departments on the campus.

According to Dr. John S. Galbraith, Chancellor of the university, "Dr. Grobstein came to UCSD from Stanford in large part because of his interest in participating actively in the development of a new school with a different approach to medical education—that of 'interdigitation' of the general campus and the medical school departments."

Born in New York City in 1916, Dr. Grobstein attended City College of New York receiving a B.S. degree in biology in 1936. He moved to the Los Angeles campus of the University of California for graduate work and received his M.A. degree in zoology in 1938 and a Ph.D. degree in zoology two years later.

He began his teaching career at Oregon State College in 1940 as an Instructor in Zoology. During the war he was commissioned as an aviation physiologist in the U.S. Army Air Force. He returned to civilian life in 1946 as Public Health Service Senior Research Fellow with the National Institutes of Health. He later served for nearly ten years as a Biologist on the research staff of the National Cancer Institute.

Clifford Grobstein, Ph.D.

Dr. Grobstein is a Fellow of the American Academy of Arts and Sciences and a Fellow of the American Association for the Advancement of Sciences. He is a member of a number of international institutes and societies dealing with biology and embryology and has served as consultant to the U.S. Public Health Service on a number of matters, most recently on the Special Program Planning Committee of the National Institute of Child Health and Human Development.

He is past president of the American Society of Zoologists and the Society for the Study of Development and Growth. He held membership on the executive committees of the American Institute of Biological Sciences of the Commission on Undergraduate Education in the Biologica Sciences.

At UCSD Dr. Grobstein has been a member of the Committee for the Development of a Dental School and the Campus Planning Committee. He has also served as chairman of the committee to develop the first year curriculum for the School of Medicine and on the President's Advisory Committee to the Dean of the UCSD School of Medicine. He is Chairman of the San Diego Academic Senate's Special Committee on the State of the University.

ALAMEDA

Dr. John L. Gompertz of Oakland was elected president-elect of the National Tuberculosis Association at the organization's annual meeting in Pittsburgh, Pa. Dr. Gompertz is a past president of both the California and the Alameda County Tuberculosis and Health Associations and has served on the NTA board of directors since 1953.

LOS ANGELES

The American College of Gastroenterology has announced that its annual course in postgraduate gastroenterology will be given at The Biltmore Hotel in Los Angeles on 2, 3, 4 November 1967.

The faculty for the course will be drawn from the medical schools in and around Los Angeles. The subject matter to be covered will be the advances in diagnosis and treatment of gastrointestinal diseases and a comprehensive discussion of diseases of the esophagus, stomach, pancreas, liver and gallbladder, colon and rectum.

Further information may be obtained from the American College of Gastroenterology, 33 West 60th Street, New York 10023.

* * *

Dr. Joel H. Lee who recently was graduated from the University of Southern California School of Medicine, is the first recipient of the **Salerni Collegium Distinguished Student Award,** to be given annually to the student whose classmates judge him to have demonstrated greatest dedication to patient care during his years in medical school.

The Distinguished Student Award was inaugurated this year by Salerni Collegium, the 908-member support group of USC's medical school.

Dr. Lee who recently received his M.D. degree, was also voted the **George S. Herron Award** by his classmates for his singular contribution of personal interest and endeavor during his four years of medical school.

SAN FRANCISCO

Dr. Harold I. Griffeath, San Francisco, was elected president of the California Heart Association at the organization's recent annual meeting in Sacramento.

SANTA CLARA

Dr. Robert M. Shelton of Pasadena is the new president of the **Stanford Medical Alumni Association,** and Dr. William H. Clark of Palo Alto is its new president-elect.

Dr. Shelton succeeds Dr. Henry Gibbons III, of San Francisco. He is past president of the Pasadena Medical Society, a director and member of the eexcutive committee of Blue Cross of Southern California and a member of the visiting committee of the Stanford University School of Medicine.

Dr. Clark is a clinical associate professor of medicine in the Stanford University School of Medicine, and a Palo Alto City Councilman.

GENERAL

Three new members have been appointed to the **California State Board of Medical Examiners** by Governor Ronald Reagan, and a fourth has been reappointed. **Dr. Genest D. de L'Arbre** of Stockton was named to succeed Dr. James V. McNulty of Los Angeles for the remainder of a term which expires 15 January 1968. The other appointees, whose terms extend to 15 January 1971, are **Dr. Philip S. Geller** of Redwood City to succeed Dr. J. M. de los Reyes of Glendale, **Dr. Tirso del Junco** of Los Angeles to succeed Dr. Shelby Hicks of Merced, and **Dr. Forest J. Grunigen** of Los Angeles to succeed himself.

* * *

The Fifteenth Annual Meeting of the **Pacific Coast Fertility Society** will be held at Mountain Shadows Resort and Country Club, Scottsdale, Arizona, 28 September to 1 October 1967.

Dr. M. C. Shelesnyak, Weizmann Institute of Science, Rehovogh, Israel, will be one of the distinguished guest lecturers.

Further information may be obtained from the Pacific Coast Fertility Society, 5410 Wilshire Boulevard, Los Angeles, California 90036.

* * *

The American Institute of Ultrasonics in Medicine will hold its annual meeting in Miami Beach, Florida, 13 to 16 November 1967. It will be held jointly with the meeting of the Acoustical Society of America.

Further information may be obtained from Virginia Krause, Administrative Assistant, American Institute of Ultrasonics in Medicine, 4833 Fountain Avenue, Los Angeles 90029.

The Physician's BOOKSHELF

CARDIAC SURGERY—By John C. Norman, M.D., Associate in Surgery, Harvard Medical School; Boston City Hospital and Children's Hospital Medical Center Boston, Massachusetts, and 56 contributers. Foreword by Francis D. Moore, M.D. Appleton-Century-Crofts (Division of Meredith Publishing Company), New York, N.Y. 1967. 603 pages, $15 (Cloth).

This is a potpourri of cardiac diseases and their surgical treatment. If not inclusive of material, and it is not, it does include almost every possible living contributor. Because of the vast number of authors the book verges on a compilation of the recent literature, but it does answer just that need. No particular level of knowledge will derive more from it than another. Medical students, house officers, and practitioners will find something of interest. The book, especially the paperbook edition for $9.75, is recommended for a good survey of the current field.

NORMAN E. SHUMWAY, M.D.

* * *

SYNOPSIS OF DERMATOLOGY—By Wm. D. Stewart, M.D., F.R.C.P. (C), Clinical Instructor (Dermatology), Department of Medicine, Subdepartment of Dermatology, Vancouver General Hospital, Vancouver, British Columbia; Julius L. Danto, M.D., Fellow, American Academy of Dermatology and Syphilology; Visiting Staff, St. Paul's Hospital and St. Vincent's Hospital, Vancouver, British Columbia; and Stuart Maddin, M.D., Fellow, American Academy of Dermatology and Syphilology, Visiting Staff, St. Paul's Hospital, Vancouver, British Columbia. The C. V. Mosby Company, 3207 Washington Boulevard, St. Louis, Mo. 63103, 1966. 664 pages, $10.85.

This book was written for the non-dermatologist as a practical text for the diagnosis and treatment of skin disease. In an attempt to reach a happy medium between an outline and a voluminous reference work, the authors achieve qualified success.

The pertinent concepts of pathogenesis, diagnosis and treatment cover the field in depth but with a clear understandable manner that has been the hallmark of the "Synopsis" series. Archaic eponyms and obsolete terminology are refreshingly absent while newer etiologic concepts and methods of treatment including some controversial works are given appropriate discussion. The core of this text is devoted to specific dermatologic disease entities with an especially well thought out chapter on allergy. A ready reference with the commonly used drugs and the type of rashes they produce is clearly outlined in this section.

The illustrative material inludes both adequate black and white photographs of the common diseases as well as a number of truly superb color plates not usually found in a text of this kind.

Unfortunately, this synopsis lacks some of the qualities that could make it most useful to the non-dermatologist. The specific entities are grouped in a somewhat confusing manner and in many instances the reader would have to know the name of the disease or the cause of it before being able to read about it. An excellent chapter on regional diagnosis tends to compensate for this problem and greatly aids in orderly classification of disease.

The authors make a commendable attempt at providing a ready reference to treatment and include the all accepted methods of therapy lacking in most texts. However, dermatologic prescriptions which are complex and need compounding are given too much emphasis over the more readily accessible and easily remembered prepackaged medications. The section on dermatologic techniques is welcome, but could benefit with a more detailed explanation and diagraming of invaluable medical mycology and patch testing methods.

In spite of these shortcomings, this text is probably the most complete and certainly the most up to date work of its kind now available. It will be of value to the student if utilized as a basic text of dermatology. The non-dermatologist will find the book a worthwhile addition to his library, but this is a text and one should not expect an illustrated outline or diagnostic manual.

* * *

GASTRIN—Proceedings of a Conference held in September, 1964; Sponsored by the School of Medicine, University of California, Los Angeles—By Morton I. Grossman, Editor. UCLA Forum in Medical Sciences, Number 5. University of California Press, Berkeley and Los Angeles, 1966. 351 pages, $12.00.

This conference constitutes, as one of the participants remarks, a milestone in gastrin history. Dr. Grossman, as Chairman and Editor was successful in bringing together notable experts in this field of enquiry and the result, both as regards the quality of the formal presentations and the verbatim discussions, is very impressive. Professor Gregory's contribution on the chemistry of gastrins 1 and 11 retails one of the really exciting stories in physiology research, in which the pace has dramatically accelerated in the past few years, culminating in the synthesis of gastrins 1 and 11 by Professor Kenner and his colleagues at Liverpool. There are excellent papers by Dr. Uvnäs and his colleagues at the Karolinska Institute on the assay of gastrin and factors controlling its release, by Dr. Card from Edinburgh on the action of gastrin on gastric secretion and a very useful review of gastrin-like activity of tumors by Dr. Hallenbeck, to name a few of the generally excellent contributions. There are still a sufficient number of problems remaining to encourage new investigators into the field, as Dr. Grossman points out in his *envoi*. The relation of the heptadecapeptide gastrins of Gregory with the large molecule of Tauber is still undetermined; the cells in the gastric antrum and possibly elsewhere, which produce gastrin, have not been identified; the role of the vagus in relation to gastrin stimulation is unclear, to name only a few outstanding questions. This book is essential reading for anyone interested in the field.

K. B. TAYLOR, M.D.

Maybe he doesn't know when he's well off. But you might want to prescribe long-acting Novahistine LP anyway.

Two tablets in the morning and two in the evening will usually provide day and night relief by helping to clear congested air passages for normal, free breathing. Novahistine LP is formulated to provide continuous therapeutic effect for 8 to 12 hours. The decongestant ingredients help restore normal mucus secretion and ciliary activity—physiologic defenses against infection of the respiratory tract. Use cautiously in individuals with severe hypertension, diabetes mellitus, hyperthyroidism or urinary retention. Caution ambulatory patients that drowsiness may result. Each Novahistine LP tablet contains phenylephrine hydrochloride, 25 mg., and chlorpheniramine maleate, 4 mg.

NOVAHISTINE® LP

DOW **PITMAN-MOORE** Division of The Dow Chemical Company, Indianapolis mudrane®

for

- EMPHYSEMA
- ASTHMA
- CHRONIC BRONCHITIS
- BRONCHIECTASIS

The fast-disintegrating uncoated tablet gives relief in 15 minutes

Each tablet contains:

Potassium Iodide	195 mg.
Aminophylline	130 mg.
Phenobarbital, Caution: May be habit forming	21 mg.
Ephedrine HCl	16 mg.

FEDERAL LAW PROHIBITS
DISPENSING WITHOUT PRESCRIPTION

Precautions: Usual for aminophylline-ephedrine-phenobarbital. Iodides may cause nausea, long use may cause goiter. Discontinue if symptoms of iodism develop.

Iodide contraindications: tuberculosis, pregnancy.

DOSAGE

One tablet, with full glass of water, 3 or 4 times daily.

Dispensed in bottles of 100 and 1000 tablets.

MUDRANE GG—Formula, dosage and package identical to Mudrane—*except*—100 mg. glyceryl guaiacolate replaces the potassium iodide. The value of Mudrane cannot be enjoyed by a small group in which K.I. is contraindicated. Mudrane GG is prepared for this group.

MUDRANE GG ELIXIR—Four 5 cc teaspoonfuls is equivalent to one Mudrane GG tablet. Dosage adjusted to age and weight of child. Mudrane GG Elixir is for pediatric patients and those who think they cannot swallow tablets. Dispensed in pint and half gallon bottles.

WM. P. POYTHRESS & CO., INC.
RICHMOND, VIRGINIA 23217
Manufacturers of ethical pharmaceuticals since 1856

College of Medicine to Move to UC-Irvine

University of California Regents have approved relocation of the California College of Medicine from Los Angeles to the Irvine campus in Orange County.

The Regents' decision at their April 20 meeting in Riverside followed the recommendation of a special Regents' committee which conducted a five-month study of alternate sites for the college, and recommended the move to Irvine. CCM trustees had previously approved the move.

The Regents' committee, headed by Edward W. Carter, noted that the volume of patients was decreasing at the present site and that a study of the Irvine area's present and future needs "indicates that a new facility at that location could well serve the rapidly growing population."

The report also noted that the present land holdings of CCM are "entirely inadequate for a medical school" but that "at the Irvine site, land already owned by the Regents is available in ample quantity for a complete medical center."

Health Education Goals Outlined by Committee

Health education is an important part of the school curriculum, and efforts to expand and improve it with an emphasis on a balanced program should continue, the Joint Committee on Health Problems in Education said at a recent meeting.

The Joint Committee is made up of representatives of the National Education Association and the American Medical Association.

A course in health education should be a certification requirement for all prospective teachers and school administrators, a Committee resolution said. The resolution asked that school districts offer health education training programs for teachers and administrators now in service.

There is a continuing need for a venereal disease education program in the schools, the Committee said. The group commended the VD-eradication efforts of the AMA and the U.S. Public Health Service. It also called for continuing education to stress the dangers of misusing, and the advantages of correctly using, drugs.

A resolution concerned with the development of programs to implement educational legislation involving a health component suggested that physicians, dentists, nurses, and allied health personnel be included in the planning as well as the execution of such programs. Both local medical societies and local educational authorities are urged to assume responsibility for seeking cooperative efforts.

School administrators who "through ingenuity and innovation" provide appropriate time in the school schedule for health education were commended by the Committee. A resolution also called for further experimentation in curriculum development for health education.

Look how many ways

Thorazine®

brand of

chlorpromazine

can help

	Tranquilizer	**Potentiator**	**Antiemetic**
Agitation	●		
Alcoholism	●		●
Anxiety	●		
Cancer patients	●	●	●
Severe neurodermatitis	●		
Drug addiction withdrawal symptoms	●		●
Emotional disturbances (moderate to severe)	●		
Nausea & vomiting	●		●
Neurological disorders	●		
Obstetrics	●	●	●
Pain	●	●	●
Pediatrics	●	●	●
Porphyria	●	●	
Psychiatric disorders	●		
Hiccups—refractory	●		
Senile agitation	●		
Surgery	●	●	●
Tetanus	●	●	

'Thorazine' is useful as a specific adjuvant in the above named conditions.

The following is a brief precautionary statement. Before prescribing, the physician should be familiar with the complete prescribing information in SK&F literature or *PDR*. *Contraindications:* Comatose states or the presence of large amounts of C.N.S. depressants. *Precautions:* Potentiation of C.N.S. depressants may occur (reduce dosage of C.N.S. depressants when used concomitantly). Antiemetic effect may mask other conditions. Possibility of drowsiness should be borne in mind for patients who drive cars, etc. In pregnancy, use only when necessary to the welfare of the patient. *Side Effects:* Occasionally transitory drowsiness; dry mouth; nasal congestion; constipation; amenorrhea; mild fever; hypotensive effects, sometimes severe with I.M. administration; epinephrine effects may be reversed; dermatological reactions; parkinsonism-like symptoms on high dosage (in rare instances, may persist); weight gain; miosis; lactation and moderate breast engorgement (in females on high dosages); and less frequently cholestatic jaundice. Side effects occurring rarely include: mydriasis; agranulocytosis; skin pigmentation, lenticular and corneal deposits (after prolonged substantial dosages).

For a comprehensive presentation of 'Thorazine' prescribing information and side effects reported with phenothiazine derivatives, please refer to SK&F literature or *PDR.*

Smith Kline & French Laboratories SK&F

Health Quackery Expose' Is Now Available

"Facts on Quacks," a complete source book touching every aspect of health quackery, is now off the press, the American Medical Association has announced.

Having been in development for 18 months, the new publication is considered a major instrument in the AMA's program of educating the public to the evils of quackery. It is especially designed for medical society use in their anti-quackery programing.

Publication of "Facts on Quacks" marks the first time in history that information on all types of quackery has been consolidated into one book.

The 32-page, two-color, illustrated booklet, written in question and answer form, is beamed at the public and covers facts on health quackery in such fields as arthritis and rheumatism, cancer, nutrition and weight reduction, over-the-counter drugs, alcoholic nostrums, health books and pamphlets, epilepsy, mental health and retardation, cosmetics, baldness, rejuvenation and sex stimulants. One section advises the reader what he should do if he suspects health quackery activities in his community.

"Never before has there been any book like this," Doyl Taylor, director of the AMA Depart-

(Continued on Page 52)

Easy on the Budget...
Easy on the Mother

Tablets & Elixir
For Iron Deficiency Anemia

FAMOUS
Fergon®
brand of FERROUS GLUCONATE

C.M.A.P. CODE # 5261

BREON LABORATORIES INC.
Subsidiary of Sterling Drug Inc.
90 Park Avenue, New York, N.Y. 10016

Tandearil®
oxyphenbutazone

Therapeutic Effects: Tandearil is a nonhormonal compound which may rapidly resolve inflammation and help restore normal joint function. Its action does not affect pituitary-adrenal function or impair immune responses. Its value in osteoarthritis is especially noteworthy because this disorder responds inconsistently to steroids and is often resistant to salicylates. Further, indomethacin is limited only to osteoarthritis of the hip, whereas oxyphenbutazone is effective in all forms of the disease.

Contraindications: Edema; danger of cardiac decompensation; history or symptoms of peptic ulcer; renal, hepatic or cardiac damage; history of drug allergy; history of blood dyscrasia. The drug should not be given when the patient is senile or when other potent drugs are given concurrently.

Warning: If coumarin-type anticoagulants are given simultaneously, watch for excessive increase in prothrombin time. Pyrazole compounds may potentiate the pharmacologic action of sulfonylurea, sulfonamide-type agents and insulin. Carefully observe patients receiving such therapy. Use with great caution in the first trimester of pregnancy.

Precautions: Obtain a detailed history and a complete physical and laboratory examination, including a blood count. The patient should be closely supervised and should be warned to report immediately fever, sore throat, or mouth lesions (symptoms of blood dyscrasia); sudden weight gain (water retention); skin reactions; black or tarry stools or other evidence of intestinal hemorrhage. Make regular blood counts. Discontinue the drug and institute countermeasures if the white count changes significantly, granulocytes decrease, or immature forms appear. Use greater care in the elderly and in hypertensives.

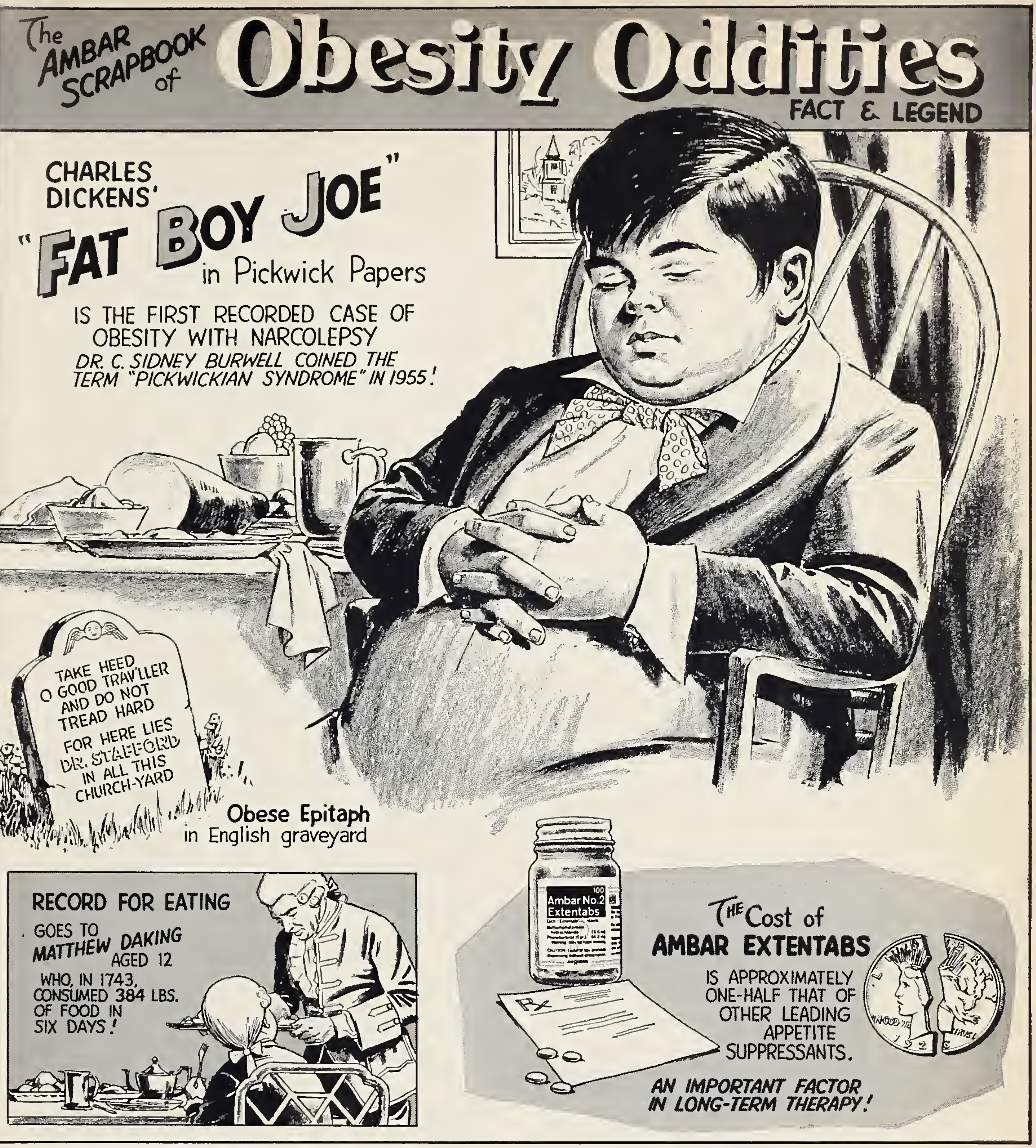
Adverse Reactions: The most common are nausea, edema and drug rash. The drug has been associated with peptic ulcer and may reactivate a latent peptic ulcer. Infrequently, agranulocytosis, or a generalized allergic reaction may occur and require withdrawal of medication. Stomatitis, salivary gland enlargement, vomiting, vertigo and languor may occur. Leukemia and leukemoid reactions have been reported but cannot definitely be attributed to the drug. Thrombocytopenic purpura and aplastic anemia may occur. Confusional states, agitation, headache, blurred vision, optic neuritis and transient hearing loss have been reported, as have hyperglycemia, hepatitis, jaundice, and several cases of anuria and hematuria. With long-term use, reversible thyroid hyperplasia may occur infrequently. Moderate lowering of the red cell count due to hemodilution may occur.

Dosage in Osteoarthritis: The initial daily dosage in adults is 300-600 mg. in divided daily doses. When improvement occurs, dosage should be decreased to the minimum effective level; this should not exceed 400 mg. daily, and is often achieved with only 100-200 mg. daily.

For complete details, please refer to full prescribing information. 6562-VI(B)R

Availability: Tablets of 100 mg.

Geigy Pharmaceuticals
Division of Geigy Chemical Corporation
Ardsley, New York


The AMBAR SCRAPBOOK of
Obesity Oddities
FACT & LEGEND
CHARLES DICKENS' "FAT BOY JOE" in Pickwick Papers
IS THE FIRST RECORDED CASE OF OBESITY WITH NARCOLEPSY
DR. C. SIDNEY BURWELL COINED THE TERM "PICKWICKIAN SYNDROME" IN 1955!
TAKE HEED O GOOD TRAV'LLER AND DO NOT TREAD HARD FOR HERE LIES DR. STAFFORD IN ALL THIS CHURCH-YARD
Obese Epitaph in English graveyard
RECORD FOR EATING GOES TO MATTHEW DAKING AGED 12 WHO, IN 1743, CONSUMED 384 LBS. OF FOOD IN SIX DAYS!
100 Ambar No. 2 Extentabs
THE Cost of AMBAR EXTENTABS IS APPROXIMATELY ONE-HALF THAT OF OTHER LEADING APPETITE SUPPRESSANTS.
AN IMPORTANT FACTOR IN LONG-TERM THERAPY!

CONTROL FOOD AND MOOD ALL DAY LONG WITH A SINGLE MORNING DOSE

AMBAR #2 EXTENTABS®

methamphetamine HCl 15 mg.,
phenobarbital 64.8 mg. (1 gr.)
(Warning: may be habit forming).

One Ambar Extentab before breakfast can help control most patients' appetite for up to 12 hours. Methamphetamine, the appetite suppressant, gently elevates mood and helps overcome dieting frustrations. Phenobarbital, the sedative in Ambar, controls irritability and anxiety... helps maintain a state of mental calm and equanimity. Both work together to ease the tensions that erode the willpower during periods of dieting.

Also available: Ambar #1 Extentabs®—methamphetamine hydrochloride 10 mg., phenobarbital 64.8 mg. (1 gr.) (Warning: may be habit forming).

BRIEF SUMMARY/**Indications:** Ambar suppresses appetite and helps offset emotional reactions to dieting. **Contraindications:** Hypersensitivity to barbiturates or sympathomimetics; patients with advanced renal or hepatic disease. **Precautions:** Administer with caution in the presence of cardiovascular disease or hypertension. **Side Effects:** Nervousness or excitement occasionally noted, but usually infrequent at recommended dosages. Slight drowsiness has been reported rarely. See package insert for further details.

A. H. ROBINS COMPANY,
RICHMOND, VA. 23220

A-H-ROBINS

Sensitive to need;

responsive to expression.

In the Devereux residential treatment plan the sensitive, responsive staff member is a vital component.

For more than half a century recognition of the value of the human resource has been the hallmark of Devereux Schools' treatment programs for mentally retarded and emotionally disturbed children and young adults.

DEVEREUX SCHOOLS
UNDER THE DEVEREUX FOUNDATION
A NONPROFIT ORGANIZATION

Helena T. Devereux
Founder and Consultant

Edward L. French, Ph.D.
President and Director

FOR INFORMATION AND LITERATURE
Santa Barbara (Box 1079), California
Keith A. Seaton, Director of Admissions

Devon, Pennsylvania and Rutland, Massachusetts
Charles J. Fowler, Director of Admissions
Devereux Schools, Devon, Penna. 19333

Victoria (Box 2666), Texas
Richard D. Grant, Registrar

Health Quackery Expose' Is Now Available

(Continued from Page 48)

ment of Investigation, says. "It should be placed in every reference library in the country and certainly in every medical library. Every local medical society should see to it that a copy is in each of their local libraries and individual physicians should place copies in their waiting rooms."

"Facts on Quacks" is being published by the AMA in full cooperation with various governmental and voluntary agencies in the health field such as the National Health Council, the Food and Drug Administration, the National Better Business Bureau, the Post Office Department, the Federal Trade Commission, the American Cancer Society, Inc. and The Arthritis Foundation.

Requests for "Facts on Quacks" should be directed to the Order Department. Single copies, 30c; 50 to 99, 28c; 100 to 499, 25c; 500 to 999, 23c; 1,000 and over, 20c.

Rural Areas Obtain Physicians

Twenty communities obtained physicians in 1966 with the help of the American Medical Association-Sears Roebuck Foundation Community Medical Assistance Plan, reports *The AMA News.*

This was the seventh year in which 20 or more communities succeeded in finding physicians.

Communities which obtained a physician include Walton, English, and Holland, Ind.; Jamestown, Blandon, and Linesville, Pa.; Camden, Cazenovia, and Bridgeport, N.Y.; Nickelsville and Coeburn, Va.; Flagler and Oak Creek, Colo.; Wisner, Neb.; Wellfleet, Mass.; Eveleth, Minn.; Claude and Sanderson, Texas; and Waterville, Kan.

Many of the communities assisted by the AMA-Sears Plan in 1966 were larger than those aided in previous years, the Foundation noted. It is quite likely, it added, that the program might be assisting communities of ten thousand within the next few years. Two cities aided in 1966 exceed five thousand population.

Other communities are seeking physicians under the program, and have raised funds and are constructing two-doctor medical centers. These include: Hubbard, Bedford, Melcher-Dallas, Coon Rapids, and Marcus, Iowa; Kirkland, Sheridan, Gardner, and Macon, Ill.; Cohocton, Greensville, and North Rose N.Y.; Springfield, Colo.; Locust and Newport-by-the-Sea, N.C.; Crawford and Rochelle, Ga.; St. Joseph and Dubach, La.; Rector and Parkin, Ark.; Huntley-Worden, Mont.; Granite Falls, Wash.; Glen Ullin, N.D.; Ansonia, Ohio; Clay Center, Neb.; Florence, Wis.; Nuremburg, Pa.; Lamberton, Minn.; Roachdale, Ind., and West Union, W. Va.

The following communities are seeking additional physicians: Loyal, Wis.; Bayboro, N.C.; Illmo, Mo.; Sandstone, Minn.; Tupper Lake, N.Y.; Hardin, Ill., and Hawarden, Iowa.

BOOKS RECEIVED

Books received by CALIFORNIA MEDICINE *are acknowledged in this column. Selections will be made for more extensive review in the interest of readers as space permits.*

APPROACH TO DIAGNOSIS IN MODERN NEUROLOGY—A Mount Sinai Hospital Monograph—Edited by Morris B. Bender, M.D., Assistant Editor, Martin Feldman, M.D. With contributions by various doctors. Grune & Stratton, Inc., 381 Park Avenue South, New York, N.Y. 10016, 1967. 186 pages, $6.50.

THE BETRAYAL OF THE BODY—By Alexander Lowen, M.D., The Macmillan Company, New York, 1967. 307 pages, $6.95.

CANCER CHEMOTHERAPY—Basic and Clinical Applications—The Fifteenth Hahnemann Symposium—Editors, Isadore Brodsky, M.D., Associate Professor of Medicine and Head, Hematology Section, and S. Benham Kahn, M.D., Assistant Professor of Medicine, Hematology Section, Hahnemann Medical College and Hospital, Philadelphia, Pennsylvania. Consulting Editor, John H. Moyer, III, M.D., Professor and Chairman, Department of Medicine, Hahnemann Medical College and Hospital, Philadelphia, Pennsylvania. Grune & Stratton, Ind., New York and London, 1967. 348 pages, $18.50.

THE CLOAK OF COMPETENCE—Stigma in the Lives of the Mentally Retarded—By Robert B. Edgerton. University of California Press, Berkeley, California 94720, 1967. 233 pages, $5.75.

CURRENT THERAPY 1967—Latest Approved Methods of Treatment for the Practicing Physician—Edited by Howard F. Conn, M.D. W. B. Saunders Company, Philadelphia and London, 1967. 844 pages, $13.00.

DIAGNOSIS AND TREATMENT OF SHOCK—By Max Harry Weil, A.B. M.D., Ph.D., F.A.C.P., Associate Professor of Medicine, University of Southern California School of Medicine and Director, Shock Research Unit, Senior Attending Cardiologist and Attending Physician, Los Angeles County General Hospital; and Herbert Shubin, A.B., M.D., F.A.C.P., Assistant Professor of Medicine, University of Southern California School of Medicine Co-principal Investigator of the Shock Research Unit; Attending Physician, Los Angeles County General Hospital. The Williams & Wilkins Company, Baltimore, 1967. 391 pages, $12.00.

DIFFERENTIAL DIAGNOSIS OF INTERNAL DISEASES—Clinical Analysis and Synthesis of Symptoms and Signs on Pathophysiological Basis—Third Revised and Enlarged Edition—By Julius Bauer, M.D., F.A.C.P., Professor of Medicine, Emeritus, Loma Linda University, Los Angeles; Lecturer in Medicine, University of Southern California; Consultant in Medicine, Los Angeles County General Hospital; former Consultant in Medicine, Long Beach Veterans Administration Hospital. Grune & Stratton, Inc., New York and London, 1967. 1,071 pages, $29.50.

DIZZINESS AND VERTIGO—Diagnosis and Treatment—By Martin Spector, M.D., Associate Professor of Otorhinology, Temple University School of Medicine and Hospital; Chief of Otorhinology, Kensington Hospital; Consultant in Otology, St. Mary's Hospital, Philadelphia, Pennsylvania. Grune & Stratton, Inc., New York and London, 1967. 299 pages, $17.75.

A DOCTOR DISCUSSES THE PRESCHOOL CHILD'S LEARNING PROCESS (and how parents can help)—By Edward B. Rosenberg, B.S., and Silas L. Warner, M.D. Budlong Press Company, 5428 N. Virginia Avenue, Chicago, Ill., 60625, 1967. 102 pages, $1.50. (Paperback, available only through professional sources.)

LABOR—Clinical Evaluation and Management—By Emanuel A. Friedman, M.D., Med. Sc.D., Chairman and Professor of Obstetrics and Gynecology, Chicago Medical School; Michael Reese Hospital and Medical Center. Foreword by Nicholson J. Eastman, M.D., LL.D., D.Sc. (hon.), Professor Emeritus of Obstetrics, The Johns Hopkins University School of Medicine. 397 pages, $13.50.

PATHOLOGY ANNUAL, VOLUME 2, 1967—Series Editor, Sheldon C. Sommers, M.D., Professor of Pathology, Columbia University College of Physicians and Surgeons; Associate Director of Laboratories, Francis Delafield Hospital, New York, New York; Clinical Professor of Pathology, University of Southern California School of Medicine, Los Angeles, California. Appleton-Century-Crofts (Division of Meredith Publishing Company), 440 Park Ave. So., New York, N.Y. 10016, 1967. 427 pages, $13.75.

PUBLIC EDUCATION ABOUT CANCER — Research Findings and Theoretical Concepts—Prepared by the Committee on Public Education of the Commission on Cancer Control. U.I.C.C. Monograph Series—Volume 5. Springer-Verlag, Berlin, Heidelberg, New York, 175-5th Avenue, New York, N.Y. 10010, 1967. 103 pages, $9.50.

SELECTED QUESTIONS AND ANSWERS, 1966, J.A.M.A.—Selected from 1966 issues of The Journal of the American Medical Association. American Medical Association, 1967. 360 pages, $2.00 each in U.S., U.S. Possessions, Mexico, and Canada; $1.00 each for Medical Students, Hospital Interns and Residents in U.S., U.S. Possessions, Mexico, and Canada; $2.50 each in all other countries.

SEXUAL PROBLEMS—Diagnosis and Treatment in Medical Practice—Edited by Charles William Wahl, M.D., UCLA Medical Center, Southern California Psychoanalytic Institute. The Free Press, New York (A Division of the Macmillan Company), 1967. 300 pages, $7.95.

THE SKIN—A Clinopathological Treatise—Second Edition—By Arthur C. Allen, M.D., Director of Laboratories, the Jewish Hospital of Brooklyn, New York, Downstate Medical Center. Grune & Stratton, Inc., 381 Park Avenue South, New York, New York 10016, 1967. 1,182 pages, 2,362 illustrations, 503 full-page plates, $48.50.

SURGERY OF RHEUMATOID ARTHRITIS—By Leonard Marmor, M.D., F.A.C.S., Associate Professor of Surgery/Orthopedics, Department of Surgery/Orthopedics, U.C.L.A., School of Medicine, Los Angeles, California. 274 illustrations. Lea & Febiger, Washington Square, Philadelphia, Pennsylvania 19106, 1967. 261 pages, $15.00.

WARD PROCEDURES AND TECHNIQUES—By Philip Cooper, M.D., Chief of Surgical Services, Veterans Administration Hospital, Bronx, New York, and Professor of Clinical Surgery, Albert Einstein College of Medicine, Bronx, New York. Illustrations by Ingram Chodorow. Appleton-Century-Crofts, Division of Meredith Publishing Company, 440 Park Ave. So., New York, N.Y. 10016, 1967. 303 pages, $6.75 (Paperback).

Your public relations problem has been our prime consideration in collection procedures during two generations of ethical service to the Medical Profession.

THE DOCTORS BUSINESS BUREAU
Since 1916
821 MARKET ST., SAN FRANCISCO 94103
GArfield 1-0460

STACEY'S for any Medical Book in Print
Retail Stores in Palo Alto and San Francisco

NEW BOOK

THE PHYSIOLOGICAL BASIS OF MEDICAL PRACTICE: A Text in Applied Physiology. By Charles Herbert Best. 8th ed. 1,793 pages. 1966. Williams & Wilkins. $17.

Stacey's
326-9955
2575 HANOVER STREET
PALO ALTO, CALIFORNIA 94304

Please send me on approval:________

Name________
Street________
City________ State________

Beatniks: Up, Down and Off

Not all beatniks are bad, says a psychiatrist in a current American Medical Association publication.

In fact, beatnikism may be just another way for a teenager to find groups with similar, sympathetic interests on his way to adulthood.

Writing in the March issue of the *Archives of General Psychiatry,* published by the AMA, Jules Masserman, M.D., classifies beatniks into three groups:

Up-Beatniks—The best of the lot, says Dr. Masserman, who is professor of psychiatry at Northwestern University, Evanston, Ill. Even with their occasional beards, tresses, play-readings, placards, proclamations, and protest marches, they are "Basically earnest, energetic, intelligent, and well intentioned."

They are a help "in prodding us oldsters to review our smug hypocrisies and revise our medieval customs and conduct," Dr. Masserman said.

Down-Beatniks—Articulate in condemning the inequalities and injustices of society, often with some courage and justification. They are strident and obnoxious in speech and manner, but the outlook for their improvement is often favorable.

"With further maturity and increasing wisdom, most of them become good citizens, competent parents and sometimes even staunch Republicans," said Dr. Masserman.

The Off-Beatniks—These are the more seriously erratic, troubled, and troublesome misfits. Despite their pretensions, they contribute little that is truly constructive or original to our culture.

What makes a beatnik? The teenager is looking for "identity" in a world of great complexity, Dr. Masserman explained.

"The late adolescent may spread himself thin trying to be simultaneously a dutiful child, a brilliant scholar, a winning athlete, a popular leader, a potent lover, a seductive nymph, a cynical sophisticate and a dozen other incompatible alter egos."

Most youngsters seek status in groups offering support, with certain patterns of dress, modes of speech, and stereotyped musical tastes.

Dr. Masserman said most such cliques are harmless and may even be beneficial as interim experiences. He cautions:

"The dividing line is this: When an adolescent becomes so immersed in extracurricular activities as to neglect his education, physical health and broader social development, or when he advocates 'sports' that endanger others, such as drag racing, or when he promotes public obscenity, sexual arrogance and physical violence, or experiments with excessive amounts of alcohol or drugs, then therapy is necessary."

Vacation trip....

Motion sickness?

This time it'll be different. Emetrol taken before the trip begins will usually prevent nausea and vomiting. Emetrol is effective and safe...most helpful where safety is most important. It acts locally—not systemically.

RORER

WILLIAM H. RORER, INC.
Fort Washington, Pa.

Emetrol®
phosphorated carbohydrate solution
emesis control

INDEX

California Medicine

Volume 106
January-June 1967

AUTHOR INDEX

PAGE

A

Adachi, Roy T., *Orange* 9
Aggeler, Paul M., *San Francisco* 255
Alexander, Marie M., *Boston* 437
Amar, Arjan D., *Walnut Creek* 120
Ambrus, Laszlo, *Martinez* 354

B

Bark, C. Jack, *Los Angeles* 199
Barrow, J. Gordon, *Atlanta, Georgia* 35
Beall, Gildon N., *Los Angeles* 296
Berk, J. Edward, *Los Angeles* 401
Berke, Meyer, *Lakewood* 468
Bilodeau, Larry, *Los Angeles* 112
Blazina, Martin E., *Los Angeles* 450
Bloomquist, Edward R., *Glendale* 346
Bodner, Henry, *Los Angeles* 242
Bradley, Edward C., *Los Angeles* 7, 9, 10
Braslow, Lawrence, *Arlington* 127
Brechner, Verne L., *Los Angeles* 290
Breslow, Lester, *Berkeley* 176
Brown, G. Stephen, *Los Angeles* 201
Buschke, Franz, *San Francisco* 12, 282

C

Camarata, Samuel J., *Orange* 7
Caponegro, Peter, *Los Angeles* 165
Casad, Donald E., *Fresno* 20
Casdorph, H. R., *Long Beach* 293
Cooper, Robert A., *Portland, Oregon* 170
Cutler, Sidney J., *Bethesda, Maryland* 98

D

Dawson, Peter J., *Portland, Oregon* 170
Day, Robert W., *Berkeley* 382
Dillon, John B., *Los Angeles* 235, 426, 444
Donnell, George N., *Los Angeles* 58
Dooley, James H., *Paso Robles* 247
Doty, Donald B., *Los Angeles* 8

E

Earll, Jerry M., *San Francisco* 56
Enelow, Allen J., *Los Angeles* 179

KEY TO ABBREVIATIONS USED

(BRP)—Bureau of Research and Planning; (CMA)—California Medical Association; (CR)—Case Report; (Ed.)—Editorial; (I)—Information; (LE)—Letter to Editor; (MSC)—Medical Staff Conference; N/N—News and Notes; (Or.)—Original Article; (PE)—Page End; (RSB)—Report of Scientific Board.

PAGE

F

Feeney, M. J., *San Diego* 372
Fischer, Ames, *San Francisco* 189
Fisher, Duke D., *Los Angeles* 49, 210
Fonkalsrud, Eric W., *Los Angeles* 451
Friedman, Richard A., *Los Angeles* 197

G

Galen, Donald I., *Los Angeles* 447
Garell, Dale C., *Los Angeles* 368
Gilbert, Arnold, *Berkeley* 382
Grodsky, Lewis, *San Francisco* 44
Gwinup, Grant, *Los Angeles* 159

H

Hanafee, William, *Los Angeles* 112
Hanashiro, Paul K., *Los Angeles* 7
Hansen, James R., *Burlingame* 462
Hassard, Howard, *San Francisco* 80
Heggli, William F., *San Francisco* 211
Herrmann, Christian Jr., *Los Angeles* 275
Heuser, Eva, *Los Angeles* 58
Horton, Richard, *Los Angeles* 87
Howe, G. E., *San Diego* 372
Huse, Wilfred M., *Loma Linda* 203

J

Jack, George, *San Francisco* 12

K

Kagawa, Yuzuru, *Los Angeles* 165
Kanter, Marvin, *Los Angeles* 198
Kawashima, Yasunaru, *Los Angeles* 165
Kay, Jerome Harold, *Los Angeles* 165
Kern, William H., *Los Angeles* 447
King, Charles G., *Los Angeles* 401
Kolb, Felix O., *San Francisco* 56

L

Landing, Benjamin H., *Los Angeles* 58
Lee, John C., *San Francisco* 465
Levan, Norman E., *Los Angeles* 20
Lewis, Arline, *Berkeley* 382
Lewis, Martin I., *Los Angeles* 247
Lieberman, Ellin, *Los Angeles* 58
Lillington, Glen A., *Palo Alto* 92
Low, Edmund M. Y., *Los Angeles* 364

M

MacLaggan, James C., *San Diego* 149, 433

MacLean, Lloyd D., *Montreal, Canada* 3
Manning, Phil R., *Los Angeles* 149
Marbach, Edward P., *Los Angeles* 6
Melmon, Kenneth L., *San Francisco* 388
Mendez, Adolfo, *Los Angeles* 165
Miller, John N., Jr., *Sacramento* 17
Mills, Don Harper, *Los Angeles* 330
Moncrief, John A., *Fort Sam Houston, Texas* 4
Mullenix, R. B., *San Diego* 372

N

Nies, Alan S., *San Francisco* 388

O

Odegaard, Charles E., *Seattle* 337

P

Phillips, Theodore L., *San Francisco* 282
Posnikoff, Jack, *Orange* 124
Prentiss, R. J., *San Diego* 372

Q

Quinn, William F., *Los Angeles* 108

R

Rambo, Oscar N., *San Francisco* 170
Redington, John V., *Los Angeles* 165
Reisner, Ronald M., *Torrance* 28
Riley, John, *Los Angeles* 359
Rockwell, Marshall A., Jr., *Los Angeles* 11
Rubin, David, *Los Angeles* 39
Russell, Findlay E., *Los Angeles* 248

S

Searcy, Ronald L., *Nutley, N.J.* 364
Seeley, Sam F., *Washington, D.C.* 1
Shaw, Walter M., *Chula Vista* 212
Shearn, Martin A., *Oakland* 465
Sherins, Richard J., *Los Angeles* 87
Shevick, Murray, *Arlington* 127
Shubin, Herbert, *Los Angeles* 9
Sorensen, Royal H., *Fresno* 20
Spink, Wesley, W., *Minneapolis, Minnesota* 2
Stare, Fredrick J., *Boston* 437
Steedman, Robert, *Los Angeles* 401
Stevens, G. Melvin, *Palo Alto* 92

T

Thal, Sam H., *Martinez* 354
Trivedi, Sarah, *Los Angeles* 196
Tromovitch, Theodore A., *San Francisco* 211, 400
Tsuji, Harold K., *Los Angeles* 165
Tu, W. H., *Oakland* 465

U

Ungerleider, J. Thomas, *Los Angeles* 49, 210

V

Visher, John S., *San Francisco* 117

W

Waldmann, William J., *Fresno* 20
Waldron, William G., *Los Angeles* 248
Walker, Phyllis C., *Long Beach* 468
Walske, Benedict R., *Tucson, Arizona* 212
Wareham, Ellsworth E., *Loma Linda* 203
Warnecke, Juanita, *Martinez* 354
Watts, Malcolm S. M., *San Francisco* 272
Wearn, Joseph T., 375
Weidner, William, *Torrance* 359
Weigen, John F., *Palo Alto* 92
Weil, Max Harry, *Los Angeles* 4, 7, 8, 10
Weinreb, Marvin S., *Hayward* 183
Weinstein, Selig B., *Martinez* 354
Whigham, Howard, *Los Angeles* 10
Wiater, Edward J., *Long Beach* 499
Williams, Hibbard E., *San Francisco* 306
Wilson, Gabriel, *Los Angeles* 359

Y

Yamada, Takashi, *Los Angeles* 165

Z

Zahn, Richard L., *Orange* 6
Zweig, Robert, *Arlington* 127

SUBJECT INDEX

A

Abortion, Therapeutic, Where We Stand on the Question of (CMA) 318
  Letter to Editor, Edward J. Wiater 499
Accomplishments and Frustrations—A Report of Two Years in the Life of the California Medical Association (President's Address), James C. MacLaggan (Or.) 433
Acne, see *Systemic Agents*
Adolescent, see *Obese Person*
Adrenocortical System, see *Hypothalamic-Pituitary*
Alcoholic Content of Blood, Breath or Urine, Statutory Consent for Chemical Test of, in Drunk Driving Cases, Howard Hassard (I) 80
AMA Judicial Council, Ethical Responsibilities in Prescribing Drugs and Devices (I) 505
Amebic Granuloma of the Cecum, Walter M. Shaw and Benedict R. Walske (CR) 212
Anaphylaxis and Endotoxin (Part of Symposium), Wesley W. Spink 2
(Anderson, Carl E.) The Chairman Retires (Ed.) 411
Aneurysm, Intracranial, see *Cure of*
Annual Meeting, Successful (Ed.) 410
Anorectal Operations, Minor, see *Unsuspected Carcinoma*
Anticoagulant Drugs, Coumarin, see *Blood Coagulation*
Antihypertensive Drugs, see *Recent Concepts in the Clinical Pharmacology of*
Aortic and Mitral Valve Replacement (MSC) 473
Aortography in the Diagnosis of Cancer, William Weidner, Gabriel Wilson and John Riley (Or.) 359
Application of Computer Techniques to Treatment of the Critically Ill Patient (Part of Symposium), Marshall A. Rockwell, Jr. 11
Audio-Digest's Benign Cycle (Ed.) 136
Autoimmune Progesterone Urticaria, Theodore A. Tromovitch and William F. Heggli (CR) 211

B

Bile Acid Sequestering Resin, A, see *Hypercholesteremia*
Biopsy, Lymph Node, see *Dermatopathic Lymphadenopathy*
Biopsy of Localized Pulmonary Lesions, see *Needle Aspiration*
Bladder, Carcinoma of the, see *Role of External Irradiation*
Bladder Tumor Diagnosis—Improved Excretory Cystograms, Arjan D. Amar (Or.) 120
Blastomycosis in California, see *North American*

Blood, Alcoholic Content of, see *Statutory Consent for Chemical Test*

Blood, Central Venous, see *Symposium on Circulatory Shock*

Blood Coagulation and the Coumarin Anticoagulant Drugs, Paul M. Aggeler (Or.) 255

Blood Transfusion, see *Cure of Intracranial Aneurysm*

Boerhaave Conference, see *Role of University in Modern Society*

Breath, Alcoholic Content of, see *Statutory Consent for Chemical Test*

Budgeting Process, The (CMA) 73

Bureau of Research and Planning:
- Federalized Health Care Systems in Western Europe and Australia 243
- Professional Registered Nurses in the United States and California, 1962 327
- Poverty in the U.S. as Defined by Federal Standards 501

C

Cancer, Diagnosis of, see *Aortography*

Cancer Patients, Poikilocytosis in, Donald I. Galen and William H. Kern (Or.) 447

Carcinoma, see *Total Perineal Prostatectomy*

Carcinoma, Cell, of the Bladder, see *Role of External Irradiation*

Carcinoma, Unsuspected, Found Histologically After Minor Anorectal Operations, Lewis Grodsky (Or.) 44

Cardiac Arrest, see *Symposium on Circulatory Shock*

Cardiac Valves, Prosthetic, see *Current Status of*

Cardiovascular Disease Programs, Community, How (They) Can Aid the Private Physician, J. Gordon Barrow (Or.) 35

Cecum, Amebic Granuloma of the, Walter M. Shaw and Benedict R. Walske (CR) 212

Central Venous Blood for Monitoring pH and PCO_2 in the Critically Ill Patient (Part of Symposium), Richard L. Zahn 6

Chairman Retires (Carl E. Anderson) (Ed.) 411

Changing Role of County Hospitals in California, The, Lester Breslow (Or.) 176

Chemical Test of Alcoholic Content of Blood, Breath or Urine in Drunk Driving Cases, Statutory Consent for, Howard Hassard (I) 80

Cherish the Capacity to Change, Joseph T. Wearn (Or.) 375

Chloroquine Retinopathy, see *Electro-Oculograms*

Cholestyramine, see *Hypercholesteremia*

Chronic Obstructive Pulmonary Emphysema—Is Exercise Beneficial? Laszlo Ambrus, Sam H. Thal, Selig B. Weinstein and Juanita Warnecke (Or.) 354

Circulatory Shock—A Symposium on Advances in the Understanding of Mechanisms and Treatment, see *Symposium on Circulatory Shock*

Clinical Use of a New Mitral Disc Valve, Jerome Harold Kay, Harold K. Tsuji, John V. Redington, Yasunaru Kawashima, Yuzuru Kagawa, Takashi Yamada, Peter Caponegro and Adolfo Mendez (Or.) 165

Clinicopathologic Conference, Residents', see *Pineal Germinoma*

CMA Goals, see *Statement of* (CMA)

Committee on the Role of Medicine in Society, see *Responsibility for Quality in Health Care*

Community Psychiatry and the Population Explosion, Ames Fischer (Or.) 189

Comparison of the Capillary and Central Hematocrit as an Indication of Circulatory Shock (Part of Symposium), Donald B. Doty and M. H. Weil.. 8

Compensable Injury, The, Allen J. Enelow (Or.) 179

Continuing Medical Education (CMA):
- Toward Improving Postgraduate Medical Education. Phil R. Manning and James C. MacLaggan 149
- Mechanism to Assure Continuing Medical Education, A, John B. Dillon 235
- Postgraduate Medical Education, John B. Dillon 426

Controls on Outlays for Medi-Cal Care (CMA) 493

Corticosteroids, see *Symposium on Circulatory Shock*

Coumarin Anticoagulant Drugs, see *Blood Coagulation*

County Hospitals in California, see *Changing Role*

Cure of Intracranial Aneurysm Without Use of Blood Transfusion, Jack Posnikoff (CR) 124

Current Status of Prosthetic Cardiac Valves, Wilfred M. Huse and Ellsworth E. Wareham (Or.) 203

Cystosarcoma Phylloides, Recurrent, James R. Hansen (CR) 451

D

Dentist-Physician Relationship in Extended Care Facilities (I) 243

Dermatopathic Lymphadenopathy—A Clinicopathologic Analysis of Lymph Node Biopsy Over a Fifteen-Year Period, Robert A. Cooper, Peter J. Dawson and Oscar N. Rambo (Or.) 170

Diabetes, Hyperosmolar Non-Ketotic (MSC) 131

Disorders of Esophageal Motility (MSC) 405

Drug Problems, see *Narcotic and Dangerous*

Drugs and Devices, Prescribing, see *Ethical Responsibilities in*

Drunk Driving, see *Statutory Consent for Chemical Test of Alcoholic Content of Blood*

Duodenal Ulcer with Massive Hemorrhage Complicating Pregnancy, Charles G. King, Robert Steedman and J. Edward Berk (CR) 401

E

Effect of Corticosteroids on the Progression of Hemorrhagic Shock in Rats (Part of Symposium), Howard Whigham 10

Electro-Oculograms in the Early Diagnosis of Chloroquine Retinopathy, Marvin S. Weinreb (Or.) 183

Emphysema, Pulmonary, see *Chronic Obstructive*

Endotoxin and Anaphylaxis, see *Symposium on Circulatory Shock*

Ethical Responsibilities in Prescribing Drugs and Devices, AMA Judicial Council (I) 505

Excretory Cystograms, Improved, see *Bladder Tumor Diagnosis*

Exercise Program for Shoulder Disability, An, David Rubin (Or.) 39

F

Facial Paralysis Occurring with Hypothyroidism—A, Report of Two Cases, Jerry M. Earll and Felix O. Kolb (CR) 56

Family Planning Clinic in a County Hospital, A, John N. Miller, Jr. (Or.) 17

Federalized Health Care Systems in Western Europe and Australia (BRP) 243

For Realism in Medical Legislation (Ed.) 136

G

Gastrointestinal Malformations, see *Recent Developments*

Generalized Mastocytosis—Fatal Case with Autopsy Findings, Meyer Berke and Phyllis C. Walker (CR) 468

Glomerulonephritis, Acute, see *Insidious Splenic Rupture*

Glomerulonephritis, Membranous, Rapidly Fatal Course, W. H. Tu, Martin A. Shearn and John C. Lee (CR) 465

Glucose Intolerance, see *Occult*

(Gold-Headed Cane) Cherish the Capacity to Change, Joseph T. Wearn (Or.) 375

Grand Mal Seizures Following Ingestion of LSD^{25}, Duke D. Fisher and J. Thomas Ungerleider (CR) 210

(Grobstein, Clifford) New Dean at UC-San Diego (N/N) 506

Guide Lines for the Selection of Fluids and Drugs for the Treatment of Patients in Shock (Part of Symposium) Lloyd D. MacLean 3

H

Head Start, see *Project*

Health Care of the People (Ed.) 222

Health Care, Quality of (Ed.) 484

Health Care Systems, see *Federalized*

Health Manpower Council (Ed.) 317

Hematologic Manifestations of Tuberculosis (MSC) 215

Hemodynamic Studies on Clinical Shock Following Pulmonary Embolism (Part of Symposium), Roy T. Adachi and E. C. Bradley 9

Hemorrhage, Massive, see *Duodenal Ulcer*

Hemorrhagic and Metabolic Defects Following Burn, with Implications for Treatment (Part of Symposium), John A. Moncrief 4

Hemorrhagic Shock, see *Symposium on Circulatory Shock*

Heritable Disorders of Mucopolysaccharide Metabolism, Hibbard E. Williams (Or.) 306

"Hip-Pointer," The—A Clinical Note, Martin E. Blazina (Or.) 450

Hirsutism and Virilization, Richard J. Sherins and Richard Horton (Or.) 87

Histamine—The View Today, Gildon N. Beall (Or.) 306

Hives, see *Urticaria*

Hospitals, County, in California, see *Changing Role*

How Community Cardiovascular Disease Programs Can Aid the Private Physician, J. Gordon Barrow (Or.) 35

Hypercholesteremia—Treatment with Cholestyramine, a Bile Acid Sequestering Resin, H. R. Casdorph (Or.) 293

Hyperosmolar Non-Ketotic Diabetes (MSC) 131

Hypothalamic-Pituitary-Adrenocortical System — Clinical Evaluation by Pharmacologic Techniques, Grant Gwinup (Or.) 159

Hypothyroidism, see *Facial Paralysis*

I

Insidious Splenic Rupture Due to Infectious Mononucleosis Presenting as Acute Glomerulonephritis, Murray Shevick, Robert Zweig and Lawrence Braslow (CR) 127

Intracranial Aneurysm, Cure of, Without Use of Blood Transfusion, Jack Posnikoff (CR) 124

Irradiation, External, in the Treatment of Transitional Cell Carcinoma of the Bladder, The—A Review of Fifty Cases, George Jack and Franz Buschke (Or.) 12

Isoproterenol, see *Symposium on Circulatory Shock*

L

Laboratory-Physician Relations, see *Medicolegal Responsibilities in*

Lactic Acidosis, see *Symposium on Circulatory Shock*

Legislation, Control and Rehabilitation, Current Status of, see *Narcotic and Dangerous Drug Problems*

LSD^{25} and Emotional Disorder, see *Problems*

LSD^{25}, Ingestion of, see *Grand Mal Seizures*

Lymph Node Biopsy, see *Dermatopathic Lymphadenopathy*

Lymphadenopathy, see *Dermatopathic*

M

Malcolm C. Todd—The New President-Elect (CMA) 412

Malignant Carcinoid Tumor, Spectrum Produced by (MSC) 64

Manpower Council, Health (Ed.) 317

Marijuana: Social Benefit or Social Detriment? Edward R. Bloomquist (Or.) 346

Mastocytosis, Generalized, Fatal Case with Autopsy Findings, Meyer Berke and Phyllis C. Walker (CR) 468

Mechanism to Assure Continuing Medical Education, John B. Dillon (CMA) 235

Medi-Cal Care, Controls on Outlays for (CMA) 493

Medical Assistant, Your, Henry Bodner (I) 242

Medical Education, Continuing, A Mechanism to Assure (CMA) 235

Medical Education, Postgraduate, Toward Improving (CMA) 149

Medical School, see *Role of University in Modern Society*

Medical Staff Conferences:
  Spectrum Produced by Malignant Carcinoid Tumor 64
  Hyperosmolar Non-Ketotic Diabetes 131
  Hematologic Manifestations of Tuberculosis 215
  Periodic Fever 312
  Disorders of Esophageal Motility 405
  Aortic and Mitral Valve Replacement 473

Medicare and Medi-Cal (Ed.) 70

Medicine in Society, see *Responsibility for Quality in Health Care*

Medicine in 2032—An Impressionist's View, Malcolm S. M. Watts (Or.) 272

Medicolegal Responsibilities in Physician-Laboratory Relations, Don Harper Mills (I) 330

Membranous Glomerulonephritis: Rapidly Fatal Course, W. H. Tu, Martin A. Shearn and John C. Lee (CR) 465

Mental Health, see *Methods of Training*

6-Mercaptopurine, Steroid Resistant Nephrosis treated with, see *Subcutaneous Fat Necrosis*

Metabolic Defects During Shock with Particular Reference to Lactic Acidosis (Part of Symposium), Edward P. Marbach 6

Metabolism, Mucopolysaccharide, see *Heritable Disorders of*

Methods of Training in Community Mental Health, John S. Visher (Or.) 117

Mitral and Aortic Valve Replacement (MSC) 473

Mitral Disc Valve, New, see *Clinical Use of*

Mononucleosis, Infectious, see *Insidious Splenic Rupture*

Mucopolysaccharide Metabolism, see *Heritable Disorders of*

Myasthenia Gravis: Brief Guide to Diagnosis and Management, Christian Herrmann, Jr. (Or.) 275

Myasthenia Gravis—The Role of Radiation Therapy, Theodore L. Phillips and Franz Buschke (Or.) 282

Myocardial Infarction and Shock (Part of Symposium), Herbert Shubin 9

N

Narcotic and Dangerous Drug Problems—Current Status of Legislation, Control and Rehabilitation, William F. Quinn (Or.) 108

Necrosis, see *Subcutaneous Fat*

Necrotic Arachnidism, see *Spider Bites*

Needle Aspiration Biopsy of Localized Pulmonary Lesions, G. Melvin Stevens, Glen A. Lillington and John F. Weigen (Or.) 92

Nephrosis, Steroid Resistant, see *Subcutaneous Fat Necrosis*

Nerve Block, Use of, see *Pain Syndromes*

New Dean at University of California-San Diego (Clifford Grobstein) (N/N) 506

Nitrous Oxide Inhalation as a Fad—Dangers in Uncontrolled Sniffing for Psychedelic Effect, John B. Dillon (Or.) 444

North American Blastomycosis in California—Diagnosis of a Case and a Survey with 28 California Observed Cases, Donald E. Casad, William J. Waldmann, Norman E. Levan and Royal H. Sorensen (Or.) 20

Nurses, see *Professional Registered*

O

Obese Person as an Adolescent, The, Dale C. Garell (Or.) 368

Obesity and Weight Control, see *Overweight*

Occipital Pole Tumors, Roentgen Findings in, Larry Bilodeau and William Hanafee (Or.) 112

Occult Glucose Intolerance: Incidence in a General Population, Ronald L. Searcy and Edmund M. Y. Low (Or.) 364

Overweight, Obesity and Weight Control, Marie M. Alexander and Fredrick J. Stare (Or.) 437

P

Pain Syndromes—The Use of Nerve Block in Diagnosis and Treatment, Verne L. Brechner (Or.) 290

Pancreatitis, Acute Hemorrhagic, see *Subcutaneous Fat Necrosis*

Paralysis, Facial, Occurring with Hypothyroidism, A Report of Two Cases, Jerry M. Earll and Felix O. Kolb (CR) 56

Perineal Prostatectomy, Total, for Carcinoma, Indications and Results, M. J. Feeney, R. B. Mullenix, R. J. Prentiss and G. E. Howe (Or.) 372

Periodic Fever (MSC) 312

Physician-Laboratory Relations, see *Medico-legal Responsibilities in*

Pineal Germinoma with Endocrine Manifestations (Residents' Clinicopathologic Conference) (Or.).. 196

Pituitary-Adrenocortical System, see *Hypothalamic*

Poikilocytosis in Cancer Patients, Donald I. Galen and William H. Kern (Or.) 447

Population Explosion, Community Psychiatry and the, Ames Fischer (Or.) 189

Postgraduate Medical Education, John B. Dillon (CMA) 426

Postgraduate Medical Education, Toward Improving (CMA) 149

Poverty in the United States as Defined by Federal Standards (BRP) 501

Pregnancy, see *Duodenal Ulcer*

Prescribing Drugs and Devices, see *Ethical Responsibilities in*

President-Elect, Malcolm C. Todd (CMA) 412

President's Address, see *Accomplishments and Frustrations*

Problems of LSD^{25} and Emotional Disorder, J. Thomas Ungerleider and Duke D. Fisher (Or.) 49

Professional Registered Nurses in the United States and California, 1962 (BRP) 327

Progress in the Treatment of Shock: A Historical Perspective (Part of Symposium) Sam F. Seeley.... 1

Project Head Start—An Evaluation of the Medical Components in California, Arnold Gilbert, Arline Lewis and Robert W. Day (Or.) 382

Prostatectomy for Carcinoma, see *Total Perineal*

Prosthetic Cardiac Valves, Current Status of, Wilfred M. Huse and Ellsworth E. Wareham (Or.) 203

Psychedelic Effect, see *Nitrous Oxide Inhalation*

Psychiatry, Community, and the Population Explosion, Ames Fischer (Or.) 189

Pulmonary Embolism, see *Symposium on Circulatory Shock*

Pulmonary Emphysema, see *Chronic Obstructive*

Pulmonary Lesions, Localized, see *Needle Aspiration Biopsy*

Q

Quality of Health Care (Ed.) 484

R

Radiation Therapy, see *Myasthenia Gravis*

Recent Concepts in the Clinical Pharmacology of Antihypertensive Drugs, Alan S. Nies and Kenneth L. Melmon (Or.) 388

Recent Developments in the Management of Gastrointestinal Malformations, Eric W. Fonkalsrud (Or.) 451

Recurent Cystosarcoma Phylloides, James R. Hansen (CR) 462

Registered Nurses, Professional, in the United States and California, 1962 (BRP) 327

Residents' Clinicopathologic Conference, see *Pineal Germinoma*

Responsibility for Quality in Health Care (Part I of a Report by the Committee on the Role of Medicine in Society (CMA) 486

Roentgen Findings in Occipital Pole Tumors, Larry Bilodeau and William Hanafee (Or.) 112

Role of External Irradiation in the Treatment of Transitional Cell Carcinoma of the Bladder, The —A Review of Fifty Cases, George Jack and Franz Buschke (Or.) 12

Role of Medicine in Society, see *Responsibility for Quality in Health Care*

Role of the University in Modern Society, A Description of the, Together with Encouragement to the Medical School to Turn Its Flirtation with the University into a Full Blown Romance, Charles E. Odegaard (Or.) 337

S

Saskatchewan, Socialism in: A Fiasco (I) 82

Shock, Circulatory, see *Symposium on Circulatory Shock*

Shock Research Unit at the Los Angeles County General Hospital (Part of Symposium), Max Harry Weil 4

Shoulder Disability, Exercise Program for, David Rubin (Or.) 39

Socialism in Saskatchewan: A Fiasco (I) 82

Spectrum Produced by Malignant Carcinoid Tumor, The (MSC) 64

Spider Bites, Tick Bites, Letters to Editor (Refers to Necrotic Arachnidism, Calif. Med. 105:457-462, Dec. 1966) (LE): James H. Dooley 247
  Martin I. Lewis 247
  Findlay E. Russell 248
  William G. Waldron 248

Splenic Rupture, Insidious, Due to Infectious Mononucleosis Presenting as Acute Glomerulonephritis, Murray Shevick, Robert Zweig and Lawrence Braslow (CR) 127

Statement of CMA Goals—A Supplementary Report of the Council to the House of Delegates (CM) 490

Statutory Consent for Chemical Test of Alcoholic Content of Blood, Breath or Urine in Drunk Driving Cases, Howard Hassard 80

Study of 150 Instances of Cardiac Arrest with Implications for Treatment (Part of Symposium), Samuel J. Camarata, Edward C. Bradley and Max Harry Weil 7

Subcutaneous Fat Necrosis with Acute Hemorrhagic Pancreatitis—A Case in a Child with Steroid—Resistant Nephrosis Treated with 6-Mercaptopurine, Eva Heuser, Ellin Lieberman, George N. Donnell and Benjamin H. Landing (CR) 58

Successful Annual Meeting (Ed.) 410

Symposium on Circulatory Shock:
  Advances in the Understanding of Mechanisms and Treatment of Circulatory Shock, An Introduction, Max Harry Weil, Coordinator 1
  Progress in the Treatment of Shock: A Historical Perspective, Sam F. Seeley 1
  Anaphylaxis and Endotoxin, Wesley W. Spink 2
  Guide Lines for the Selection of Fluids and Drugs for the Treatment of Patients in Shock, Lloyd D. MacLean 3
  Hemorrhagic and Metabolic Defects Following Burn, with Implications for Treatment, John A. Moncrief 4
  Shock Research Unit at the Los Angeles County General Hospital, The, Max Harry Weil 4
  Metabolic Defects During Shock with Particular Reference to Lactic Acidosis, Edward P. Marbach 6
  Central Venous Blood for Monitoring pH and PCO^2 in the Critically Ill Patient, Richard L. Zahn 6
  Ventilatory Failure as a Major Clinical Cause of Fatal Progression of Shock, Paul K. Hanashiro 7
  Study of 150 Instances of Cardiac Arrest with Implications for Treatment, Samuel J. Camarata, Edward C. Bradley and Max Harry Weil.. 7
  Comparison of the Capillary and Central Hematocrit as an Indication of Circulatory Shock, Donald B. Doty and M. H. Weil 8
  Myocardial Infarction and Shock, Herbert Shubin 9
Hemodynamic Studies on Clinical Shock Following Pulmonary Embolism, Roy T. Adachi and E. C. Bradley 9
  Treatment of Circulatory Shock with Isoproterenol, Edward C. Bradley and M. H. Weil 10
  Effect of Corticosteroids on the Progression of Hemorrhagic Shock in Rats, Howard Whigham 10
  Application of Computer Techniques to Treatment of the Critically Ill Patient, Marshall A. Rockwell, Jr. 11

Systemic Agents in the Management of Acne, Ronald M. Reisner (Or.) 28

T

Therapeutic Abortion, Where We Stand on the Question of (CMA) 318
  Letter to Editor, Edward J. Wiater 499

Todd, Malcolm C., The New President-Elect (CMA) 412

Total Perineal Prostatectomy for Carcinoma—Indications and Results, M. J. Feeney, R. B. Mullenix, R. J. Prentiss and G. E. Howe (Or.) 372

Toward Improving Postgraduate Medical Education, Phil R. Manning and James C. MacLaggan (CMA) 149

Treatment of Circulatory Shock with Isoproterenol (Part of Symposium), Edward C. Bradley and M. H. Weil 10

Tuberculosis, Hematologic Manifestations of (MSC) 215

Tumor Registry Data, Use of, Sidney J. Cutler (Or.) 98

Tumors, Roentgen Findings in Occipital Pole, Larry Bilodeau and William Hanafee (Or.) 112

U

Ulcer, Duodenal, with Massive Hemorrhage Complicating Pregnancy, Charles G. King, Robert Steedman and J. Edward Berk (CR) 401

Unsuspected Carcinoma Found Histologically After Minor Anorectal Operations, Lewis Grodsky (Or.) 44

Urine, Alcoholic Content of, see *Statutory Consent for Chemical Test of*

Urticaria, see *Autoimmune Progesterone*

Urticaria from Contact with Water, Theodore A. Tromovitch (CR) 400

Use of Tumor Registry Data, Sidney J. Cutler (Or.) 98

V

Venous Blood, Central, see *Symposium on Circulatory Shock*

Ventilatory Failure as a Major Clinical Cause of Fatal Progression of Shock (Part of Symposium), Paul K. Hanashiro 7

Virilization and Hirsutism, Richard J. Sherins and Richard Horton (Or.) 87

W

Weight Control, see *Overweight*

Where We Stand on the Question of Therapeutic Abortion (CMA) 318
  Letter to the Editor, Edward J. Wiater 499

Y

Your Medical Assistant, Henry Bodner (I) 242

MEDICAL PROGRESS ARTICLES

Current Status of Prosthetic Cardiac Valves, Wilfred M. Huse and Ellsworth E. Wareham 203

Heritable Disorders of Mucopolysaccharide Metabolism, Hibbard E. Williams 306

Hirsutism and Virilism, Richard J. Sherins and Richard Horton 87

Histamine—The View Today, Gildon N. Beall 296

Hypothalamic-Pituitary-Adrenocortical System — Clinical Evaluation by Pharmacologic Techniques, Grant Gwinup 159

Problems of LSD^{25} and Emotional Disorder, J. Thomas Ungerleider and Duke D. Fisher 49

Recent Developments in the Management of Gastrointestinal Malformations, Eric W. Fonkalsrud 451

EDITORIALS

Audio-Digest's Benign Cycle 136

California Health Manpower Council, The 317

Chairman Retires, The 411

For Realism in Medical Legislation 136

Health Care of the People 222
Medicare and Medi-Cal 70
Quality Health Care 484
Successful Annual Meeting 410

CALIFORNIA MEDICAL ASSOCIATION

Budgeting Process, The 73
Committee on the Role of Medicine in Society: Part I—Responsibility for Quality in Health Care 486
Continuing Medical Education:
  Toward Improving Postgraduate Medical Education, Phil R. Manning and James C. MacLaggan 149
  Mechanism to Assure Continuing Medical Education, A, John B. Dillon 235
  Postgraduate Medical Education, John B. Dillon 426
Controls on Outlays for Medi-Cal Care 493
Council Meeting Minutes:
  526th Meeting, 22 November 1966 138
  527th Meeting, 10 December 1966 139
  528th Meeting, 15 January 1967 224
  529th Meeting, 25 February 1967 413
Malcolm C. Todd—The New President-Elect 412
New Dean at UC-San Diego (Clifford Grobstein) 506
Statement of CMA Goals 490
Where We Stand on the Question of Therapeutic Abortion 318
  Letter to Editor, Edward J. Wiater 499

BUREAU OF RESEARCH AND PLANNING

Federalized Health Care Systems in Western Europe and Australia 243
Professional Registered Nurses in the United States and California, 1962 327
Poverty in the U.S. as Defined by Federal Standards 501

INFORMATION

Dentist-Physician Relationship in Extended Care Facilities 243
Ethical Responsibilities in Prescribing Drugs and Devices, AMA Judicial Council 505
Medicolegal Responsibilities in Physician-Laboratory Relations, Don Harper Mills 330
Socialism in Saskatchewan: A Fiasco 82
Statutory Consent for Chemical Test of Alcoholic Content of Blood, Breath or Urine in Drunk Driving Cases, Howard Hassard 80
Your Medical Assistant, Henry Bodner 242

BOOK REVIEWS

Appraisal of Current Concepts in Anesthesiology—Vol. 3, *Adriani* 253
Atlas of Vascular Surgery—2nd Ed., *Hershey & Calman* 432
Cardiac Surgery, *Norman* 508
Children of Time and Space, of Action and Impulse, *Ekstein* 157
Ciba Foundation Symposium—The Thymus: Experimental and Clinical Studies, *Wolstenholme and Porter* 254
Citanest,® *Wiedling* 86
Clinical Management of Behavior Disorders in Children—3rd Ed., *Bakwin and Bakwin* 254
Clinical Obstetrics and Gynecology—Vol. 9, No. 3, Sept. 1966, *Carrington and de Alvarez* 252
Color Atlas of Dermatology (Frieboes/Schönfeld), *Kimmig and Jänner* 336
Consumers Union Report on Family Planning, *Consumers Union of U.S., Inc.* 335
Disorders of the Respiratory Tract in Children, *Kendig* 252
Dr. Thomas Sydenham, *Dewhurst* 432
Epilepsy and the Law, Rev. 2nd Ed., *Barrow & Fabing* 158
Family Planning, Consumers Union Report, *Consumers Union of U.S., Inc.* 335
Frieboes/Schönfeld Color Atlas of Dermatology, *Kimmig and Jänner* 336
Gastrin, *Grossman* 508
Hematologic Problems in the Newborn, *Oski & Naiman* 85
Mental Health in a Changing Community, *Brockbank and Westby-Gibson* 336
Metabolic Toxemia of Late Pregnancy, *Brewer* 85
Neuro-Ophthalmology—Vol. II, Symposium of the University of Miami and the Bascom Palmer Eye Institute, *Smith* 85
Primer on Prematurity and High-Risk Pregnancy, *Babson and Benson* 251
Progress in Clinical Psychology—Vol. VII, *Abt and Riess* 251
Psychiatry in the Practice of Medicine, *Enelow* and *Wexler* 157
Psychotherapy: Myth and Method, *Ehrenwald* 156
Scientific Yearly of the Lincoln Chiropractic College 253
Scleroderma, *Sackner* 335
Surgery in America, *Earle* 85
Surgical Diseases of the Chest—2nd Ed., *Blades* 157
Survey of Surgical Shock With Special Reference to Fluids, Electrolytes and Metabolites, *Pollock* 254
Symposium of the University of Miami and the Bascom Palmer Eye Institute—Neuro-Ophthalmology, Vol. II, *Smith* 85
Synopses of Dermatology, *Stewart* et al 508
Synopsis of Neurology—2nd Ed., Forster 86
Thymus, The: Experimental and Clinical Studies, Ciba Foundation Symposium, *Wolstenholme and Porter* 254
Treatment of Fractures (Supplementary Volume to 5th English Edition, *Bohler et al.* 156
Year Book of Anesthesia (1966-1967 Year Book Series), *Cullen* 335

DEATHS

Aronstein, Ernst, Fresno, 16 Nov. 1966 320
Barton, John C., Riverside, 15 March 1967 423
Berlin, Harold D., Los Angeles, 11 Jan. 1967 423
Bigotti, Richard Thomas, San Jose, 21 Dec. 1966 148
Blondin, Edward A., La Mesa, 25 Jan. 1967 233
Brindamour, Jean-Louis Edmond, Palo Alto, 24 March 1967 423
Brown, William Edward, Los Angeles, 25 Nov. 1966 75
Bruck, Edwin L., San Francisco, 5 Jan. 1967 148, 232
Churchill, James Forrest, San Diego, 21 Apr. 1967 494
Claman, Zalman Bernard, Los Angeles, 2 Mar. 1967 320
Clark, George Sherman, Los Angeles, 20 Apr. 1967 494
Clark, Roscoe Loyal, Pittville, 9 Apr. 1967 494
Cox, Thomas J., San Rafael, 17 Apr. 1967 494
Craycroft, Charles Burr, San Jose, 2 Feb. 1967 233
Dahlgren, Roland Walfred, Fresno, 4 Oct. 1966 148
Dale, Philip M., Los Angeles, 29 Jan. 1967 233
Delamere, Granville Sinclair, Marysville, 21 March 1967 423
Dieterle, Karl Lyonal, Los Angeles, 6 Nov. 1966 75

Enterline, Joseph Donald, Reedley, 5 Apr. 1967 494
Farquharson, R. E. Hughes, Redwood City, 15 Apr. 1967 494
Forteza, Rosendo, Jr., 31 Jan. 1967 320
Garn, Don Carlos, Bakersfield, 17 Feb. 1967 320
Gattuccio, Bartholomew, San Jose, 21 Jan. 1967 233
Giovannetti, Rinaldo P., San Francisco, 6 Apr. 1967 494
Goddard, Edward P., Oroville, 5 Feb. 1967 320
Godett, Michael Robert, San Marino, 24 Dec. 1966 148
Goodman, Martin, North Hollywood, 3 Dec. 1966 75
Grant, William Thomas, Sierra Madre, 13 Apr. 1967 494
Gray, Frederick Lyle, Long Beach, 3 May 1967 494
Greene, Clarence Orville, Lodi, 2 Jan. 1967 233
Greenwood, Earl Norman, San Francisco, 28 March 1967 423
Griffith, William H., Los Angeles, 13 Feb. 1967 320
Griggs, Donald Ezra, Glendale, 9 March 1967 423
Griner, Ernest C., Stockton, 30 Oct. 1966 75
Groff, Sutten Henry, Granada Hills, 20 March 1967 423
Guenther, Robert Edward, Midway City, 11 Mar. 1967 494
Hamilton, Michael J., Redding, 26 Mar. 1967 494
Hamrick, Jewel G., Santa Ana, 7 Feb. 1967 320
Hand, Delbert Wilfred, San Francisco, 2 Jan. 1967 148
Harris, Robert Henry, Valley Center, 15 March 1967 423
Heilbronn, Alexander, Oakland, 25 Nov. 1966 75
Hilgesen, James, Campbell, 16 Jan. 1967 233
Hollingsworth, Loren D., Laguna Beach, 26 Dec. 1966 233
Horswill, Harry Lee, Tulare, 2 Dec. 1966 75
Hunter, William Edward, Los Angeles, 27 Feb. 1967 423
Imerman, Harold Maurice, Beverly Hills, 25 March 1967 423
Irvine, Alexander Ray, Beverly Hills, 14 Apr. 1967 494
Johnson, William T., Alhambra, 5 Nov. 1966 75
Johnstone, Rutherford T., Los Angeles, 27 Apr. 1967 494
Jones, F. Burton, Vallejo, 2 Jan. 1967 148
Joslin, Eric Francis, Visalia, 11 Feb. 1967 320
Katz, Benjamin, Los Angeles, 6 Nov. 1966 75
Kohlhase, Lawrence H., Los Angeles, 4 May 1966 148
Krull, Frank, Sacramento, 5 Dec. 1966 75
Lachman, Harry, Santa Monica, 1 Dec. 1966 75
Langton, John Nesbitt Kent, Berkeley, 13 Apr. 1967 494
Lee, Lawrence Dewey, Glendale, 21 Nov. 1966 75
Low, Chin Y., San Francisco, 5 March 1967 320
Lusby, Fletcher H., Inglewood, 13 Sept. 1966 148
Manson, Guy, Fresno, 27 Dec. 1966 148
Mayes, Duward Lacy, Santa Ana, 29 Mar. 1967 494
McCurdy, Theodore E. A., Berkeley, 25 Nov. 1966 75
McGee, William (Buster), San Diego, 15 Feb. 1967 320
McNeil, Edwin Ewert, Los Angeles, 16 March 1967 423
McRae, Colin Christopher, San Francisco, 6 Feb. 1967 233
Medoff, Murray H., North Hollywood, 24 Jan. 1967 233
Moore, E. Nelson, Hollister, 22 Nov. 1966 75
Moorman, Henry Dean, Pasadena, 29 Jan. 1967 320
Munoz, Rafael, Rolling Hills, 23 Feb. 1967 320
Murphy, William K., Veterans Home (Napa Co.), 6 Feb. 1967 233
Niemand, Frederick George, Fullerton, 22 Apr. 1967 494
Nierenberg, Harry H., Beverly Hills, 14 Apr. 1967 494
Owen, Lincoln L., Vista, 26 Apr. 1967 494
Parry, Gronway R., Jr., Mountain View, 4 February 1967 233
Pearl, Felix L., San Francisco, 31 Dec. 1966 495
Pizey, Bryce A., San Pedro, 11 Nov. 1966 75
Pottenger, Francis M., Jr., Monrovia, 4 Jan. 1967 148
Pyle, Howard R., San Mateo, 23 January 1967 233
Raitt, G. Emmett, Santa Ana, 30 Dec. 1966 148
Riesenfeld, Fred Fritz, Los Angeles, 13 Jan. 1967 233
Rogers, Thomas L., Long Beach, 6 Feb. 1967 320
Rosner, Samuel, Santa Ana, 7 Feb. 1967 320
Roth, Harold Alan, Riverside, 5 Jan. 1967 233
Ryan, Andrew Morris, San Francisco, 13 Apr. 1967 495
Schierman, Daniel William, Whittier, 12 March 1967 423
Schinagel, Geza, Burbank, 4 Jan. 1967 148
Schneider, John L., Imperial Beach, 5 May 1967 495
Schneider, Kenneth F., Los Angeles, 24 Jan. 1967 233
Schwartz, Arthur Henry, Beverly Hills, 16 Feb. 1967 320
Schwartz, Joseph Leon, Sr., Los Angeles, 22 Apr. 1967 495
Schweitzer, S. Rowland, Sacramento, 18 Jan. 1967 233
Seal, Alfred L., Beverly Hills, 13 Apr. 1967 495
Shearer, Wallace C. Jr., Palos Verdes, 26 Jan. 1967 234
Shepardson, Harry Clare, San Francisco, 5 March 1967 321
Siefert, Alfred Camile, Oakland, 10 March 1967 423
Skleba, Leonard Frederick, Glendale, 1 Feb. 1967 321
Smith, Arthur E., Los Angeles, 18 Nov. 1966 75
Smith, Charles E., Berkeley, 18 Apr. 1967 495
Smith, Freid Lynn, Colfax, 18 Feb. 1967 423
Sorenson, Einer I., Porterville, 21 Jan. 1967 234
Spasser, Irvin Charles, Los Angeles, 11 March 1967 423
Spirtos, George Nicholas, Woodland Hills, 8 Aug. 1966 148
Stephens, Brooks Palmer, Ben Lomond, 15 Jan. 1967 234
Stewart, David, Covina, 21 Nov. 1966 75
Stillwell, Tyler C., Oxnard, 16 Nov. 1966 76
Stone, Robert Spencer, San Francisco, 18 Dec. 1966 148
Sturgeon, Charles T., Santa Barbara, 20 Jan. 1967 234
Taylor, Merrel H., San Diego, 8 Oct. 1966 148
Thomas, Roy E., Los Angeles, 26 Oct. 1966 76
Thompson, Edgar Allen, Glendora, 27 Jan. 1967 234
Vincent, Elmo H., Rolling Hills Estate, 8 Oct. 1966 321
Voorsanger, William C., San Francisco, 24 Jan. 1967 234
Wagenseller, Samuel Miller, Ahwahnee, 26 Mar. 1967 495
Weber, Robert, Los Angeles, 15 March 1967 423
Weeks, Walter G., Oakland, 11 Apr. 1967 495
Welch, Sylvester H., Glendale, 5 Dec. 1966 148
Welcome, Earl H., National City, October 1966 76
Wharton, George Murdock, Los Angeles, 21 Nov. 1966 148
Willson, Lamont Roy, Fresno, 17 Dec. 1966 148
Wuerthele, Herman William, La Jolla, 5 Jan. 1967 148
Wyckoff, Harry A., Piedmont, 26 Feb. 1967 321
Wyers, Robert E., Whittier, 25 Apr. 1967 495
Ziegler, Julius, Los Angeles, 22 Feb. 1967 321

pp 275 – 282 [illegible]
[illegible]